Evolution of the Gene Rotation Concept for Rice Blast Control

A Compilation of 10 Research Papers

International Rice Research Institute

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The responsibility for this publication rests with the International Rice Research Institute.

Contents

Introduction

Blast, caused by the fungus pathogen *Pyricularia oryzae* remains the world's most important disease of rice, despite intensive efforts to control it. Blast makes the cultivation of rice impossible in many areas.

Chemical control of rice blast can be satisfactory, but the technology required is complicated, expensive, and, thus, often beyond the reach of the world's smaller and poorer rice farmers. The use of horizontal and monogenic resistance for blast control has also been ineffective in many areas.

The rotation of specific genes for resistance is a new and effective method of blast control. The method is based on local studies of the evolution of pathogenicity within the blast fungus population. Through the blast nurseries of the International Rice Testing Program (IRTP), information on the pathogenicity of local blast fungus populations can be made available to local plant breeding programs, which can then synthesize appropriate resistant varieties to combat new blast races before they develop.

The concept of plant disease control through gene rotation based on race prediction studies evolved over a 10-year period. This publication comprises a collection of ten research papers that document and trace that evolution. The concept originated with tomato disease control and was finally utilized on a national scale with monogenic blast resistance in rice. This program illustrates the value of research to develop concepts: regardless of the crop or problem, proven concepts can be extended to all types of research.

The value of monogenic resistance and its advantages and proper uses were stressed in the 1972 paper "Controlling Fusarium Wilt of Tomatoes With Resistant Varieties." This paper, which contradicted the concept of polygenic resistance for disease control as advocated by Van der Plank in 1968, appeared about the same time as the report on *Genetic Vulnerability of Major* Crops (which generally discouraged the use of monogenic resistance to control diseases). The 1972 paper was the first to seriously question the value of horizontal resistance and to state that monogenic resistance was a preferred method for certain diseases.

The 1973 papers ''Failure of 'Horizontal Resistance' to Control Fusarium Wilt of Tomato" and "Prevalence of the Vertifolia Effect in Fusarium Wilt Disease of Tomato" were based on intensive field research. Monogenic resistance was conclusively shown superior to horizontal resistance in the field and the "vertifolia effect" was shown to be invalid. An extension and integration of these findings was published in 1 974 as "Evaluation of Some Concepts of Variety Development and Disease Control With Host Resistance."

These concepts were extended in an invited paper "Host Resistance and Disease Control in Tomato," presented at the 1974 *Symposium on Crop Protection in the Caribbean* held at the University of West Indies, St. Augustine, Trinidad. In that paper, the concepts of intensive and subsistence agriculture were defined and characterized for the first time with the objective of integrating programs of varietal development and crop production. Also explored was the

concept of monitoring crops to detect new diseases and to rapidly replace varieties that are rendered susceptible. The sixth paper is a monograph whose core was two 1973 papers published at the University of Florida on the use of F_1 hybrid varieties as a tool to rapidly introduce new sources of resistance to new races or diseases. The concept of rotating specific monogenes to manage the evolution of pathogen races was explored in detail in the 1977 paper "An Assessment of Stabilizing Selection in Crop Variety Development."

The theoretical aspects of controlling rice blast were explored in detail in the 1979 paper "Effective and Stable Control of Rice Blast with Monogenic Resistance." The failure of horizontal resistance to control rice blast was documented and the control strategy of rotating monogenes for resistance based on race prediction studies was fully explored.

Recognition that the concepts established for tomato disease control were equally applicable to rice diseases led to the establishment of a gene rotation program in 1979 in the Republic of Korea. The final paper describes that blast-control program. This collection of research papers was published because gene rotation for blast control is difficult to grasp without an understanding of several basic principles of host resistance for disease control. The principles of gene rotation are embodied in the first papers and their application to control disease in the last.

IRRI recognizes that gene rotation cannot presently be used to control rice blast in certain rice-growing regions - for example, in areas where access to the required support technology and methods is limited. For success in gene rotation, all concerned must be fully committed to increased rice production. Gene rotation cannot now be used everywhere by everyone, but we assume that, with time, gene rotation will increasingly become an important technology.

The recent development of F_1 hybrid rice varieties opens new horizons for gene rotation. Today about 6 million hectares of hybrid rice are cultivated, mostly in China. F_1 hybrids are an excellent vehicle to rapidly incorporate new resistance genes. Because the monogenes that control blast resistance are primarily dominant gene rotation can be incorporated into a program for the development of hybrid rice varieties.

It is extremely rare that a new concept is transferred from its conception to inception as rapidly as was gene rotation based on race prediction in Korea. The evolution of the gene rotation program is even more significant when one considers that it was a cooperative effort of scientists, administrators, and government officials in Korea and at the International Rice Research Institute (IRRI).

Truly cooperative and collaborative research between Korean and IRRI scientists since 1964 set the stage for Korean Government funding of the project and led to the concept's rapid application for practical blast control in farmers' fields on an unprecedented scale.

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Controlling Fusarium wilt of tomato with resistant varieties[1]

Pat Crill, John Paul Jones,
D. S. Burgis, and S. S. Woltz[2]

ABSTRACT

The historical aspects of breeding tomatoes for resistance to *Fusarium oxysporum lycopersici* and the resulting development of new races is described. Inoculation techniques and methods of evaluating screening tests are discussed. Two types of host : pathogen interaction are described: polygenic tolerance and monogenic resistance. The merits and use of each interaction are dis cussed.

INTRODUCTION

The University of Florida tomato breeding program was established in 1924 at what is now the Agricultural Research and Education Center, Bradenton, at the request of several tomato growers in the area. Because Fusarium wilt was the primary problem with varieties grown at that time, a plant pathologist, Dr. George F. Weber, was given the responsibility of developing wilt resistant varieties. Weber isolated sources of tolerance to *Fusarium oxysporum lycopersici* from which three highly tolerant varieties, Newell, Cardinal King, and Ruby Queen, were released (7).

In 1941, the variety Pan America was released which possessed the I gene for resistance to Fusarium wilt (8). This gene was immediately utilized and almost all tomato varieties grown commercially in Florida since 1949 have contained this gene (1 4). Race 2 of the fungus was detected in 1945 in Ohio (2), but was not

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found in Florida until 1960 (9, 10). This pathogen spread rapidly into all the tomato sand-land areas of Florida and became the limiting factor in tomato production. Resistance to race 2 was first reported by Alexander (1). Stall and Walter (11) selected a single gene, 12, which was transferred to commercial types and conferred resistance to race 2. In 1969, the variety Walter was released which had resistance to both race 1 and 2 (12) and by 1971, it was the most widely grown variety in Florida.

SCREENING TECHNIQUES

A dependable technique for the determination of resistance to Fusarium wilt is indispensable for breeding and genetic studies. A modification of the root-dip method reported by Wellman (15) provides the quickest, most dependable results. This method consists of uprooting 10-14-day-old seedlings which have been grown at 80° F in a steam-sterilited sandy soil, washing the roots in running tap water momentarily to remove most of the adhering soil, immersing roots in a dense suspension of Fusarium cells, transplanting seedlings into steam-sterilized soil and incubating at 82° F.

Stock cultures of *Fusarium* are maintained in soil tubes. These are prepared by isolating the wild fungus from a diseased plant, singlemicrosporing the wild type, increasing the mono-microconidial cultures on potatodextrose agar (PDA), and testing for virulence. The most virulent isolates are used to infest soil tubes and each tube containing a monoconidial culture is stored at 55° F. Starter plates are made by seeding PDA plates with a small amount of the stock fungus-soil culture which are incubated 5-7 days at 82° F. One-cm-diameter plugs are removed from the starter plates, placed on thinly poured PDA agar plates (one/plate) and incubated 5-7 days at 82° F. The agar-mycelium is homogenized aseptically with a microblender, and the resulting suspension adheres well to the seedling roots. From numerous experiments it was concluded the agar has no effect on infection or pathogenesis. This technique assures proper inoculum concentration which is important inasmuch as wilt may not develop, and there will be many escapes if concentration is low (Tables 1 and 2). Conversely, if the inoculum is too concentrated, tolerant varieties are rapidly killed.

It is essential that susceptible checks be included in every test, and in every replication. When screening for resistance to either race 1 or race 2 it is desirable to include a tolerant, but not resistant, variety such as Rutgers or Marglobe and a completely susceptible nontolerant variety such as Bonny Best. The inclusion of such control varieties is often essential in interpreting the results of a screening test.

Three criteria for evaluating screening tests have been developed: a) number of diseased plants (Table 1), b) disease index (Table 2), and c) number of dead plants (Table 3). With all three criteria the variety Walter is resistant (no symptom development occurs). Likewise, Bonny Best is the most susceptible of all varieties, presumably because it contains no genes for either resistance or tolerance. Inoculum level and date of evaluation of the test are quite important in interpreting results of screening tests to evaluate tolerance (Tables 1, 2 and 3). Neither is

worthy of significant consideration when resistance rather than tolerance is being evaluated (Tables 1, 2 and 3).

Although Fusarium wilt development is affected by day length, light intensity, temperature, nutrition, soil pH and soil moisture, these factors have been ignored by many workers using the root-dip method because it was assumed pathogene sis was not seriously affected within the ranges regarded as optimum for crop

Table 1. Host response of tomatovarieties to races 1 and 2 and number of diseased plants per 75 inoculated with *F. oxysporum f. lycopersici,* race 2.

Variety	Host response [a]		Number of days after inoculation									
			5			10			20			
	Race 1	Race 2	Inoculum level [b]			Inoculum level			Inoculum level			
			High	Med	Low	High	Med	Low	High	Med	Low	Mean
Walter	R	R	0	0	0	0	0	0	0	0	0	0
Bonny Best	S	S	55	43	10	68	72	28	69	73	32	58
Indian River	R	T	15	26	6	33	46	14	40	46	18	35
Floradel	R	T	18	20	1	36	30	7	40	46	28	38
Manapal	R	T	22	30	3	46	52	14	52	61	19	44
Homestead 24	R	T	21	12	1	41	35	7	51	40	7	33
Tropic	R	T	14	16	6	33	38	11	42	50	14	35
Marglobe	T	T	38	24	4	53	35	13	63	60	13	45
Rutgers	T	T	30	25	3	46	42	16	55	51	26	44
LSD .05			3.6	3.6	3.6	2.7	2.7	2.7	2.7	2.7	2.7	1.5

[a] R = resistant, S = susceptible, T = tolerant. [b] Low, Med, and High = 0.25 X 10^6, 4.25 x 10^6, and 10.5 x 10^6 spores/ml, respectively.

Table 2. Disease index [a] for 75 tomato plants inoculated with *F. oxysporum f. lycopersici,* race 2.

Variety	Number of days after inoculation									
	5			10			20			
	Inoculum level [b]			Inoculum level			Inoculum level			
	High	Med	Low	High	Med	Low	High	Med	Low	Mean
Walter	0	0	0	0	0	0	0	0	0	
Bonny Best	2.0	1.5	0.3	3.5	3.4	0.8	4.2	4.3	1.4	
Indian River	0.5	0.7	0.2	1.0	1.4	0.4	1.4	1.9	0.76	
Floradel	0.5	0.6	0.01	1.2	1.0	0.1	1.5	1.7	0.9	
Manapal	0.6	1.0	0.1	1.5	1.9	0.4	1.7	2.5	0.7	
Homestead 24	0.6	0.3	0.04	1.4	1.0	0.2	1.9	1.6	0.25	
Tropic	0.5	0.4	0.2	1.0	1.0	0.3	1.5	1.7	0.5	
Marglobe	1.2	0.8	0.08	2.0	1.3	0.3	2.5	2.5	0.5	
Rutgers	0.9	0.7	0.07	1.7	1.5	0.4	2.3	1.9	0.9	
LSD .05	0.1	0.1	0.1	0.12	0.12	0.12	0.13	0.13	0.13	0.07

[a] 0 = no disease, 5 = dead. [b] Low, Med, and High = 0.25 x 10^6, 4.25 x 10^6, and 10.5 x 10^6 spores/ml, respectively.

Table 3. Host response of tomato varieties to races 1 and 2 and number of dead plants per 75 inoculated with *F. oxysporum f. lycopersici,* race 2.

Variety	Host response[a]		Number of days after inoculation									
			5			10			20			
	Race 1	Race 2	Inoculum level[b]			Inoculum level			Inoculum level			
			High	Med	Low	High	Med	Low	High	Med	Low	Mean
Walter	R	R	0	0	0	0	0	0	0	0	0	
Bonny Best	S	S	0	1	0	19	21	2	49	45	6	
Indian River	R	T	0	0	0	2	2	1	2	14	6	
Floradel	R	T	0	0	0	6	4	0	8	6	0	
Manapal	R	T	0	1	5	4	6	0	4	14	2	
Homestead 24	R	T	1	0	0	5	1	0	11	10	1	
Tropic	R	T	0	0	0	0	0	0	3	2	3	
Marglobe	T	T	0	1	0	6	5	0	13	16	2	
Rutgers	T	T	1	0	0	5	6	0	14	11	1	
LSD .05						1.4	1.4	1.4	2.7	2.7	2.7	1.6

[a] R = resistant, S = susceptible, T = tolerant. [b] Low, Med, and High = 0.25 x 10^6, 4.25 x 10^6, and 10.5 X 10^6 spores/ml, respectively.

development. For consistent results which avoid escapes that confound genetic ratios, however, all environmental factors are controlled as far as feasible.

Fusarium will not grow well, will not sporulate, and is weakly virulent when grown in liquid cultures devoid of micronutrients (16). These micronutrients are, for the most part, unavailable at soil pH values of 7.0-8.0. These micronutrients are so essential for infection and pathogenesis that the root-dip method will yield poor and inconsistent results if seedlings are transplanted to micronutrient-deficient soils (6). To avoid this problem, either the soil pH should be adjusted to less than 6.0 or the high pH soil should be amended with lignosulfonate-micronutrient complexes or other sources which are available at high pH. Seedlings which are to be tested should be grown in low nitrogen soils because plants deficient in nitrogen are more susceptible to Fusarium wilt than plants high in nitrogen (6).

Using the techniques described above, symptom development is usually apparent 5-7 days after inoculation and pronounced after 10-14 days. The most obvious symptoms are: a) stunting or reduced growth, b) loss of cotyledons or cotyledons become chlorotic and abscise easily when disturbed, c) presence of dark streaks in the stem when the epidermal layers are scraped or cut away. A very efficient and effective method of rating seedling inoculation screening results has been described (5).

BREEDING AND GENETICS

Two sources or types of reactions to *F. oxysporum* lycopersici have been recognized in tomato and used to control this disease (14). The first is a polygenically controlled mechanism. This host : pathogen interaction has been termed horizontal resistance (13). This reaction does not, in fact, represent a resistance

mechanism at all. Consistent with the terminology of this paper, this reaction represents tolerance. The pathogen invades the host, produces classical symp toms and eventually kills the plant; thus the host is susceptible to the Fusarium wilt pathogen. Such plants are indeed susceptible but tolerate the presence of the pathogen for a longer period of time before succumbing. This type of interaction properly referred to is termed polygenic tolerance. This term immediately conveys the true meaning of the disease reaction situation whereas the term horizontal resistance does not.

The second type of host:pathogen interaction is a true resistance mechanism which is monogenically controlled. This type of reaction has been termed vertical resistance (13). Plants possessing this type of resistance mechanism when exposed to the pathogen do not react in any macroapparent way. There are undoubtedly numerous chemical reactions which result from such an interaction, but there are no apparent gross differences between an inoculated plant which possesses the monogene for resistance and a noninoculated one. Such plants are not susceptible to attack by *F. oxysporum lycopersici* regardless of environmental conditions. This type of reaction, when properly referred to, is termed mono genic resistance and for tomato is exemplified by the variety Walter (Tables 1, 2 and 3).

Historically, individuals responsible for the development of tomato varieties to control Fusarium wilt have preferred to use monogenic resistance (3, 8, 12, 14). Recently it was proposed that for best results polygenic tolerance should be utilized to control tomato Fusarium wilt (13). If such advice had been followed, it would be impossible to produce tomatoes commercially in Florida at the present time except with strict soil fumigation to control Fusarium wilt. The first report of race 2 in a commercial tomato field was at Delray Beach, Florida, in a field planted to ''Manalucie' and ''Manapal' (9). These varieties are similar to 'Floradel' in that they have the I gene which confers resistance to race 1 Fusarium wilt (4). Manapal and Floradel are susceptible to race 2 because they lack the l_2 gene but more tolerant to race 2 than is Bonny Best, the susceptible nontolerantvariety (Tables 1, 2 and 3). Despite the fact that Floradel and Manapal are highlytolerant to race 2, it is obvious the Florida tomato growers preferred the monogenic resistance as evidenced by the rapid acceptance and popularity of the variety Walter.

The tomato industry of Florida has survived primarily because of the University of florida's extensive and comprehensive tomato breeding program (3). The value of monogenic resistance has been repeatedly demonstrated with the development, release and grower acceptance of 13 tomato varieties possessing monogenic resistance to Fusarium wilt by the University of florida since 1949. The appearance of race 2, eleven years after the practice of growing race 1 resistant varieties was widely used in Florida indicates the value of monogenic resistance. For 11 years no tomato crop suffered reduced yields because of Fusarium wilt. Even though race 2 was first discovered in 1960, it did not become a statewide problem until the late 1960s. By this time varieties with monogenic resistance to race 2 were developed by the breeding program (3, 12) and were rapidly utilized by the tomato industry. The philosophy underlying the 48-year-old University of Florida tomato breeding program has been to utilize monogenic

resistance for disease control whenever possible. The primary reasons for this are: 1) the host reaction to the pathogen is obvious which makes it possible to develop workable screening techniques to produce new varieties in the shortest time period; 2) varieties are not susceptible to the pathogen when released to farmers which results in higher yields than would be obtained with only tolerant varieties; and 3) the spread of the pathogen is curtailed with a resistant variety; whereas, susceptible but tolerant varieties encourage dissemination of the pathogen. The approach to disease control which will be used by the University of Florida tomato breeding program in the future will be the same as in the past. The preferred type of host : pathogen interaction will be monogenic resistance. Polygenic tolerance mechanisms will be utilized only when monogenic resistance is not available, or when the utilization of monogenic resistance mechanisms is not feasible.

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Failure of "horizontal resistance" to control Fusarium wilt of tomato[1]

Pat Crill, John Paul Jones, and D. S. Burgis[2]

ABSTRACT

It was demonstrated that Fusarium wilt incited by race 2 could be, and is, a very serious disease of tomato in second-year sandland production areas of Florida. Tolerance or "horizontal resistance" to race 2 of *Fusarium oxysporum* f. sp. *lycopersici* was found to be of little, if any, value in controlling this disease. It was further concluded that stabilizing selection with respect to race 2 of Fusarium wilt in tomato does not exist as reported by other researchers.

Plant Dis. Reptr. 57: 119-121.

It was recently stated as being unlikely that Fusarium wilt, incited by *Fusarium oxysporum* (Schlecht.) f. sp. *lycopersici* (Sacc.) Snyd. & Hans. race 2, would ever be the menace to tomato (*Lycopersicon esculentum*) crops that race 1 was before the introduction of varieties with the I_1 gene (4). The reasoning given for this statement was that the soil-host-pathogen system is inherently more stable than the host-pathogen system. It was concluded that race 2 had occurred often enough and has had time enough to be common, but that it had not become common because stabilizing selection curbed it. It also was pointed out that stabilizing selection operates in favor of races of the pathogen without unneces-

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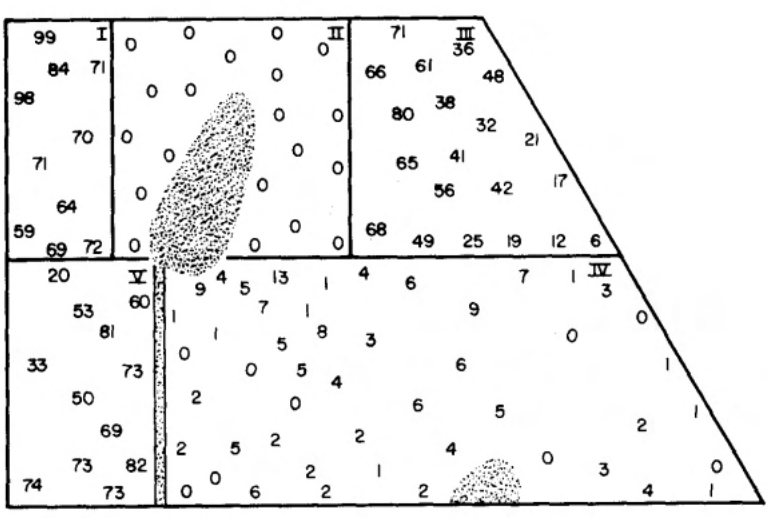
sary virulence and that the stability of monogenic (vertical) resistance depends upon stabilizing selection which can be properly measured only by surveys (4).

Considerable criticism was aimed at plant breeders who are engaged in the development of disease resistant varieties for participating in and promoting what has been termed the "boom and bust cycle of variety production." It was suggested that in order to avoid this boom and bust period the plant breeder should utilize tolerance (horizontal resistance) mechanisms (4).

The objectives of this study were to determine in a commercial Florida tomato field operation (a) the importance of race 2 Fusarium wilt, (b) if "stabilizing selection" against race 2 Fusarium wilt does occur and (c) if tolerance (horizontal resistance) to race 2 Fusarium wilt is of value in tomato varieties.

MATERIALS AND METHODS

A 120-acre commercial farming operation (Fig. 1) typical of Florida sandland stake tomato production was selected for this study. The native vegetation of pine (*Pinus elliottii*) and palmetto (*Serenoa repens*) was removed from the Leon fine sand soil in 1969-70 with conventional land clearing procedures. The first tomato crop was grown during the spring of 1971 and the second crop during the spring of 1972. Normal soil fertilization and pest control programs were practiced by the farmer. About 50% of the acreage (all of field IV) was planted to the variety Walter and the remainder to Homestead 24 in the 1971 season (Fig. 1). In the 1972 season Field II was planted to Walter and the remainder to Homestead 24. This resulted in three fields (I, III, V) having a Homestead 24 following Homestead 24 rotation, one (IV) with a Homestead 24 following Walter rotation and one (II) with a Walter following Homestead 24 rotation. The percentage of plants exhibiting



1. Percent of Fusarium-infected tomato plants and approximate location of each 100-plant sample in each of five fields. The dotted areas represent sloughs and drainage.

Fusarium wilt symptoms was not measured in detail in 1971. In 1972 a comprehensive survey of all five fields was conducted. Approximately 5% of all plants in the 120-acre area were evaluated for presence or absence of Fusarium wilt A total of 102 samples, each consisting of 100 plants, were examined (Fig. 1). Both Walter and Homestead 24 are resistant to Fusarium wilt incited by race 1. Walter is resistant to race 2 while Homestead 24 is susceptible but has been termed horizontally resistant (1).

REULTS AND DISCUSSION

The occurrence of race 2 Fusarium wilt in land which had been cropped only two seasons to the susceptible variety Homestead 24 varied from 43-74% (Table 1). This indicated race 2 Fusarium wilt is as serious a menace to tomato production as race 1 has ever been. The incidence of wilt in Homestead 24 in fields I, III and V was quite high and symptoms were severe when compared with Homestead 24 in field IV and Walter in field II. Crill, et al. (1) demonstrated that Homestead 24 is highly tolerant (possesses good horizontal resistance) to race 2; however, symp tomatic plants in fields I, III and V were stunted, unthrifty, and lacking in vigor and yield. The farmer was able to harvest fields I, III and V only once in 1972, and the yields were reduced by 50% when compared with fields II and IV.

It is obvious from these data that "stabilizing selection'' as reported (4) did not exist with respect to Fusarium wilt of tomato caused by race 2 of *F. oxysporum* f. sp. *lycopersici.* When as high as 74% of the plants in the terminal crop of a rotation consisting of virgin land - highly tolerant variety - highly tolerant variety are infected with race 2 and when race 2 Fusarium wilt is found in 14 of 19 surveyed fields (2), no claim for stabilizing selection concerning race 2 can be considered valid. From these studies it can be concluded that tolerance (horizontal resistance) to race 2 Fusarium wilt, as exemplified by Homestead 24, is not worthy of consideration in a tomato breeding program.

Fourteen rules Concerning the use of monogenic (vertical) resistance have been formulated by Robinson (3). The results of this field study are applicable to two of these in that they lend support to rule eight and fail to confirm rule four. Rule eight states, "Crop patterns of vertical resistance in time are valuable chiefly against simple interest diseases." The low incidence of Fusarium wilt in field IV lends considerable support to this rule because growing the race 2 resistant

Table 1. Percentage of race 2 Fusarium-infected plants in five tomato fields in Florida with three different host rotations.

Field no.	Host rotation		% infected 1972	No. samples	No. acres
	Cultivar 1971	Cultivar 1972			
I	Homestead 24	Homestead 24	74	10	10
II	Homestead 24	Walter	0	20	20
III	Homestead 24	Homestead 24	43	20	25
IV	Walter	Homestead 24	3	50	50
V	Homestead 24	Homestead 24	62	12	15

variety Walter in 1971 very effectively reduced the amount of disease in the susceptible but tolerant variety Homestead 24 the following season (Table 1). Rule four states, "Vertical resistance is unlikely to be valuable when the host population is genetically uniform and is grown in large acreages of a single cultivar." The data presented here coupled with the fact that the race 2 resistant variety Walter was grown on over 75% of the Florida tomato acreage does not lend credence to rule four.

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Prevalence of the vertifolia effect in the Fusarium wilt disease of tomato [1]

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ABSTRACT

Thirty-six tomato varieties developed by seven State experiment stations, two Federal experiment stations, and five commercial seed and food processing companies were evaluated for tolerance and resistance to *Fusarium oxysporum lycopersici* and the occurrence of a vertifolia effect. It was concluded the vertifolia effect was not as inevitable as portrayed and perhaps there are more exceptions to the rule than there are cases to support it.

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Robinson (4) defined the vertifolia effect as the loss of horizontal resistance during the process of breeding for vertical resistance. In more conventional terminology this can be stated as the general loss of tolerance to a given pathogen by a plant variety which was developed with specific monogenic resistance to the Pathogen (3).

Van der Plank(7) coined the term vertifolia effect to explain the loss of tolerance in the potato variety Vertifolia to *Phytophtora infestans.* Vertifolia was a variety developed with monogenic resistance to *P. infestans.* A race of *P. infestans* evolved which could successfully attack the monogenic resistance of Vertifolia and the result was severe late blight. From this, it was concluded the genes for tolerance to late blight had been lost in the development of the variety Vertifolia.

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The development of the vertifolia effect concept was based primarily on this single observation. Van der Plank concluded "a vertifolia effect seems to be almost inevitable wherever resistance is needed and vertical resistance is great" and "great selection pressure and great vertical resistance are needed for a great vertifolia effect" (7).

It was also pointed out that the I gene which controls resistance to *Fusarium oxysporum* (Schlecht.) *lycopersici* (Sacc.) Snyd. & Hans. race 1 in tomato is a strong gene which confers great vertical resistance, resulting in great selection pressure (7). The development and isolation of race 2 of *F. oxysporum lycopersici* and the subsequent location of a resistance gene (6) provided a system of measuring the vertifolia effect in tomato with respect to Fusarium wilt. Previous work with two races of *F. oxysporum lycopersici* and two resistant tomato genotypes (2, 3) has indicated the absence of a vertifolia effect in this host-pathogen interaction. This study was designed to determine whether a vertifolia effect did exist in Fusarium wilt resistant tomato varieties developed by seven State experiment Stations, two USDA tomato breeding programs and five commercial seed and processing tomato companies.

MATERIALS AND METHODS

Seed of 36 tomato varieties was sown in flats of steamsterilized amended soil (1 part Myakka fine sand : 1 part peat; v : v). After seeding, all flats were placed in a growth chamber where the temperature was maintained at 80° F during 12 hours of light (1000 ft-c) and 70° F during 12 hours of darkness. Ten days after sowing, seedlings were uprooted and the excess soil shaken from the roots before they were washed momentarily in running tap water to remove adhering soil particles. Roots were then immersed for 10-30 sec in inoculum and aseptically transplanted into steam-sterilized flats of soil identical to that of the seedling flats.

Inoculum of races 1 and 2 of Fusarium oxysporum lycopersici was produced by growing the fungi on petri plates of potatodextrose agar (PDA) for 10 days at 82°F with continuous light of 150 ft-c. The race 1 culture originated from successive single spore isolations which had been tested for pathogenicity and virulence and maintained in soil tubes under refrigeration. The race 2 culture was derived from a wild type isolate which also had been tested for pathogenicity and virulence and maintained in soil tubes. Isolates of race 1 and race 2, respectively, were 621-1-1-BK and 43-BK-BK. The contents of the plates were placed in a microblender with a small amount of sterile distilled water and briefly comminuted to produce a thick inoculum suspension. Aseptic techniques were used to avoid any cross contamination during the preparation of inoculum. Average spore concentrations (ignoring hyphal fragments) were measured as 9.0×10^6 and 8.25×10^6 spores/ml, respectively, for race 1 and race 2. Twenty plants of each variety were transplanted without inoculation to serve as comparison controls. Flats of plants were placed in a plant production house in a split-plot design (races = whole plots; varieties = subplots) with 5 replications on October 16,1972 and maintained at ambient conditions. All equipment, as well as the hands of the people doing the transplanting, were scrubbed with 70% ethanol to avoid cross

contamination of inoculum. Each treatment consisted of 20 plants of each of the 36 varieties inoculated with each race. The high, mean and low temperatures during the time of the experiment were respectively, 85, 75 and 64°F. Disease readings were made, as described previously (3), IO and 20 days after inoculation. Inoculated plants were evaluated as (a) percent diseased, (b) percent dead, and (c) disease index. Disease index was determined by: numerically rating each seedling according to symptom development, summing the ratings, and dividing by the total number of seedlings evaluated to obtain an average rating.

RESULTS AND DISCCISSION

No disease developed in noninoculated plants. This study confirmed an earlier report (3) that insofar as evaluation of screening tests are concerned there is little difference among the three methods. Ranking of varieties with respect to disease severity was identical with all three methods in this experiment; therefore only disease index results are presented (Table 1).

None of the so-called immune varieties (those having the I gene), excluding Walter and Florida MH- 1, was free from wilt symptoms when inoculated with either race 1 or race 2. The three susceptible varieties which are assumed to have no genes for tolerance to either race 1 or race 2 are Earliana, Highlander and Bonny Best. The disease index average for race 1 and race 2, respectively, on these three varieties after 20 days was 3.28 and 3.25 (Table 2). For the tolerant varieties (Pritchard, Grothens Globe and Marglobe) index averages were 2.58 and 2.30 (Table 2). Although the disease index for Pritchard inoculated with race 1 was 2.93, the variety was placed in the tolerant group because it has long been recognized as a tolerant variety (1) and because of its race 2 reaction (Table 2).

To determine whether a vertifolia effect has occurred with respect to Fusarium wilt of tomato two references are necessary: a) the disease index range for susceptible varieties and b) the disease index range for tolerant varieties (Table 2). The disease index range for any variety being considered must be compared with these two references. When the race 2 Fusarium wilt index of any variety is established and the index does not exceed the maximum for tolerant varieties, then it can be concluded a loss of tolerance genes did not occur. If the index of the variety in question does exceed the maximum range established for tolerant varieties it can be concluded that a loss of tolerance genes did occur. For the vertifolia effect to have occurred, a variety containing the I gene must be more susceptible to race 2 than the tolerant varieties.

When the 30 varieties resistant to race 1 were analyzed for their reaction to race 2 only 10 of these (Roma (2.98), Manasota (3.11), Manapal (3.17), Tropi-Red (3. 19), Tropic (3.09), Healani (3.60), Atkinson (3.17), El Monte (2.96), Campbell 17 (3.24) and Campbell 19 (3.17) exceeded the maximum disease index for tolerant varieties of 2.75 (Table 1). Only one variety, Healani, exceeded the mean disease index of 3.25 which was established for the susceptible varieties. There was a definite lack of tolerance genes to race 2 of *F. oxysporum lycopersici* in Healani and an apparent deficiency of lesser significance in the other 9 varieties which exceeded the maximum disease index range for tolerant varieties. These results

must be interpreted with the knowledge that the isolates of the two races used as inoculum were selected for maximum virulence. If a less virulent isolate had been used a lower index for tolerance varieties could possibly have occurred.

The loss of tolerance genes to race 2 did not occur in most of the race 1 monogenic resistant tomato varieties evaluated in this study as was suggested by general statements of Robinson (4,5), and Van der Plank concerning the vertifolia effect (7). Two-thirds of the varieties containing the I gene (resistant to race 1) were as tolerant to race 2 as the polygenic race 1 tolerant varieties, demonstrating that the vertifolia effect certainly did not occur uniformly in all tomato breeding

Table 1. Host response as measured by disease indices at two dates for 36 tomato varieties inoculated with two races of *Fusarium oxysporum lycopersici.*

Variety	Developer	Date released	Mean Fusarium Wilt Index[a]			
			Race 1		Race2	
			10 days	20 days	10 days	20 days
Marglobe	USDA-ARS	1925	1.52	2.75	0.99	2.75
Pritchard	USDA-ARS	1931	1.61	2.93	0.36	1.99
Roma	USDA-ARS	1955	1.04	7.86	1.20	2.98
Homestead 24	USDA-VBL	1952	0.51	0.58	0.47	2.23
Homestead 61	USDA-VBL	1952	0.42	0.58	0.46	2.18
Homestead 600	USDA-VBL	1952		0.48	0.19	1.48
Highlander	USDA & Colo AES	1968	0.98	3.40	1.18	3.52
Manasota	Florida AES	1949	0.57	0.96	0.80	3.11
Manalucle	Florida AES	1953	0.37	0.81	0.85	2.45
Indian River	Florida AES	1961	0.40	1.11	0.25	2.06
Manapal	Florida AES	1961	0.76	1.12	1.57	3.17
Floralou	Florida AES	1962	0.46	0.99	0.32	2.25
lmmokalee	Florida AES	1964	0.51	1.12	0.55	2.53
Floradel	Florida AES	1964	0.76	0.84	0.45	2.08
Tropi-Gro	Florida AES	1967	0.25	0.74	0.61	2.19
Tropi-Red	Florida AES	1967	0.30	0.66	1.16	3.19
Tropic	Florida AES	1969	0.40	0.84	0.91	3.09
Walter	Florida AES	1969	0.02	0.03	0.00	0.00
Florida MH-1	Florida AES	1971	0.08	0.08	0.02	0.09
Healani	Hawaii AES	1967	0.26	1.21	1.69	3.60
VF 145	California AES	1961	0.10	0.21	0.30	2.08
Atkinson	Alabama AES	1966	0.42	1.20	1.28	3.17
El Monte	Texas AES	1966	0.19	0.62	0.54	2.96
Marion	S. Carolina AES	1961	0.33	0.74	0.77	2.29
Earliana	Johnson & Stokes	1900	1.01	2.74	0.82	2.59
Campbell 17	Campbell Soup Co.	1958	0.10	0.70	1.01	3.24
Campbell 19	Campbell Soup Co.	1967	0.21	1.28	0.79	3.17
Campbell 28	Campbell Soup Co.	1971	0.31	0.56	0.44	1.65
Globemaster	W. A. Burpee Co.	1962	0.23	1.33	0.18	1.51
Basket-Pac	W. A. Burpee Co.	1963	0.69	2.16	0.27	1.35
VF Hybrid	W. A. Burpee Co.	1968	0.24	1.17	0.49	2.42
Bonus VFN	Peto Seed Co.	1967	0.19	0.45	0.59	2.65
Jefferson	AsgrowSeed Co.	1948	0.22	0.38	0.18	1.64
Supermarket	Asgrow Seed Co.	1963	0.01	0.19	0.10	1.58
Bonny Best	?	1908	1.96	3.69	1.18	3.65
Grothens Globe	?	?	0.62	2.05	0.48	2.14
LSD .05			0.060	0.096	0.085	0.108

[a] 0 = no disease, 4 = dead; no symptoms occurred on uninoculated control plants.

Table 2. Disease indices from susceptible and tolerant varieties inoculated with two separate races of *F. oxysporum lycopersici* and evaluated after 20 days.

	Disease indices [a]	
Susceptible tomato varieties	Race 1	Race 2
Earliana	2.74	2.59
Highlander	3.40	3.52
Bonny Best	3.69	3.65
Average Disease Index	3.28	3.25
Tolerant varieties		
Grothens Globe	2.05	2.14
Pritchard	2.93	1.99
Marglobe	2.75	2.75
Average Disease index	2.58	2.30

[a] 0 = healthy, 4 = dead.

programs, and most programs, as evaluated in this study, did avoid the loss of tolerance genes.

Plant breeders have long been aware of the problems and difficulties associated with the manipulation of polygenically controlled characters and the theory of vertifolia effect does little, if anything, to elucidate these problems.

These data support the theory that the 10 varieties which exhibited less tolerance than the range established for known tolerant varieties never possessed such tolerant genes. Parental stocks into which the I gene was introduced to produce the 10 varieties were probably not tolerant to race 1. There is no requirement and no scientific basis for assuming that all varieties which contain the I gene also contained tolerance genes. Likewise, there is no evidence to suggest that varieties which contain the I gene do not contain the tolerance genes. We suggest the vertifolia effect, if real, is not as inevitable as portrayed and perhaps there are more exceptions to the rule than there are cases to support it.

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Evaluation of some concepts of variety development and disease control with host resistance[1]

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ABSTRACT

Results previously published by the authors are summarized with respect to the imposition of federal controls on breeding programs. Some of the evidence cited by pro-control advocates of variety development programs was not confirmed in studies using the *Lycopersicon : Fusarium* host : pathogen disease model. Four widely accepted theories of disease control using host resistance are discussed.

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Established methods and objectives of variety development and plant breeding have recently been questioned (1,2,7,11). In 1968, Van der Plank reprimanded plant breedersfor using monogenic resistance and recommended that polygenically controlled tolerance mechanism be utilized for disease control (11). He further recommended that federal controls be placed on plant breeders and the use of certain genes be restricted. In 1972, the National Academy of Sciences published its view in a report entitled Genetic Vulnerability of Major Crops (7). Much of this report was based upon the unproven axioms developed by Van der Plank (11) and Robinson (9,10). The committee responsible for the report of the National Academy of Sciences also advocated establishing a national committee

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to regulate the activities of plant breeders (7, page 299). The committee suggested "the establishment of a national monitoring committee to keep a watchful eye on the development and production of major crops . . . and issue warnings wherever and whenever it feels them justified." Clearly this was a recommendation for the regulation and control of the activities of plant breeders and seed companies.

The widespread acceptance of proposed, but unconfirmed, theories of variety development and the recommendations by the proponents of such theories for federal controls and restriction of genes available to plant breeders stimulated the research summarized and discussed in this report.

The purpose of this paper is to discuss those recent theories which have been advanced and are now considered by many to be axiomatic. Four concepts considered basic to the overall theory of disease control with variety development based upon host resistance were presented in 1968 (11) and illustrated with the Fusarium wilt disease of tomato as follows:

A The I gene which controls resistance to race 1 *Fusarium oxysporum lycopersici* is a strong gene (11, page 1 14).

B. Race 2 of *F. oxysporum lycopersici* has arisen many times by mutation or other means, but never became established because stabilizing selection curbed it (11, pages 114-115).

C. Those tomato varieties developed with monogenic vertical resistance to race 1 *F. oxysporum lycopersici* should be “intensely susceptible” to race 2 *F. oxysporum lycopersici* because of the vertifolia effect (11, pages 153-159).

D. Horizontal resistance (polygenic tolerance) is preferred to vertical resistance (monogenic resistance) as a genetic mechanism for controlling plant dis eases (11, pages 129-143).

Concept A. The I Gene is a Strong Gene:

Van der Plank stated that the I gene is strong, but not quite strong enough (11, page 114) and concluded that race 2 would never become the menace race 1 was before the gene I was used. In this statement it was implied that all Fusarium wilt prior to the introduction of varieties with the I gene was incited only by race 1. On the next page this statement was contradicted as follows: “Alexander and Tucker‘s discovery of race 2, in a variety without the I gene . . . is evidence enough that race 2 had occurred tens of thousands, possibly millions of times. To dispute this is to assume that providence staged a special and unique show for Alexander and Tucker” (11, page 1 15).

Prior to the release, and utilization by farmers, of varieties with the I gene, it was not possible to determine whether wilt was caused by race 1, race 2, or any other race. This is because it is not possible to identify races unless there are at least two tomato genotypes which will differentiate one *Fusarium* isolate from another as being genetically unique for pathogenicity. Therefore, the statement that race 2 would not be the menace race 1 was before the gene I was used was invalid when written because it was based upon contradictory and improperly interpreted data. The further statement that because the gene I is a strong gene it is unlikely to become useless to tomato growers (11, page 114) is also incorrect. Crill, et al.

have shown repeatedly (3,4,5) that the I gene is not effective against race 2, and without the I_2 gene Fusarium wilt caused by race 2 is the limiting factor in tomato production on Florida sand land. The statement that race 2 is not the menace that race 1 was before the gene I was used may be true, but impossible to prove. If the statement is true, it is because varieties resistant to both race 1 and race 2 have been developed. introduced and cultivated on virtually 100% of Florida's tomato land since 1970.

Plant breeders have been aware that some genes for resistance were more effective than others ever since the first diseaseresistant crop varieties were specifically developed by Orton. The terminology used by plant breeders and pathologists to explain the effectiveness of control has involved words and phrases such as: immune, highly resistant, moderately resistant, field resistant, and so forth. The usefulness of disease resistance genes has been measured by how long they remain effective when placed into varieties and released for commercial use. The "strong gene - weak gene" concept does not provide any better means of predicting the useful time span of a resistance gene than other procedures plant breeders were using previously. When the I gene was first incorporated into commercial tomato varieties, it was not possible to predict how long it would be effective against Fusarium wilt. The same situation was true for the gene controlling resistance to race 2 Fusarium wilt, gray leafspot, and nailhead rust in tomato. These resistance genes have all been very effective and long-lived. They would all have to be classified as strong genes based upon our present knowledge, but at the time they were first utilized by the breeder the length of time they would be effective was impossible to determine. The most probable reason these genes were effective is that suitable mechanisms of variation did not exist in the pathogen whereby it could adapt to the host population with the resistance gene. The formation of new races is most likely an interaction among both host and pathogen genes and not just a function of the host as implied by the "strong gene -weak gene'' concept.

Concept B. Stabilizing Selection Curbed Race 2:

The concept of stabilizing selection as presented by Van der Plank is a proposal to explain the selection and survival of pathogen races in nature. Stabilizing selection states that simple races are more fit to survive than complex races, or race 1 *Fusarium oxysporum lycopersici* is more fit to survive than race 2 *F. oxysporum lycopersici.* The presumed reason for race 1 surviving better than race 2 is that race 1 is a more simple race, that is, it has fewer genes for pathogenicity than race 2. Quite likely the only difference between race 1 and race 2 is a single gene which controls the ability of the fungus to cause disease in varieties with the I genes. Because no perfect stage exists for *F. oxysporum lycopersici* the genetic differences between race 1 and race 2 have not been determined; however, no evidence is available to suggest more than one gene. Crill, et al. (6) have assumed that virulence (measured by degree of disease development on a wide range of host genotypes among isolates within a single race of the pathogen) is polygenically controlled, but that pathogenicity (difference between race 1 and 2 in ability to cause disease on differential varieties with the I genes) is monogenically

controlled. We do not think that just because a fungus has one more gene for pathogenicity than another it is better adapted to survive, either as a saprophyte or a parasite.

In 1968 Van der Plank stated ''race 2 has occurred often enough and has had time enough to be common, had stabilizing selection not curbed it" (11, page 115). Crill, et al. (4) have offered evidence to refute this statement. They studied Fusarium wilt developing in a commercial tomato farming operation of 120 acres over a 3-year period and concluded that there was no evidence of stabilizing selection. It was not possible to duplicate the field conditions exactly as specified as being necessary (11). To test this hypothesis adequately it would be necessary to grow varieties that are susceptible to both race 1 and race 2 on a commercial scale for several years in soil uniformly infested with equal amounts of race 1 and race 2. At the termination of the experiment the amount of race 1 would be compared with the amount of race 2, and if race 1 was significantly predominant, stabilizing selection could be assumed operative. Such an experiment is not feasible because (i) no tomato farmer is going to grow a suitable large acreage of a wilt-susceptible variety, (ii) a method of uniformly infesting soil with race 1 and race 2 in equal amounts has not been developed, and (iii) no assay technique is available to differentiate race 1 from race 2 on the large scale that would be necessary. The concept of stabilizing selection is so defined and worded that it is nearly impossible to disprove. Any data that are anti the stabilizing selection hypothesis can be dismissed by invoking the "weak gene" philosophy (8). Inasmuch as much of the evidence for the stabilizing selection theory was based on the Fusarium wilt of tomato interaction and on the stated fact that the I gene is a strong gene, it becomes much more difficult to defend stabilizing selection by invoking the "weak gene" philosophy as discussed by Nelson (8).

There is little doubt that the phenomenon termed "stabilizing selection" does exist. Many plant breeders and pathologists have observed that some races are more predominant than others. They have also noted that race formation varies from species to species of pathogens. Van der Plank termed this phenomenon stabilizing selection and noted that those races most fit to survive are those with the fewest genes for pathogenicity. Nelson (8) has recently questioned the validity of the stabilizing selection hypothesis and concluded that the concept itself may not really exist. Hare (comments at the discussion session entitled "Stabilizing Selection: A Controversy' presented at the 1972 annual meetings of the American Phytopathological Society) has indicated that the sole difference among races 1, 2 and 3 of Fusarium wilt of pea, excluding their different genes for pathogenicity, is their rate of growth. Each race presumably has the same number of genes for pathogenicity, yet in mixed cultures race 1 always predominates over race 2 and race 2 always over race 3. Growth rate rather than the number of genes for pathogenicity determined which race predominates. Until some evidence is offered to associate growth rate with a single specific gene for pathogenicity, it would appear that the phenomenon of stabilizing selection cannot be explained by presence or absence of genes for pathogenicity.

Concept C. The Vertifolia Effect Occurs in the Lycopersicon : Fusarium System: The vertifolia effect with respect to breeding tomatoes for resistance to Fusarium

wilt has been discussed by Crill, et al. (4). Robinson (9) defined the vertifolia effect as the loss of horizontal resistance during the process of breeding for vertical resistance. In tomato breeding jargon this translates as the general loss of tolerance to a given pathogen by a host plant variety which has been developed with specific monogenic resistance by the plant breeder (5). The term "vertifolia effect" was coined to explain the loss of tolerance in the potato variety Vertifolia to *Phytophtora infesbns.* 'Vertifolia' was developed with monogenic resistance to *P. infestans.* A race of *P. infestans* evolved which could successfully attack the monogenic resistant 'Vertifolia', and the result was severe blight symptoms. From this incident it was concluded that the genes for tolerance to late blight had been lost by the plant breeders in the development of 'Vertifolia'. Van der Plank stated "a vertifolia effect seems to be almost inevitable wherever resistance is needed and vertical resistance is great" (11, page 159) and "great selection pressure and great vertical resistance are needed for a great vertifolia effect" (11, page 155).

In a series of experiments designed specifically to test for the vertifolia effect, Crill, et al. (4) concluded that the vertifolia effect did not necessarily operate in the Lycopersicon: *Fusarium* system. They evaluated 36 tomato varieties which were developed by 15 different breeding programs including commercial seed companies, food processors, State and Federal experiment stations. A definite loss of tolerance was noted in one variety and a possible loss in nine others.

When they compared the monogenically resistant (vertically resistant) variety Floradel, which had the gene I, with the tolerant (horizontally resistant) varieties Marglobe and Rutgers, in all instances Floradel had less disease from race 2 than did Marglobe and Rutgers. They suggested that the vertifolia effect concept was not valid in the Lycopersicon : Fusarium host : pathogen interaction because there were far more exceptions to the concept than instances to support it (4).

Most plant breeders working with disease resistance have experienced the loss of resistance or tolerance in certain breeding lines. It is, in fact, a rather common occurrence, especially if screening techniques employed by the plant breeder are not fully adequate. The most common explanation of loss of resistance is that the plant selected from the screened population was not a resistant plant but rather an escape. In those breeding programs where progeny testing is routinely conducted, it is usually possible to determine how often susceptible escapes were selected as being resistant. In those programs where screening programs are inadequate, or where progeny testing is not done routinely, it would be quite easy to lose resistance genes, particularly those associated with polygenic tolerance or horizontal resistance. It is not a foregone conclusion that tolerance genes will be lost when the plant breeder is concentrating on monogenic resistance as has been claimed (11). Rather, the possibility does exist that tolerance genes will be lost when the breeder is concentrating on monogenic resistance unless he makes an effort not to lose such tolerance genes.

Concept D. Horizontal Resistance is Preferred to Vertical Resistance for Disease Control:

This concept is the basic theme of Van der Planks thesis (11). He stated "in certain circumstances . . . . . vertical resistance ought to be as stable and enduring as horizontal resistance" (11, pages 88-90) and "stability of vertical resistance

depends on stabilizing selection" (11, page 98). "The stability of horizontal resistance is ascribed to polygenic inheritance of aggressiveness and genetic homeostasis" (11, page 119). He concluded that horizontal resistance is more stable than vertical resistance and this stability is attributed to the stability of the races of the pathogen, and furthermore, this stability does not exclude new races from appearing or old races from disappearing, but a stable balance is maintained among all the various races (11, page 122). The facts obtained from the *Lycopersicon : Fusarium* system thus far do not support the theory that horizontal resistance, because of stability, is superior and therefore preferred to vertical resistance (3, 4, 5, 6) in a system of intensive agriculture.

Crill, et a1 (3) have shown in field studies of commercial acreages of tomatoes when highly tolerant varieties (possessing good horizontal resistance) are compared with varieties that are monogenically resistant (vertically resistant), the monogenic resistance is superior. Yields from monogenic-resistant varieties were much greater than those from tolerant varieties. Yield data from the crop rotations involving tomato varieties both tolerant (horizontally resistant) and resistant (vertically resistant) to Fusarium wilt were compared. Yields of the tolerant variety-tolerant variety rotation were reduced by one-half in the second crop season compared with the resistant variety-tolerant variety rotation (4).

Crill, et al. (5) have discussed the advantages and disadvantages of monogenic resistance versus polygenic tolerance. They cited several reasons for plant breeders to use monogenic resistance in preference to tolerance, or vertical resistance in preference to horizontal. These included the following: (a) the host reaction to the pathogen is quite obvious with monogenic resistance, which makes it possible for the breeder to develop workable screening techniques in the shortest time period, (b) monogenic-resistant varieties are not susceptible to the pathogen when released to the farmer, which results in higher yields than would normally be obtained with only tolerant varieties, (c) the spread of the pathogen is curtailed with a monogenic-resistant variety; whereas, tolerant but nevertheless susceptible varieties encourage dissemination of the pathogen.

They did not mention the very serious problem that a plant breeder would confront if he utilized only horizontal resistance (polygenic tolerance) and was working with multiple disease resistance. From the plant breeders' viewpoint, the most serious objection to the use of horizontal resistance (polygenic tolerance) rather than vertical (monogenic) resistance is that it is unmanageable. If it is assumed the breeder has five unlinked monogenes which control resistance to five diseases, it is obvious he must screen very large populations of recombinant progeny if he is ever to find a plant that is resistant to all five pathogens. Not only must the breeder find the one plant, he must find numerous others, some of which must possess desirable horticultural characters. In most breeding programs this objective would not be rapidly accomplished because of the sheer numbers of progeny which must be evaluated. If the progeny that are resistant to the five diseases are evaluated, they will be found to segregate, and in the simplest of cases would have to be grown for at least three generations to establish a fixed line. In all probability these homozygous resistant inbred lines which are resistant to all five diseases will be deficient in horticultural characters. To improve the plant type,

a series of backcrosses must be initiated, which unfixes the homozygous state of the five disease resistance genes and the same involved screening and selection process for all five diseases must be gone through again by the breeder.

In the above discussion it was assumed that the five monogenes were dominant, dominance was complete and resistance was not associated with any undesirable characters. Such conditions are unlikely to exist in nature and even with monogenes, the procedure of variety development is going to be long and tedious. If, however, the plant breeder receives a dictate from a Federal control agency that he must use only horizontal resistance (polygenic tolerance), the problem of disease control with host resistance truly becomes unmanageable. If it is now assumed the same five diseases will be controlled by horizontal resistance and a minimum of three genes control each character, the breeder must keep track of at least 15 genes. Most polygenic tolerance mechanisms behave like Fusarium wilt tolerance in tomato, that is, there is usually a lack of dominance and resistance is additive. Also, to detect such resistance, it is of utmost necessity to control the environment to obtain desired pathogenesis (6) and utilize the proper inoculum potential (5, 6). This requires rather elaborate plant pathogenic techniques for just one disease and it becomes impossible to evaluate a single plant simultaneously for five separate diseases, each of which has its own special climatic conditions for optimum development including temperature, pH, day length, and so forth. In addition, it is also extremely important to have the isolate of the pathogen that possesses the necessary genes for pathogenicity. When dealing with monogenic resistance (vertical resistance), the only important criteria are to have adequate inoculum and that it be pathogenic and then resistance is either present or absent with the monogene. With polygenic tolerance (horizontal resistance) the host-pathogen interaction varies from slightly susceptible to severely diseased or dead. The facilities necessary to synthesize an agronomically or horticulturally desirable plant which possesses the maximum in horizontal resistance to five diseases are incomprehensible.

SUMMARY AND CONCLUSIONS

Four concepts developed by Van der Plank (11) and illustrated with Fusarium wilt of tomato are discussed. The observations made by Van der Plank are not disputed; rather, alternative explanations of these same observations are presented, based upon experience with the genetics and breeding of multiple disease-resistant tomato varieties. Hopefully, those who are advocating the placement of controls on plant breeders and seed companies and the monitoring of variety development activities will realize that there usually exists more than one explanation for many of the phenomena associated with disease control via host resistance. The ultimate conclusion that we have drawn is that scientific information is not yet available whereby any governmental or control agency can dictate to the plant breeder how he should develop varieties. The government-dictated policy of plant breeding in the U. S. S. R. served to stifle variety development in Russia. The dictates based upon the faulty theories of one man resulted in retarded variety development programs in Russia and, according to some, contributed much to

the present worldwide shortage of food grains. If the National Academy of Sciences Committee on the Genetic Vulnerability of Major Crops and other federal groups are successful in their attempts to establish a national board of control over plant breeders, seed companies and food processing companies, the same could happen to variety development in the United States.

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Host resistance and disease control in tomato[1]

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ABSTRACT

Host resistance through variety development has long been recognised as usually the most economical means of disease control. This is especially true with short-lived annual crops if the pathogen involved does not consist of numerous physiologic races. Pathogens which predominate on tomato in the Caribbean for the most part consist of usually only one or a very few races. Results of several surveys of tomato production areas in the Caribbean indicated development of multiple disease resistant varieties adapted to local conditions was feasible and offered a practical means of control while increasing production.

The problem in developing varieties for the Caribbean depends upon where and how they are to be utilized by the farmer. The ability to set fruit under hot, humid and otherwise adverse environmental conditions is of prime importance. In some areas of the Caribbean where tomatoes are grown all the techniques of modem agriculture are utilized including adequate irrigation, high levels of fertilizer, good soil management, and good disease, insect and weed control. In other areas of the Caribbean only the most primitive methods are used in tomato production. With

[1] Crop Protection in the Caribbean. Proceedings of a Symposium on the Protection of Horticultural Crops in the Caribbean held at Department of Crop Science. The University of West Indies, St. Augustine, Trinidad. April 811, 1974.

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such primitive methods, only primitive varieties will perform suitably. A variety which is high yielding under good or excellent farming practices will yield almost nothing when placed in conditions of primitive agriculture. Like wise, those varieties which perform best in primitive conditions do not have the capability to utilize effectively the benefits of increased pest control, coupled with adequate fertilizer and irrigation. Varieties developed for use in the areas of primitive agriculture must of necessity be considerably different from those developed for use in areas where the most advanced agricultural practices are utilised.

This programme has concentrated on the development of varieties which have the ability to set fruit under the environmental stresses of high temperature and high humidity and also respond to the use of modern agricultural practices by producing high yields of high quality fruit.

INTRODUCTION

Host resistance via variety development has long been recognized as usually the most economical means of disease control. This has been especially true with short-lived annual crops if the pathogen involved does not consist of numerous physiologic races.

Tomato production in the Caribbean has historically been inconsistent and sporadic. The purpose of the studies described in this report was to evaluate Caribbean tomato production and determine the causes of often poor production.

HISTORY OF U.W.I./U.F.* COOPERATIVE TOMATO PROGRAMME

The cooperative tomato variety development programme which now exists between the University of the West lndies and the University of Florida was initiated very informally in 1970 by Dr. L. H. Purdy of the University of Florida, Mr. Felix Mederick of the St. Lucia Department of Agriculture, Mr. Rex Frederick and Mr. Percy Arthurton of the Montserrat Department of Agriculture and Mr. Brian Honess of the British Ministry of Overseas Development in Montserrat. Dr. St. Clair Forde of the University of the West lndies and a commercial tomato farmer in Antigua, Mr. Gardiner, soon became involved in the initial project. Results of experiments in Montserrat and Antigua were utilized in conducting variety trials in Haiti as requested by Mr. David Warren of the U. S. Embassy in Haiti and Haitian tomato growers, Mr. Estime and Mr. Fink

The cooperative effort generated considerable data and information but because of personnel transfers and changes, no measurable progress in variety

*U.W.I. = University of the West Indies; U.F. = University of Florida.

development occurred. In 1973, Mr. Egbert Tai of the Crop Science Department of the University of the West Indies, with assistance from Dr. L. H. Purdy, agreed to act as coordinator of the cooperative tomato project and extensive cooperative studies were begun by Dr. Ralph Phelps and Mr. Winston Charles in Trinidad and Dr. Pat Crill in Florida. All major cooperative studies between the University of the West Indies and University of Florida relating to the tomato project are presently conducted in Trinidad with accessory field trials elsewhere in the Caribbean coordinated by the Crop Science Department of the University of the West Indies.

OBJECTIVES OF THE U.W.I./U.F. TOMATO PROGRAMME

The original objective of the cooperative programme was for the University of Florida tomato breeding programme to supply advanced lines to different locations in the Caribbean for adaptability and performance evaluations. It was soon obvious that conditions of crop production in the Caribbean were so diverse and different that a more organized effort was required for development of a successful Caribbean variety. The major objectives of the current programme can be briefly described.

Development of varieties for use in the Caribbean:
Successful Caribbean varieties will probably be different from those which are successful in Florida. The primary use of tomatoes in the Caribbean will be for local consumption with possibly a limited amount of inter-island trade and export. Exports to North America, given present conditions, are not considered feasible.

Attention is being given only to varieties which will be grown in intensive agriculture. No effort is being made concerning varieties for use in subsistence agriculture. It does not appear possible to achieve the maximum adaptability within a single variety since two distinct seasons occur throughout the Caribbean and it appears a wet season variety as well as one for the dry season will be required.

The successful Caribbean tomato variety will differ from the Florida varieties in that (i) extremely large fruit are not required in the Caribbean as in the U.S.; (ii) fruit are sold by the pound with no grade requirements in the Caribbean, except that no breaks or rots are allowed; (iii) a dual purpose fresh market-processing fruit is considered desirable for the Caribbean by local industry; and (iv) the shipping qualities SO necessary for Florida tomatoes are not required for varieties in the Caribbean.

The specific characteristics which are being concentrated on for Caribbean varieties are (a) maximum yields of fruit per unit production cost, (b) suitable taste, texture and flavour for local consumption, (c) concentrated fruit set, (d) early maturity, and (e) multiple disease resistance. The producer of tomatoes in the Caribbean should be able to consistently produce a good crop with the minimum amount of labour and pest control using varieties with these characteristics.

Isolation of breeding lines with the *hot-set* characters:
The normal tomato or usual tomato variety supposedly does not set fruit when night temperatures are above a certain minimum. The hot-set characteristic is

expressed in breeding lines which set fruit under high night and day temperatures. Recently we have emphasized fruit set under conditions of high relative humidity as well as high temperatures. This characteristic can be screened for in Florida during the summer and almost year round in the Caribbean.

The *hot-set* characteristic has been isolated and is being incorporated into breeding lines for the Caribbean as well as Florida. The ultimate goal with respect to the Florida industry is to develop a variety which can be produced in the summer and thereby transform Florida into an area of year around production which should assist in stabilizing the industry.

Isolation of new genes for use in Florida lines:
In addition to the hot-set characteristic, several others can be screened for most effectively in the Caribbean. These include disease resistance, insect resistance, yield and yield component factors, fruit size and fruit firmness in hot weather, shipping quality and numerous others. These characters for the most part can be better evaluated in the Caribbean environment than anywhere else and in general these characteristics are just as valuable for future Florida varieties as for Caribbean varieties.

Monitoring current varieties for new diseases and pests:
The National Academy of Sciences as well as other groups have pointed out the desirability, even the necessity, of maintaining breeding programmes and pest monitoring programmes in the tropics. The cooperative U.W.I./U.F. programme provides the University of Florida an economical and effective way to monitor current tomato varieties for new diseases and pests. All breeding lines and varieties are evaluated throughout the growing season for disease and pest problems. Thus far, no new diseases have been recorded which were not already in Florida, but if one is found this would trigger an immediate response and, hopefully, effective control measures would be developed before the disease could become prevalent in Florida.

TOMATO DISEASE PROBLEMS IN THE CARIBBEAN

Although disease control is properly a part of production problems, diseases are of such importance on tomatoes in the Caribbean we have chosen to discuss this topic as a separate subject.

Cooperators in the U.W.I./U.F. tomato project have conducted extensive surveys of tomato diseases and problems in the Caribbean Islands of Montserrat (1) and Haiti (2) and Phelps is preparing a list of tomato diseases in Trinidad. It was concluded there is no single disease which is devastating the tomato industry in the Caribbean but rather a multitude of diseases, each of which contributes to yield reductions (Tables 1 and 2). The most serious problems in Montserrat were determined to be bacterial canker and bacterial soft rot on the fruit and root knot nematode and southern blight on the plant itself (1). The two most serious pathogens in Haiti were determined to be root-knot nematode and the target spot fungus (2). Preliminary data indicate early blight, potato leaf roll virus, bacterial

Table 1. Tomato diseases and pests observed in 1973 in the Cul de Sac Plain area of Haiti.

Disease or pest	Presumed causal agent	Time of survey [a]	
		Winter	Spring
Blossom drop	adverse weather for fruit set	+	0
Damping-off	pre- and post-emergence complex	+	0
Southern blight	Sclerotium rolfsii	+	0
Early blight	*Alternaria solani*	+	+
Target spot	*Corynespora cassiicola*	0	+
Stemphylium blight	*Sternphylium botryosum*	0	+
Soft rot	*Erwinia carotovara*	+	+
Soilirot	*Rhizoctonia solani*	+	+
Fruit pox	the *fpx* gene	+	+
Gold fleck	the *Gdf* gene	+	+
Bacterial wilt	*Pseudomonas solanacearum*	+	0
Virus diseases	symptoms of tobacco mosaic and tomato spotted wilt viruses	+	+
Leaf miner	*Lirimyza munda*	+	+
Root knot	*Meloidogyne incognita*	+	+
Stem borer	possibly *Phthorimaea operculla*	+	0
Russet mite?	*Aculops lycopersici*	0	+
Spray damage	copper incompatibility?	+	+

[a] + = present, 0 = not observed. Source: Plant Disease Reporter 57:921-923, 1973.

Table 2. Occurrence of tomato disease and pests in Montserrat, W.I., 1971 and 1972.

Disease or pest [a]	Dates of survey		
	Winter 1971	Summer 1971	Winter 1972
Early blight	+	+	+
Bacterial spot	0	+	0
Bacterial canker	+	+	+
Bacterial wilt	+	0	+
Fusarium wilt	+	0	+
Southern blight	+	+	+
Botrytis rot	+	0	+
Soft rot	+	+	+
Soil rot	+	+	+
Fruit pox	+	+	+
Gold fleck	+	+	+
Virus diseases	+	+	+
Leaf miner	+	+	+
Russet mite	+	+	+
Root knot	+	+	+
Corn earn worm	0	0	+

[a] + = disease or pest was observed, 0 = not observed. Source:- Plant Disease Reporter 56:817-819, 1972.

wilt, nematodes and blossomend-rot are the primary disease problems of tomatoes produced in Trinidad.

Currently, sources of resistance or tolerance to bacterial canker and bacterial soft rot are being evaluated in the Florida breeding programme. No predictions can be made as to how soon breeding lines possessing tolerance or resistance to these two diseases will be available for evaluation in the Caribbean. At present no attempt is being made to incorporate the Mi gene which confers resistance to root

knot nematode into desirable stocks as it is not considered feasible. Also, there is no known source of resistance to the southern blight disease so it does not appear at present that variety development offers any possible control of these two problems. Sources of resistance to the target spot disease are known and have been evaluated for one season. Incorporation of resistance to target spot into desirable plant types for both Florida and the Caribbean is one of the current primary objectives of the breeding programme. Sources of tolerance to early blight are present in many of the lines previously evaluated in the Caribbean and emphasis has been placed on selection of highly tolerant lines. It was only recently demonstrated by studies at the University of the West Indies that potato leaf roll virus may be a serious problem. No positive action has been taken to locate sources of resistance as yet.

Research conducted thus far by the cooperative programme indicates host resistance is not going to provide exclusive control of tomato diseases in the Caribbean. Rather, disease control through methods of crop rotation, sanitation, and eradication coupled with control by use of protective sprays and fumigants is going to be required for successful intensive tomato production in the Caribbean.

TOMATO PRODUCTION PROBLEMS IN THE CARIBBEAN

Water:

The most limiting problem of tomato production throughout the Caribbean is water. The scope for the problem varies from too much water in certain islands in certain seasons to near drought conditions at other times in other islands. The most typical condition is a lack of water rather than an excess. This factor is evident from the disease surveys when it is observed that the most serious problem is root-knot nematode and that blossom-end-rot is so prevalent. Most tomato varieties can tolerate very high root-knot populations and considerable galling of the roots if an adequate level of soil moisture is maintained. As soon as soil moisture levels drop in fields of tomatoes infected with root-knot nematodes, symptoms become readily evident and damage with resulting losses can be quite severe. Also, blossom-end-rot is normally not observed even on the most susceptible of varieties, when proper soil moisture conditions and calcium levels are maintained. When soil moisture is allowed to fluctuate from wet to dry, a physiological deficiency of calcium occurs and then blossomend-rot becomes a problem even on the most tolerant of varieties.

The more primitive varieties are not nearly so affected by unfavourable water relations as are the newer varieties and breeding lines which have been developed for conditions of intensive agriculture. If Caribbean tomato growers are to make the most and best use of the newer varieties, systems of drainage and irrigation must be utilized to avoid the tremendous fluctuations in soil moisture which occur naturally.

Fertilizer:

A second limiting factor in Caribbean tomato production is inadequate fertilizer use by the farmer. The primitive varieties do not respond well to supplemental

fertilizer but the more recent Florida tomato varieties and breeding lines have all been genetically engineered to utilize larger amounts of fertilizer to produce higher yields. If the fertilizer is not applied, then the production of the new varieties will probably be no better and in some cases less than that of the older and more primitive ones.

Pest Management:

The production of tomatoes within a system of intensive agriculture using the newer varieties requires an understanding of pest control. The primitive varieties were able to tolerate a large amount of pests because of their growth habits. They were rank, indeterminate vines producing a lot of foliage and little fruit. By virtue of being indeterminininate in growth habit, the vines could grow for many months and often live through several disease epidemics. Such varieties are ideal for conditions of subsistence agriculture but wholly unacceptable for use in a programme of intensive and extensive agriculture.

The new varieties are determinate in growth habit and produce in six weeks as many fruit as the primitive varieties did in six months of harvesting. An uncontrolled disease problem spells economic disaster in a system of intensive tomato production. The successful Caribbean tomato farmer must become more familiar with pest management practices and learn how to control those disease and pest problemsfor which varietal resistance is not available. Varietal characteristics which dictate the type of pest management techniques utilized in intensiveversus subsistence types of tomato production can be characterized as follows:

Intensive	*Subsistence*
1. short bush	1. rank, indeterminate bush
2. early maturing	2. maturity variable
3. concentrated maturity	3. no concentrated maturity
4. uniform size fruit	4. fruit vary in size
5. all fruit same shape	5. shape of fruit not important
6. good interior and exterior	6. appearance of minor concern
7. high quality and grade of fruit	7. quality and grade of minor concern
8. good response to fertilizer	8. fertilizer response usually poor
9. good response to irrigation	9. most are drought hardy
10. disease resistance monogenic	10. susceptible or only tolerant to disease

Labour:

Labour is one of the most important problems to be considered in the production of tomatoes in the Caribbean. The usual reference to labour brings to mind the people who work in the fields with their hands doing the menial tasks of tomato production including hand shaping of the seed and plant beds, hand forming

irrigation ditches, hand hauling of harvested fruit to the packing house, etc. The supply of labour available to perform such menial tasks is declining and the successful Caribbean tomato grower must mechanize some parts of his farming operation in order to get the job done. This can best be illustrated by contrasting the characteristics of intensive versus subsistence tomato production in the Caribbean.

Intensive	*Subsistence*
1. large acreages of one variety	1. scattered small fields inter-planted with many crops and many varieties
2. power equipment utilized	2. all hand labour
3. adequate capital available	3. no capital or source of finances
4. good cultural practices	4. very primitive and inadequate practices
5. pest management programmes followed	5. no disease, insect, weed, or nematode control
6. good marketing system	6. no organized marketing system
7. fertilizer, pesticides, irrigation machinery parts, etc. readily available	7. usually nothing available
8. high quality seed used	8. usually home saved seed
9. good roads from farm to market centres for rapid transport of supplies	9. no or very poor roads
10. good means of transportation for export	10. transportation not needed
11. large acreages planted simultaneously	11. planting occurs year around
12. harvests made selectively and scheduled	12. no scheduled harvests
13. adequate packing and storage facilities	13. no controlled temperature storage

It is most obvious that a source of reliable and economical labour is required for any kind of intensive tomato production whereas skilled or even semi-skilled labour is not required for subsistence farming.

DISCUSSION AND CONCLUSIONS

The problems in developing tomato varieties for the Caribbean depend upon where and how the varieties are to be utilized by the farmer. The ability to set fruit

under hot, humid and otherwise adverse environmental conditions is of prime importance. In some areas of the Caribbean where tomatoes are grown, all the techniques of modern agriculture are utilized, including adequate irrigation, high levels of fertilizer, good soil management and good disease, insect and weed control. In other areas of the Caribbean only the most primitive of methods are used in tomato production. With such primitive methods of subsistence farming, only primitive varieties will perform suitably. A variety which is high yielding under good or excellent intensive farming practices will yield almost nothing when placed in conditions of subsistence agriculture. Likewise, those varieties which perform best in subsistence conditions do not have the capability to utilize effectively the benefits of increased pest control coupled with adequate fertilizer and irrigation. Varieties developed for use in the areas of subsistence agriculture must of necessity be considerably different from those developed for use in areas where the most advanced agricultural practices are utilized.

We are of the opinion that development of varieties for primitive areas of agriculture is not meaningful. Furthermore, we are of the opinion that an excellent environment for year around tomato production exists in the West Indies and there is no reason Caribbean adapted varieties cannot be developed which have the potential in intensive culture to provide the tomato needs for the West Indies. We have concentrated on the development of varieties which have the ability to set fruit under the environmental stresses of high temperature and high humidity and also respond to the use of modern agricultural practices by producing high yields of high quality fruit.

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Tomato variety development and multiple disease control with host resistance[1]

Pat Crill, D. S. Burgis, J. P. Jones, and Jimmy Augustine[2]

FOREWORD

Since its inception the Florida tomato breeding program has resulted in many achievements of benefit to growers, professional plant breeders, and consumers alike. Several men have devoted much of their professional lives to breeding to improve the horticultural qualities of the tomato and to bring about disease control through the use of host-plant resistance. This monograph presents a discussion on host resistance and provides a written record of the history, objectives, methods, techniques, and achievements of this breeding program. It should be a useful document of considerable interest to professional horticulturists interested in plant breeding and of special interest to those who are involved in the breeding of tomatoes.

Florida, by virtue of its rather high ambient temperatures, high relative humidities, and comparatively high annual rainfall, provides an environment in which tomato diseases flourish. This has presented a critical problem to commercial tomato production. Disease problems became so acute that in 1925 a Florida Tomato Disease Laboratory was established by the State Legislature at the request of growers at Palmetto, Florida. This was the precursor to the Gulf Coast Experiment Station, established when the "Laboratory" was moved in 1937 from Palmetto to a new location in Bradenton. With the move, a wider research responsibility was given the new Gulf Coast Station, but tomato breeding remained the major responsibility for that Station. Still later, in the 1950s, the Gulf Coast Station was moved to yet another location in the Bradenton area, to what is now known as the Florida Agricultural Research and Education Center.

Through all of these changes, the tomato breeding project has been the primary research responsibility for this Station. So much has been accomplished,

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[1]Florida Agricultural Experiment Stations Monograph Series No. 10.

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and such great benefit was resulted to the people of Florida as a result of this work, that I encouraged Dr. Crill and others who have made valuable contributions to document the story of "Tomato Variety Development and Multiple Disease Control with Host Resistance."

As you will see, the monograph is not specific to work conducted at the Bradenton Stations. Although these Stations have served as the nucleus for tomato breeding work in Florida, splendid contributions to the tomato breeding program have of course also been made at Homestead, Gainesville, and other Research Centers.

This monograph is dedicated to all who have made real contributions to the development of tomato breeding technology. I would gratefully acknowledge, with thanks from the citizens of Florida, our sincere appreciation to the authors, professional workers, and administrators for the dedicated and concerted efforts put forth to make this publication possible.

John W. Sites
Dean for Research, Emeritus

INTRODUCTION

The tomato is of tropical American origin. The earliest information on the tomato is from the European explorers' records of the sixteenth century (Figure 1). The Europeans carried the tomato back to the Old World, and no new markedly different types have been developed since.

The tomato was introduced into the United States of America from Europe, where it served as a botanical curiosity and was thought to be an aphrodisiac. Despite the association with such a stigma (or perhaps because of it), Americans began to consume tomatoes in ever-increasing quantities. The Creoles of New Orleans were the first "new" Americans to recognize the culinary merits of the tomato. As early as 1812 they were using tomatoes to create some of their distinctive foods. The popularity of tomatoes as a food grew rapidly as more people were introduced to this new crop.

Tomato production was first practiced in Florida in 1870 by Perry and Wilson of Alachua Country. As early as 1872 tomatoes were grown by E. S. Blund on Sanibel Island and in 1875 by Gillette in Manatee Country. The tomato shipping industry in Florida was established in the Palmetto-Manatee area when Joel Hendrix grew, and then shipped Florida tomatoes to market in New York City in 1879. These winter-grown Florida tomatoes were a welcome sight on the Yankee dinner table where fresh vegetables were rare. The Florida tomato became an increasingly popular item with northern consumers, primarily because it was sold cheaply enough to be available to anyone, regardless of economic status. Because of increasing consumer demand, more Florida farmers engaged in tomato production. Further successful shipments to the north were made, and more consumers demanded more winter tomatoes. In this way, the Florida shipping tomato pro duction area expanded from Hendrix's small plot in 1879 to more than 6,675 acres on a very intensive scale by 1900, to over 29,000 in 1930, and over 50,000 in

1. Early illustrations of the ancestors of present day cultivated tomato (from W. W. Tracy) as grown in Europe in the 16th and 17th centuries.

1970. The maximum acreage planted was 66,000 acres in 1957.

The first tomato crops were evidently not seriously bothered by diseases. However, by the turn of the century plant diseases were the limiting factor in tomato production. In 1899 Smith reported that a "Fusarium-caused" disease had put an end to the production of shipping tomatoes in some areas of the state, and in 1903 Orton stated that Fusarium wilt of tomato caused a loss of $500,000. It had become quite evident that if this relatively young industry was to survive, something had to be done to reduce or eliminate losses from diseases.

Although the problem was obvious at the turn of the century, it was some 20 years later before a tomato breeding program was initiated by the University of Florida. There were various reasons for the delay. Even though the University of Florida was chartered in 1853, it was not a permanent institution headquartered in Gainesville until 1906, and the Florida Agricultural Experiment Stations had not become firmly established. Plant breeding was itself a young science; Darwin's theories of evolution were just becoming accepted, Mendel's laws of inheritance had just been rediscovered, and Orton had just demonstrated that disease resistance was a varietal characteristic which could be manipulated by plant breeders. Furthermore, there appeared to be an almost endless supply of land and water in Florida which could be used for tomato production, and it was known that a good tomato crop could be produced by avoiding the old "sick' soils and moving to newly cleared virgin land. By the early 1920s all these factors had evolved and

developed to a point where it became desirable, as well as politically and economically expedient, to establish the University of Florida tomato breeding program, and through it to emphasize the development of new varieties for Florida with disease control through resistance.

EFFECT OF DISEASES ON TOMATO PRODUCTION

Plant disease becomes the limiting factor in tomato production in Florida when varieties without multiplediseaseresistance are grown commercially. There are 191 known diseases of tomatoes (Table 1), and many of these occur in Florida. Two of the diseases which have been limiting factors in tomato production in Florida are nailhead rust and Fusarium wilt. Both have been satisfactorily controlled through the development of resistant varieties. Other very serious diseases, but in many cases not the limiting factor in production, are: gray leafspot, leafmold, Verticillium wilt, bacterial leafspot, southern bacterial wilt, graywall, blossom-end rot, and tobacco mosaic virus. They are, with the exception of bacterial leafspot and southern bacterial wilt, satisfactorily controlled through genetic resistance and tolerance inherent in varieties developed by the University of Florida tomato breeding program.

HOST RESISTANCE IN DISEASE CONTROL

The study of host resistance and pathogen variation is a recent scientific endeavor, the basic foundations of which go back to Gregor Mendel, founder of modern genetics. The application of Mendelian principles to the study of pathogen variation and host resistance was hindered for many years because of the wide acceptance of the theory of spontaneous generation and the fact that Mendel's work was not widely accepted since it was overshadowed by the nearly simultaneous publication of the theories of Wallace and Darwin.

HISTORICAL ASPECTS

Utilization of host resistance to various pathogens as a means of disease control has probably been practiced since the beginning of agriculture when man ceased his nomadic existence and started cultivating plants for consumption by himself and his animals. The records of attempts to obtain disease-resistant varieties of

Table 1. Diseases of tomato.

Causal agents	Number
Fungi	86
Bacteria	9
Nematodes	4
Parasitic Phanerogams	3
Viruses	75
Non-parasitic	10
Unknown	4
Total diseases of tomato	191

crop plants prior to 1900 are very meager. The Greek Theophrastus writing circa 350 B.C. noted that rust was a very serious problem on cereals and some varieties were more susceptible than others. This same observation was later made by the Roman known as Pliny the Elder, circa 60 A.D.

Since the causes of disease were unknown in ancient times, people often assumed their crops became diseased because the gods were displeased with them. The only way one could insure a bountiful, disease-free crop, therefore, was to placate these gods with sacrifices. The Greeks offered the first heads from their cereal crops to the goddess Ceres in thanksgiving for allowing them to produce the crop. The Romans celebrated annually the Robigalia, which was a religious ceremony by which the god of rust, Robigus, was asked to spare the crops from rust. In America, the Incas sacrificed the first ears harvested from the corn plants to Cinteutl, the goddess of corn, in thanksgiving for the year's crop.

An advance in disease control came about with the discovery of Bordeaux mixture in 1885. Prior to this, elemental sulfur and various forms of copper had been sporadically used as therapeutants in various parts of the world since about 1000 B. C. Still, most of the progress made in disease control before the twentieth century was in the selection, both natural and by man, of disease resistant or tolerant types.

By 1900 the knowledge available to those seeking to control plant diseases could be summarized as follows:

1) the germ theory of disease was well established,
2) the techniques for hybridizing plants were known,
3) variation was known to exist in crop varieties,
4) some varieties of certain crop plants were not susceptible to certain diseases,
5) the diseaseresistant plants usually had certain undesirable horticultural characters, and
6) no control measures were known for most diseases.

In 1900 W.A. Orton developed, with plant breeding techniques, a cotton variety which was resistant to Fusarium wilt. This was the first variety of any crop developed specifically for disease resistance. In 1905 Biffen reported that resistance to yellow rust of wheat was controlled by a single gene. With this limited background knowledge, plant breeders now challenged the anti-Darwinists by initiating variety development programs with emphasis placed on genetically controlled disease resistance as well as yield and quality.

SOURCES OF RESISTANCE

Four sources of resistance are usually available to tomato breeders: 1) resistance exhibited by available varieties, 2) resistant plants within a variety, 3) resistance in wild types of cultivated plants, and 4) induced mutations.

1) *Resistance among varieties* is the first possible source of resistance which should be checked by the plant breeder. If the resistance is present in varieties already in use, then adequate yield and quality are already present.

The first example of utilization of this source of resistance was by W.A. Orton who demonstrated that 'Iron' cowpea was resistant to Fusarium wilt

and root knot nematode. One of the first examples in tomato was the demonstration by Weber that the variety 'Marvel' was resistant to nailhead rust.

2) *Resistant plants within a variety.* The varieties being grown in an area may be heterozygous for resistance to a particular disease. Selection of those plants free from disease has been used in many instances as the initial source of resistance. The first record of such efforts was by W. A. Orton who developed the first Fusarium wilt resistant cotton variety through the selection of wilt-free plants he found in the established varieties. Numerous examples of locating resistance from segregating populations of established varieties are known. This is the second source of resistance checked in the breeding program since such resistant plants will also possess many desirable characteristics of yield and quality.
3) *Resistance in wild types of the cultivated plant.* The first example of this type of resistance and its incorporation into a variety was again provided by W. A. Orton. Orton determined that the wild African citron was resistant to the watermelon Fusarium wilt pathogen, and from this he developed a wilt-resistant variety. Porte and Wellman of the USDA and Tucker at Missouri simultaneously determined in 1937 that one line of *Lycopersicon pimpinellefolium* was resistant to race 1 of the Fusarium wilt fungus. Alexander and later, Stall and Walter demonstrated that tomato plant introduction 12691 5 was resistant to race 2 of the Fusarium wilt pathogen.

   When resistance is located in a wild type plant, much more effort is required by the plant breeder to incorporate this into a plant with desirable horticultural or agronomic qualities.
4) *Induced mutations.* Mutations induced with various mutagenic agents have provided some advancements in agronomic and horticultural characteristics of various crops during the past 20 years. Induced mutations, however, have been remarkably unsuccessful as sources of disease resistance. When the plant breeder seriously considers the evolutionary development of plant pathogens in conjunction with the gene-for-gene and the one gene-one enzyme theories and the postulation that disease resistance is due to the presence, rather than absence, of a gene, it becomes obvious that induction of mutants for host resistance is futile. Mutagenic agents do not create new genetic material; they can only alter the genetic material present, which usually results in the absence of a function. Since host : pathogen interactions are evolutionally mutually dependent and so closely interrelated, it is unlikely that application of mutagenic agents to a susceptible host could convert it into a resistant one. Although examples of inducing host resistance with mutagenesis have been reported in peanuts, oats, and soybeans, for the most part such attempts have been unsuccessful.

GENETIC LIMITATIONS IN BREEDING FOR HOST RESISTANCE

The most economical and effective solution for the past 50 years to the question of controlling various plant diseases has been to control them by developing

resistant varieties. However, in this period of time it has become obvious that not all plant diseases can be controlled by the development of resistant varieties. A number of reasons why this is the case have been given by R.R. Nelson as follows:

1) There may not be a source of genes controlling resistance to the disease present in the crop being investigated or its near relatives. Nelson has pointed out that if the pathogen has a wide host range - such as occurs with *Phymatotrichum omnivorum, Sclerotium rolfsii,* or the damping off fungi - it is unusual to find genes for resistance.
2) If a source of resistance exists, it may be located in a species that is too remotely related to make hybridization possible (cross-sterility).
3) The genes for resistance may be closely linked with an undesirable gene or genes or the resistance gene may be associated with undesirable genes as a pleiotropic character. For example, crown rust resistance in oats is linked with susceptibility to Victoria blight.
4) The number of genes controlling the disease resistance reaction influences whether it is feasible to breed for resistance. If resistance is monogenic or controlled by only a few genes, the possibilities of developing resistant varieties is much greater than if a larger number of genes are involved.
5) The genetic diversity a pathogen possesses has the same end result as (4), but is manifest by a different mechanism. If the pathogen possesses a large number of genes for pathogenicity, then progress in breeding for disease resistance will probably be very limited.

METHODS FOR DETECTING HOST RESISTANCE

There are basically two types of screening programs plus a combination of the two. These three methods or types of screening programs are discussed with respect to advantages and disadvantages.

1) *Screen for resistance in the field where disease is a problem.* This is the kind of screening program which has been used to make many advance ments in the development of disease-resistant varieties. One of the primary reasons it has been popular is that most plant breeders are not knowledgeable of many plant pathological techniques, and none are needed to use this method. Other advantages of this method are as follows:
   a) Field screening is relatively inexpensive. Breeding lines must be evaluated in the field anyway, and a little more effort is usually all that is required to establish a breeding nursery in an area of disease prevalence.
   b) Large numbers of progeny and large populations can be grown in the field, so it is relatively easy to establish the relative resistance or susceptibility of a large number of breeding lines.
   c) When resistant plants are detected in the field, it is usually possible to obtain seed or planting stock in relatively large amounts from which the next generation is produced.
   d) In most instances, if the breeding lines are grown in the area where the disease is rampant, then the plant breeder can be fairly safe in assuming the resistant lines are actually resistant to the correct strains of the pathogen.

The disadvantages of screening for resistance under field conditions are as follows:

a) Disease development may be retarded or disease may not develop at all in the field nursery because of abnormal environmental conditions. If no disease occurs, then it is impossible to evaluate resistance.

b) Symptoms of some diseases are quite difficult to distinguish in the field, and the expression of some symptoms often depend upon special conditions. If symptoms are not readily obvious, it is normally quite difficult to evaluate resistance.

c) Field screening is normally done in order to evaluate resistance to a single pathogen. In many breeding nurseries there may be a number of diseases present, and it becomes exceedingly difficult to evaluate the nursery for a single disease because of overlapping symptoms of diseases other than the one under consideration.

d) In many cases the disease being investigated, and for which resistance is being evaluated, occurs primarily in an area away from the headquarters. In such instances it often becomes expensive and time-consuming to evaluate disease resistance in the field.

e) In most instances it is highly desirable to know which strains of the pathogen are producing disease on the breeding lines in the field screening nursery. This information is almost impossible to obtain in the field.

2) *Screen for resistance with artificial conditions.* With this method the screening tests and evaluations for disease resistance are conducted in the laboratory, greenhouse, growth-chamber, or with other artificial procedures. In general, those things which were advantages with field screening become disadvantages for artificial methods and vice versa. The advantages of screening with artificial conditions are listed as follows:

a) Environmental conditions can be controlled and repeated so that disease development is assured and the number of "escapes" can be reduced to zero or nearly so.

b) Disease symptoms are usually evident, and when a uniform environment is utilized, resistant plants are usually relatively easy to distinguish. There should be no complicating factors of environment in distinguishing resistant from susceptible plants.

c) Most diseases can be evaluated with artificial conditions of screening, and it is not necessary to screen the plant populations for resistance in the area where they are to be grown. Tropical crops can very effectively be evaluated for resistance to certain diseases in greenhouses and growth chambers located in the temperate zones.

d) Often the most important advantage to using artificial conditions is that of knowing exactly what isolates, strains, or races of the pathogen are capable of causing disease. Artificial screening techniques are normally required to generate any useful genetic data pertaining to the genetic control of resistance.

The major disadvantages of using artificial methods for evaluation and detection of host resistance are as follows:

a) Screening with artificial conditions may be quite expensive.
b) Normally, only a relatively few lines can be screened for resistance using artificial conditions. If it is necessary to evaluate large populations, then screening becomes quite expensive and laborious.
c) It may be difficult to obtain seed or planting stock from plants which are found to be resistant. The amount of planting stock which can be pro duced directly controls the size of the screening generation and rate of progress in the breeding program.

3) *Combination of 1 and 2.* In the majority of cases it is better to select those conditions which are most applicable to the crop and pathogen being studied, and those which are easiest and most economical to manage. If feasible, lines should be routinely checked and screened under artificial conditions and the survivors of this screening further evaluated for resistance in the field.

CARDINAL POINTS TO CONSIDER IN A SCREENING PROGRAM

The following important points should be considered in a screening program:

1) Ample and adequate populations of both host and pathogen must be used. It is most important to use large populations, especially when working with resistance mechanisms controlled by more than one gene. The smaller the population evaluated, the greater the chances for error.
2) Proper experimental design is essential. This means that adequate controls must be used in both field and greenhouse tests, and these must be located in the proper areas in the field.
3) An adequate sample of strains or races must be used in screening. There are many cases of the development of resistant varieties which turned out not to be resistant at all just because they were never screened for resistance to more than one race or strain. Pathogens often lost their ability to cause disease when cultured for a long period of time in artificial conditions. Pathogenicity and virulence of the pathogen should never be in doubt.
4) Procedures should be standardized for the production of inoculum, dose level of the pathogen, length of incubation period before disease evaluation, method of evaluation, and *environmental conditions* for disease develop ment.

TOMATO BREEDING

Plant breeding defined

There are numerous textbook definitions of plant breeding, but essentially the process results in a successful variety which represents the selection of a single genotype or phenotype based upon the application of the principles of genetics. In reality, a successful plant breeder is more of an artist than a true scientist. Just as in conventional artistry, the success of the creation depends upon the artist's know ledge and utilization of the raw materials. With the plant breeder this means a thorough and intimate knowledge of the crop from seed to seed. The breeder

must be aware of and have an appreciation for the problems of seedsmen - including producers, contractors, buyers, wholesalers, and retailers. He needs to be aware of farming practices, including local variety adaptation for yield and quality, as well as planting, cultivating, fertilizing, irrigating, and harvesting procedures. He must consider wholesalers of the crop who are concerned about economic factors as well as components of quality, storage, shipping and longevity. He also should consider retailers of the crop who have all of the problems of wholesalers plus all the problems of processing and marketing. Finally, the plant breeder must appreciate the needs of consumers who must have a nutritious, attractive, palatable product free from toxic substances.

The more familiar the plant breeder is with all these problems, the more effective he is in developing varieties. The basis of plant breeding is selection of plants, and before a selection can be made, a decision must be made as to what constitutes a desirable plant. The only way the breeder knows what is desirable is to know all the problems and needs of the total industry from planting stock to the consumer.

Successful plant breeding then, is the art of creating a variety which fits the needs of the total industry based upon the application of proven principles of genetics, plant pathology, nematology, entomology, crop production, economics, marketing, food technology, and plant physiology.

Tomato breeding methods

The two primary objectives in a breeding program are (1) to obtain maximum yield, and (2) to obtain maximum quality. All other objectives which can be listed are a part of either yield or quality, or both. So it is with disease resistance. By itself, resistance is worthless to the farmer or consumer. It is only when disease resistance is combined with desirable horticultural characters affecting yield and quality that a diseaseresistant tomato variety is useful and of commercial value in food production.

Because of the large number of genes involved in yield, quality, and disease resistance, progress with the usual type of tomato breeding program is by necessity slow, laborious, and expensive. Several techniques have been devised and evaluated recently to accelerate the process of variety development. If past standards of excellence in variety development are followed, future varieties will be higher yielding, produce better quality fruit and have more genes for disease resistance.

Several breeding methods are commonly used by plant breeders to develop new tomato varieties.

Pure-line breeding method

Selection was the first breeding method used by early tomato breeders, and this procedure remains the basis of present day breeding programs. Genetic variability within a population is the scientific basis of selection, and such selection in naturally variable populations of tomatoes generally followed the pure-line breeding method. By selecting the phenotypes which were desirable, the new populations of plants resulted in improved varieties.

W.A. Orton popularized this method of plant breeding when he developed Fusarium wilt resistant varieties of cotton and cowpea by selecting resistant plants from locally grown varieties at the turn of the century. Selection was practiced in many crops by numerous plant breeders for improved disease and pest resistance as well as horticultural and agronomic characters. It was only logical that early-day tomato breeders should also emphasize selection from local varieties as the primary means of variety development.

It is significant that no large-fruited types of tomato have been found growing naturally in a wild state. This indicates the American Indian did an outstanding job of varietal improvement with the tomato by selecting for large fruit size. These types were carried back to the Old World by explorers from where they were later reintroduced from Europe into the U.S. The five basic types which were re introduced were the Cherry, Plum, Pear, Old Round, and Large Red. It is also significant that no new markedly different types have been developed since. The first American developed variety was the 'Tilden,' introduced by Henry Tilden in 1865. This was followed by numerous other varieties, developed mostly by farmers and by food and seed companies.

The advantage of pure-line breeding is that new types or varieties are easily obtained if desirable characteristics are found to occur naturally. The disadvantage is that variation has to be present in the population from which selections are made.

Pedigree method

In 1903 Johannsen demonstrated to plant breeders that selection within populations of limited genetic diversity was futile. The problem now became one of inducing more variation in a population so progress could be made by selection. In 1761 Koelreuter verified and extended the work of Camerarius, which was published in 1694, by demonstrating the presence of sexuality in plants, and it is well documented that numerous people were making controlled crosses with plants before 1900. Plant hybridization was, in fact, a hobby pursued by several farmers, seedsmen, and botanists during the nineteenth century, but little progress was made in developing improved varieties with this technique.

Plant hybridization as a breeding technique was stimulated tremendously by two developments: In the early 1900's W. A. Orton developed a Fusarium wilt resistant watermelon, and in the 1920's, a new crop known as grain sorghum was created by John B. Sieglinger in Oklahoma. The pedigree method consists essentially of the procedures developed by Orton and Sieglinger.

Orton applied his selection techniques to the development of a wilt resistant watermelon variety in the same way that he had attacked the cotton and cowpea wilts, but without success. He determined that the wild citron was resistant to wilt and conceived the idea of crossing the wilt resistant citron with the cultivated susceptible watermelon. From such a cross he was able to select recombinant types in the segregating generations containing desirable horticultural characteristics and resistant to wilt. This was the first example of the development of a disease resistant variety through hybridization and selection. Sieglinger created the grain sorghums using essentially the same techniques as Orton, but with

different objectives. His success in creating new sorghums which could be mechanically harvested, by crossing two plants, each possessing some desirable characters, and then selecting the desirable recombinants in segregating generations, formed the basis for the pedigree method of breeding.

In the pedigree method, parents containing the desirable characters are selected for crossing, and records are kept on the progeny. Crosses are made to provide sufficient seed to grow an adequate F_1 population. An adequate F_1 is one that will produce enough seed to grow an F_2 population of the desired size and leave enough reserve seed to sow another F_2 population in case of a crop failure.

The F_2 population is the critical population for the plant breeder, as this is the first population in which selections are made. The size of the F_2 population depends solely upon how many plants the breeder is able to evaluate. The F_2 and F_3 populations are where the most critical screening for disease resistance should be done. All the susceptible plants should be eliminated in the F_2 and F_3. After eliminating all the disease-susceptible F_2 plants, the remaining resistant plants are evaluated, and the ones possessing the most desirable horticultural characteristics are retained. The maximum number of selections in the F_2 is dictated by how many F_3 populations the breeder can handle.

Each F_2 plant which is selected is allowed to self-pollinate, and the seed from each plant is harvested individually. An F_3 population or family is produced from each F_2 plant. Each F_3 population should be screened for disease resistance. From this progeny testing data it is possible to determine for monogenically controlled resistances which F_3 populations are homozygous resistant and which are heterozygous. The size of the F_3 depends upon the facilities available, but each F_3 family should contain enough individuals so the general features of the family as well as the degree of homozygosity within the family becomes evident.

The number of selections in the F_3 are again influenced by several factors. It is often the case that none of the families are promising, and the entire cross is discarded; on the other hand, there may be a relatively large number of promising plants within a family. Seed of the single plant selections in the F_3 family are planted to produce the F_4 populations. Homozygosity is not normally attained in this generation and some genetic diversity remains. If the disease screening record in the F_3 indicated the population was heterozygous, it should be screened again. If the line was homozygous, it is probably not justifiable to screen the F_4 again. Single superior plants are selected, from which the F_5 populations are grown. Commercial trials are probably justified in the F_5 because the potential of the individual families is usually well fixed after the F_5 generation. A common practice is to bulk harvest the F_5 plants and produce a large quantity of F_6 seed for variety trial evaluations.

By the time the F_6 generation has been reached, the number of families has usually been drastically reduced by discarding the least desirable ones. Selection in the F_6 and F_7 is normally a continuation of earlier selection procedures.

The next step is final evaluation of the most promising lines. This involves (1) replicated yield tests, (2) quality tests, and (3) final screening for disease resistance and observation for any other weaknesses. The criteria for releasing a new variety vary greatly. For example, in most cases a new variety to be released must be just as good in all respects as the varieties currently being grown and superior to all

varieties in at least one characteristic. The notable exception to this rule is the release of a variety resistant to a particular disease which has become the limiting factor in crop production.

The primary advantage of the pedigree method of breeding self-pollinated crops is that the plant breeder has the chance to exercise his skill in selection, and from the record keeping he has an account of the pedigree and progeny performance. The main disadvantage is that the amount of material he can successfully manage is restricted.

Bulk-population breeding

This method is suited primarily for seed crops and has been used by some plant breeders seeking to develop disease resistant varieties by placing the screening program strictly at the whims of nature. The first person to use the bulk-population method was Nilsson Ehle of Sweden, who used it to combine winter-hardiness of one cereal parent with the high yield of another.

In the bulk-population method of breeding, the F_2 generation is planted in a large plot containing several hundred or thousand plants. When the seed is mature, it is harvested in bulk, and is used to plant another large plot the following year. This process is repeated as many times as the plant breeder thinks is necessary. It is assumed that during the period of bulk propagation, natural selection is operating and the shift in gene frequencies is toward desirable characteristics. In practice. the breeder usually helps out natural selection by eliminating the obvious undesirable and unproductive plants in the population. After the plant breeder has decided that bulking has accomplished its purpose, single plant selections are made and evaluated as in the pedigree method.

There are five advantages of the bulk-population method:

1) The screening process is by natural selection - no inoculations or disease ratings are made.
2) Some slight selection by the plant breeder greatly aids natural selection.
3) Growing of large populations is feasible, and this increases the chance of recovering high-yielding, well adapted types.
4) The bulk population method eliminates the necessity of keeping pedigree records.
5) Homozygosity increases during the period of bulk handling, so the final selections made after bulking would be expected to breed true. The average percentage of homozygosity under selfing is very high by the F_6 generation and approaches 100% by the F_{10}.

The disadvantage of this method is that it is not well suited for many crops including fruit, tree, and most vegetable crops, although modifications of this method can be used. A modification of the bulk-population method was used to develop the University of Florida hot-set breeding-lines. These lines were developed by bulk-selecting plants from segregating populations for several generations in environments of high temperature and humidity.

Backcross method

The backcross method has been used by plant breeders more than any other in the development of resistant varieties. This method provides a precise technique

of improving varieties that are superior for a large number of characters but are deficient in one or a few.

A variation of the method was first used by W.A. Orton to develop the Fusarium wilt resistant watermelon. He used the adapted and commonly grown watermelon varieties as the recurrent parent and transferred the genes for resistance from the citron into the commercial watermelon. In 1922 Briggs began a very comprehensive backcross breeding program to develop bunt-resistant varieties of wheat.

The backcross method makes use of a series of backcrosses to the variety to be improved, during which the character or characters in which improvement is being sought is maintained by selection. At the end of the backcrossing, the gene or genes being transferred will be heterozygous. After the last backcross the population is selfed, and by selection, homozygosity for the particular gene pair is obtained and will result in a variety which has the same adaptation, yield potential and quality of the recurrent parent, but superior to this parent in having the particular characteristic which was obtained from the nonrecurrent parent.

The method is particularly useful in disease resistance breeding, as it provides a means of producing a variety which has all the morphological and agricultural characteristics of the improved variety as well as the few genes for resistance from the wild type parent. There are three requirements for a successful backcross program:

1) a satisfactory recurrent parent must exist,
2) ability to maintain a high intensity of the character which is being transferred is essential, and
3) sufficient backcrosses must be made to reconstitute the recurrent parent to a high degree.

A hypothetical example of the backcross method as it has been used to transfer a resistance gene (R) from a wild variety (B) to an adapted variety (A) can be diagrammed as follows:

A = adapted variety; disease susceptible rr
B = wild variety; disease resistant RR

1) Cross A X B and plant out the F_1 seed
   (50% A) = 100% resistant
2) F_1 X variety A = BC_1- screen for resistance and cross
   (75% A) = 50% resistant
3) BC_1 X variety A = BC_2- screen for resistance and cross
   (87.5% A) = 50% resistant
4) BC_2 X variety A = BC_3- screen for resistance and cross
   (93.8% A) = 50% resistant
5) BC_3 X variety A = BC_4- screen for resistance and cross
   (96.9% A) = 50% resistant

After five or six backcrosses the population is selfed and the resulting progeny are screened for disease resistance. If rigid selection for the recurrent type with the resistance has been satisfactory, it should be possible to release the new variety by bulking the seed from this population for increase. The genetics of the character involved may require another generation of selfing and individual progeny analysis for disease resistance before bulking the seed.

The primary disadvantage of the backcross method is that no progress is made in general productivity or yield of the new variety. The technique is suitable only when a superior variety exists and it is desirable to transfer a single or at most a few genes from an inferior variety to the superior one. This method has been most useful in transferring disease-resistance genes from wild types of tomatoes to adapted varieties.

F_1 hybrid varieties

F_1 hybrid tomatoes have become increasingly popular, especially in the packet seed and home garden plant sales. Also, there are several F_1 hybrid varieties available for use in the fresh market industry of the East, Midwest, and California. It was generally accepted for many years that heterosis did not occur with F_1 hybrid tomatoes; however, several researchers have reported in recent years the occurrence of heterosis.

Advantages of F_1 hybrid varieties are enumerated below:

1) *Increased seed quality* is an objective seedsmen have been striving to obtain for many years. F_1 hybrids are by no means the complete answer to production of high quality seed, but they can provide a high quality initial product for the grower. F_1 hybrid seed usually germinate more rapidly and uniformly than seed of either parent. This is the first visible expression of heterotic behavior, which carries over into final yields. Associated with the increase in germination rate is an increase in uniformity and rate of seedling emergence. Increased seed quality is beneficial to plant producers, whose seedling production operation is programmed to produce plants in a given period of time. The more uniform the emergence of seedlings, the more efficient the entire plant production operation can be. The only way plant producers can mechanize the plant production operation is to use seed which gives a uniform stand of tomato seedlings in a programmed operation.

   Those farmers and growers who direct-seed their tomato crop also enjoy the benefits of increased germination and uniform emergence. Precision seeding becomes much more feasible and practical, and much less seed is required to produce a uniform stand of plants in the field. If the farmer who direct seeds in the field is planning to machine-harvest his tomato crop, the more uniform the crop is, the more efficient machine-harvesting will be. It is quite feasible to assume that F_1 hybrid varieties will eliminate the practice of overseeding and thinning of excess plants to obtain a uniform stand.

2) *Increased uniformity of stands, fruit set, and fruit size* is feasible with F_1 hybrid varieties. If complete mechanization of tomato production and harvest is to occur, the more uniform the field population, the more efficient mechanization will be. One key to machine-harvest of fresh market tomatoes is once-over destructive harvesting. For such a system to be effective, harvest must take place when the maximum number of fruit per unit area are mature. Ideally, every plant in the field to be harvested should have germinated, emerged, flowered, set fruit, and matured the maximum number of fruit per plant simultaneously. It is obvious that environment will affect these

developments; but when F_1 hybrid varieties are utilized in the production of machineharvested tomatoes, uniformity in general will be increased, and thereby yields should be increased. It should be emphasized that standard varieties grown for hand harvest will probably out-yield machine harvest F_1 hybrids if they are evaluated using hand harvest methods.

3) *Increased earliness.* F_1 hybrids have been reported to have a shorter maturation period resulting in increased earliness. In most cases, early harvest results in higher prices and increased returns from sales.
4) *Increased yields* have been demonstrated with F_1 hybrid tomato varieties when compared to standard varieties. Yield increases, however, are often not significant, and often in yield trials a good standard variety will out-yield the F_1 hybrid. For this reason many tomato breeders have been dubious of F_1 hybrids. Yield increases from F_1 hybrid varieties will be much easier to substantiate with a completely mechanized culture of crop production. As discussed earlier, yields will be increased not necessarily by the production of more fruit, but by the increased uniformity and heterosis which results in more fruit per plant and per acre being ready to harvest at one time.
5) *Better management of the breeding program* is a primary factor in using F_1 hybrids as varieties. The varieties in use in a given area depend upon a) availability of seed, b) occurrence of diseases and pests, c) handling procedures, d) quality of the end product, and e) yield of the end product.

In 1969, virtually 100 percent of Florida's mature-green tomato industry was planted with the variety 'Homestead,' In 1969 the variety 'Walter' was released and by 1972 essentially 100 percent of the acreage was planted with 'Walter.' Varietal changes normally do not occur so rapidly; but 'Walter' was resistant to race 2 of the Fusarium wilt pathogen, seed was made available to Florida farmers by progressive seedsmen of the U.S., and the variety produced high yields of high quality fruit, thereby fulfilling the requirements of the interest groups previously discussed.

F_1 hybrid varieties of tomatoes appear most promising as a means of controlling diseases. The diseases occurring on tomatoes in Florida vary from year to year. Furthermore, the development, release, and acceptance of a new variety may serve to change the disease situation, as do widespread changes in cultural practices. If those concerned with tomato variety development in Florida are aware of this, then the disease control situation may become more effective. Shortly after the release of 'Walter' and its widespread acceptance, two new diseases, Cercospora leafblight and target spot were found on this variety by Dr. C. H. Blazquez, a cooperator in the tomato breeding program. After Dr. Blazquez's observations, screening tests were conducted, tolerant breeding lines were isolated, and the epidemiology of the two diseases was vigorously studied in the field. It was concluded that these two diseases do not pose a serious threat to Florida's tomato industry, and that if they become more serious than anticipated, a resistant variety can be rapidly developed as an F_1 hybrid. If F_1 hybrids are utilized as varieties, and if the costs of labor, space, and time are not allowed to become a factor, yield losses due to the appearance of new diseases should be minimized.

6) *Control of seed production by the breeder* is a distinct advantage of F_1 hybrids. It makes a variety into a proprietary item rather than being available to anyone regardless of their expertise in seed production. F_1 hybrid varieties, if properly managed, should be advantageous to the breeding program, seed producers, seed retailers, farmers, and consumers alike. A means of maintaining high quality, truetotype seed is inherent with F_1 hybrid varieties.

Disadvantages of F_1 hybrid varieties are:

1) *Increased size and cost of the breeding program* is the first disadvantage of using F_1 hybrids as varieties. For this reason almost all F_1 hybrid tomato breeding programs are conducted by profit-making organizations and not educational institutions. A successful F^1 hybrid program requires the successful development of inbred lines as well as conducting genetic studies, combining ability studies, and extensive evaluation of F_1's as varieties to support variety release. In effect, the program required for development of inbred lines is similar in size to the normal breeding program required for development of standard varieties. Standard varieties are sometimes developed by hit or miss techniques; however, the breeder relying on such techniques for F_1 hybrids is probably going to be only modestly, if at all, successful.

   Normally, the increased costs of an F_1 hybrid program are absorbed in a commercial enterprise: however, non-profit institutions are unable to do this. For these reasons, F_1 hybrid tomato variety development programs have not been as popular as standard variety development programs.

2) *Increased cost of seed production* is a very distinct disadvantage of F_1 hybrids. Seed is obtained from crosses which have to be hand emasculated and pollinated. When seed of standard varieties is available for $1 5 to $30 per pound, most farmers, growers, and plant producers are somewhat reluctant to pay $250 to $350 per pound for hybrid seed.

Methods of reducing the above disadvantages of F_1 varieties are:

1) Several plant producers are now successfully growing tomato transplants in florida. For the most part these are some type of containerized or self-containerized transplant. The number of transplants which can be produced from a pound of seed will normally vary from 100,000 to 250,000, depending upon seed size and germination. When it is considered that a farmer will transplant from 2,500 to 5,000 plants per acre, the cost of seed is a very small factor when transplants are utilized.

2) Improved seeding techniques have also reduced the amount of seed required to sow an acre of fresh market tomatoes in Florida. Advancements in precision seeders as well as advancements in pelletizing seed now make it feasible to direct-seed and obtain a near 100 percent stand without thinning.

   The development of plug-mix seeding has also made the use of F_1 hybrid varieties appear more feasible. Presently, plug-mix seeding is practiced extensively in all tomato producing areas of Florida and is becoming more popular each season.

   F_1 hybrid varieties are better than standard varieties for both plug-mix and precision seeding because of increased germination and emergence. If near

100 percent germination can be achieved, and if seedlings will emerge uniformly, then the amount of seed used per acre can be reduced by at least 65 percent. Furthermore, if it is possible to achieve stands of near 100 percent, then the cost of thinning will be eliminated. Elimination of thinning costs alone would more than offset the increased cost of hybrid seed.

3) F_1 hybrid varieties produced using male sterility will serve to reduce seed costs further. The advantages of F_1 hybrids produced using male sterility are the same as for conventionally produced hybrids plus the following:
   a) female parents are easy to maintain, and no emasculation is necessary.
   b) cost of commercially produced seed would be considerably less than for conventional hybrids.

PROBLEMS IN BREEDING TOMATOES FOR MULTIPLE DISEASE RESISTANCE

Pathogen variability, the number of genes controlling disease resistance reactions, and desirable horticultural characteristics are the most important factors to consider in developing multiple-diseaseresistant tomato varieties.

In tomato, as with most crops, genetic diversity of the pathogens inciting diseases has been ignored for the most part. This has not been due to a lack of interest, but rather lack of techniques with which to explore the genetics of many pathogens. One notable exception is the leaf mold fungus, *Cladosporium fulvum,* which has been studied intensively. Knowledge of pathogen genetics becomes more and more critical to the success of multiple disease resistant varieties as more and more resistance genes are incorporated into a single variety.

Resistance or tolerance or both have been identified for most of the major tomato diseases occurring in Florida and elsewhere (Table 2). But only a very few of these pathogens have been investigated with regard to the genetics of resistance in tomato. Many of the inheritance studies have been inadequate, and for most resistances listed in Table 2 no genetic studies have been conducted. For this reason, only a minimum number of genes can be deduced. For all those characters which behaved as polygenic, the number of genes controlling the resistance reaction was estimated as two. In actual fact, there are probably many more than the estimated minimum total number of 65 (Table 2). Many of these resistances are being evaluated by University of Florida plant pathologists and horticulturists and are being incorporated into tomato variety development programs as rapidly as possible. It is obvious that the more genes for disease resistance the plant breeder has to manipulate, the more difficult his job is.

Artificial as well as field screening tests have shown some University of Florida tomato breeding lines to be resistant or tolerant to 17 diseases. The number of genes controlling these specific resistance and mechanisms is estimated to be a minimum of 33. To incorporate another disease resistance factor into one of these inbred lines, it is necessary to cross the line with a parent which possesses the gene controlling resistance to the new disease. If the new disease resistance parent does not posses any of the resistance genes of the multiple disease resistant line, the F_2 population will segregate for susceptibility to all 18 diseases.

Table 2. Genetic resistances or tolerances to diseases currently utilized and/or evaluated in the Florida Tomato Breeding Program.

Disease	R or T[a]	Minimum no. of genes	Disease	R or T	Minimum no. of genes
Nailhead rust	R	1	Fusarium wilt	R&T	4
Early blight	R&T	3	Verticillium wilt	R&T	3
Gray leaf spot	R	1	Southern blight	T	2
Cladosporium leaf mold	R	3	Bacterial wilt	T	4
Late blight	T	2	Bacterial canker	T	2
Cercospora leaf spot	R	1	Bacterial leaf spot	T	2
Target leaf spot	R	1	Bacterial soft rot	T	2
Septoria leaf spot	T	2	Sour rot	T	2
Phoma black spot	R	1	Rhizoctonia fruit rot	T	2
Powdery mildew	T	2	Rhizopus soft rot	T	2
Gold fleck	R	1	Alternaria fruit rot	T	2
Fruit pox	R	1	Stemphylium fruit spot	T	2
Autogenous necrosis	R	2	Blossom-end rot	R&T	3
Various nematodes	R&T	3	Graywall	T	2
			Virus diseases	R&T	7
Minimum total number of genes for tolerance and resistance = 65					

[a] R = resistance, T = Tolerance.

Assuming each resistance is controlled by single dominant genes, to recover in the F_2 generation one plant which is resistant to all 18 diseases, it would be necessary to grow, on the average, and inoculate successfully, without any escapes, 262,144 plants. This would require approximately 88 acres if the average plant populations per acre were those for fresh market tomatoes as grown in Florida. The enormity of this problem is overwhelming. Considering that our knowledge of tomato pathogen genetics is extremely scant, and our knowledge of tomato host genetics is only slightly better, it is obvious that present techniques of developing multiple disease resistant varieties need improvement.

Present techniques dictate the best procedure to follow in incorporating resistance to the eighteenth disease into a new tomato variety is to initiate a backcross program with a desirable multiple disease resistant breeding line as the recurrent parent, and screen for as many diseases as possible in each breeding cycle. Several such programs are actually underway, but progress with such a program does not occur at a rapid rate.

The next factor to consider is the horticultural characteristics of a variety. Selection for desirable horticultural characters will be made in each breeding cycle, but when emphasis is placed on resistance to 18 different diseases, many of the good horticultural type segregates may be lost.

One of the alternatives to this problem is development of F_1 hybrid varieties. The new resistance gene is incorporated into a parent of good horticultural type and good combining ability as soon as possible to develop a parental line. This parent can then be crossed with the multiple disease resistant line which has good horticultural characteristics, and an F_1 hybrid variety can be produced which not only has good horticultural characteristics but also is resistant or tolerant to all 18

diseases. The level of tolerance to certain diseases would be expected to be lower in the F_1 hybrid than in the multiple disease resistant line, because the tolerance genes would be diluted. Even so, such an approach has tremendous advantages over the current methods utilized.

During its development and after release of the F_1 hybrid, the backcross program with the multiple disease resistant line and the new resistance line would be continued to synthesize a new inbred line with resistance or tolerance to all 18 diseases. This breeding line would then provide the initial genetic material for development of a future parent when the next new disease arises. In addition, breeding lines which contain the tolerance genes as well as the gene for resistance to the new disease would be developed for use as parents to provide the maximum of tolerance genes in the F^1 hybrid variety.

TOMATO VARIETY DEVELOPMENT BY THE UNIVERSITY OF FLORIDA

Historical aspects

The Florida tomato breeding program was formally established in 1925 at the University of Florida Tomato Laboratory in Palmetto, Florida, at the request of the Florida growers led by Mr. J. P. Harllee, Sr., who donated five acres of land for this purpose. Leaders of this new program were two plant pathologists, Dr. George F. Weber and Mr. David G. A. Kelbert. The program was in reality established by Weber in July 1922, but no official commitment was made until 1924, when the state legislature established the Tomato Laboratory. Objectives of the tomato breeding program were to develop varieties adapted to Florida's environmental conditions and resistant to the prevalent diseases. The most serious diseases in 1924 were Fusarium wilt and an unknown foliage disease complex. Weber began work on the unknown complex, and in a series of experiments he demonstrated that several specific pathogens were involved. He differentiated between the tomato pathogens *Alternaria tomato, Alternaria solani,* and *Stemphylium solani* and the diseases they caused which are, respectively, nailhead rust, early blight, and gray leafspot. These fundamental plant pathological studies formed the basis of the breeding program. Weber located resistance to nailhead rust and incorporated this resistance into *Fusarium* wilt tolerant breeding lines. One of his significant accomplishments as a plant breeder was the identification and selection of the nailhead rust-resistant, as well as Fusarium wilt tolerant plants which were subsequently released by the USDA as the varieties 'Marglobe' and 'Glovel.' These varieties were widely grown throughout Florida and other states for several years. Had not these basic plant pathological studies been conducted and these varieties developed, Florida's faltering tomato industry would undoubtedly have collapsed. He and Kelbert eventually released three varieties, 'Newell,' 'Cardinal King,' and 'Ruby Queen,' which were very tolerant to *Fusarium* wilt and resistant to nailhead rust (Table 3). Weber also supplied breeding lines to other tomato breeders who utilized these resistances in their program.

Significant developments by the Univ. of Florida Tomato Breeding Program

Since its inception in 1922, the University of Florida tomato breeding program has

Table 3. Tomato varieties developed by the University of Florida.

Variety	Date released
Marglobe (COOP. USDA)	1925
Glovel (COOP. USDA)	1935
Newell	1940
Cardinal King	1940
Ruby Queen	1940
Manasota	1949
Manahill	1949
Manalucie	1953
Homestead (COOP. USDA)	1953
Manalee	1954
Indian River	1958
Manapal	1960
Floralou	1962
Floradel	1965
Immokalee	1966
Tropi-Red	1967
Tropi-Gro	1967
Tropic	1969
Walter	1969
Florida MH-1	1971
Floradade	1976
Floramerica	1977

emphasized development of high yielding, high quality, multiple disease resistant fresh market varieties. Nineteen such varieties have been released by the program and these have been grown around the world along with three varieties developed cooperatively with the USDA (Table 3). Where they are not utilized directly as varieties, they have been used as breeding lines and gene sources in breeding programs in numerous other states and countries. Currently, many of the popular F_1 hybrid varieties utilize one and sometimes two University of Florida breeding lines or named varieties as parents. The University of Florida tomato breeding program has produced five major accomplishments:

1) The first was the complete eradication of a pathogen through the use of host resistance. When resistance to the causal agent of the nailhead rust disease was identified by Weber and incorporated into the varieties being grown, the pathogen was apparently eradicated. There has been no confirmed report of nailhead rust in the United States for many years. As far as the authors are aware, this is the only known instance of eradicating a pathogen with host resistance.
2) Another major accomplishment in plant breeding history was the development and release of the variety 'Manalucie' in 1953. In this variety, Dr. J. M. Walter combined genetic resistance to eight different diseases with many desirable horticultural characters. Connoisseurs of fresh market tomatoes consider the fruit of 'Manalucie' to be unsurpassed in flavor, texture, and quality. The development of 'Manalucie' demonstrated that multiple disease resistance was a feasible objective. No other variety of any crop had ever been developed with resistance to so many diseases.
3) In the early 1960's race 2 of Fusarium wilt was discovered in Florida and it posed a serious threat to the Florida tomato industry. In 1969 the variety

'Walter' was released in honor of Dr. J. M. Walter and it was the first tomato variety released which was resistant to race 2 of Fusarium wilt. In addition to race 2 resistance and the multiple disease resistance possessed by 'Manalucie,' 'Walter' has tolerance to catface, graywall, tobacco mosaic virus and fruit cracking and produces a high yield of large, deepglobe, smooth, firm and flavorful fruit. 'Walter' is the most widely winter-grown hand harvest tomato variety.

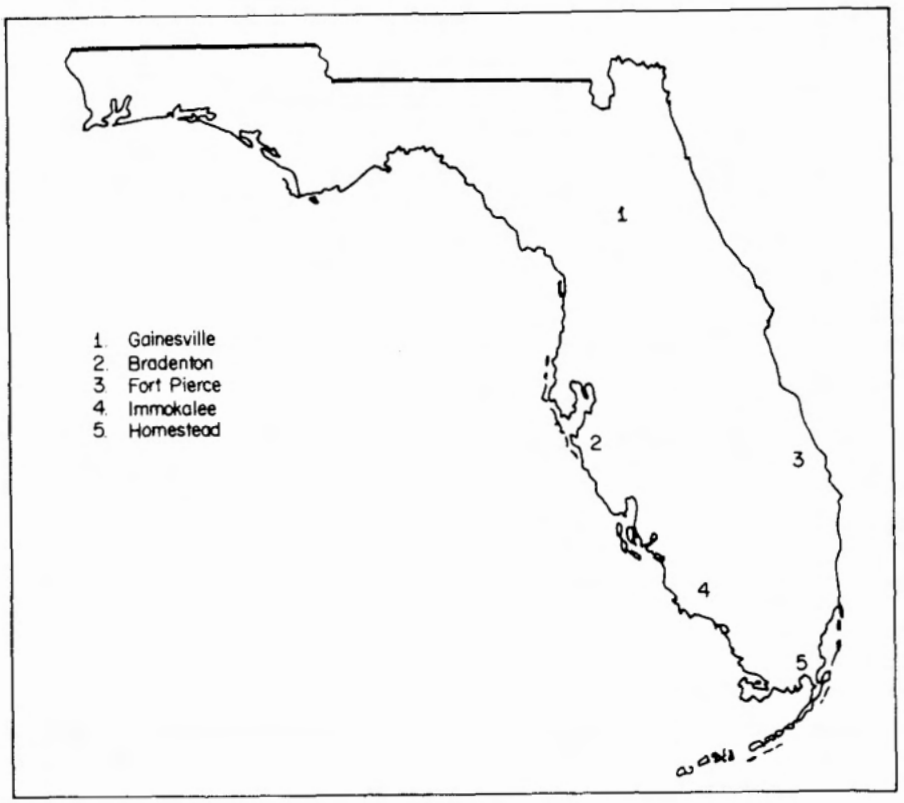
4) The release of 'Florida MH-1' provided the first fresh-market tomato suitable for machine harvest. In addition to this, 'Florida MH-1' had a minimum of 33 disease resistance genes which is considerably more than has been reported for any other variety of any crop species. Because of very firm fruit texture, freedom from cracking, and the inherent tolerance to the causal agents of fruit rot, 'Florida MH-1' was recognized as a high quality tomato available to the consumer of fresh market winter-grown fruit.
5) Floramerica was the first Florida variety to ever win recognition as an All-America selection. Floramerica received adequate points for a bronze medal award in the 1974 All-America trials and was the tenth variety to ever achieve such a high rating. It was the third variety developed with public research funds to ever receive such recognition and the first since 1940. Floramerica received many votes from the All-America judges because of its outstanding performance in a large number of diverse growing areas which was due to its widespread adaptation and multiple disease resistance.

Procedures used in the Florida Tomato Breeding Program

All of the tomato varieties developed and released by the University of Florida, with the exception of Floramerica, have their basis in hybridization, using primarily the pedigree and backcross methods coupled with selection. The pedigree and backcross methods have never been utilized exclusively in developing Florida tomato varieties. A variation of the bulk-population method is being used to develop hot-set varieties for tropical environments; however, the success of this venture is not yet assured. F_1 hybrid variety development has been successful with the release of Floramerica which was designated by the All-America judges as a bronze medal winner.

The current University of Florida tomato variety development program is a cooperative effort involving five research centers (Figure 2) and over 35 University of Florida faculty. Only one of the faculty devotes full time to the tomato breeding program, and most of the 35 spend less than 10 percent of their time on this program. Most are called upon to make evaluations or inputs at strategic times during which a variety is being developed.

Varieties are initiated from crosses, and F_1 populations are normally grown in the field the season after the cross is made. The F_1 would normally consist of 25 to 60 plants, with some populations inoculated with the appropriate pathogen(s), two of the common ones being the incitants of Fusarium and Verticillium wilt. Inoculated plants are evaluated in the greenhouse. Usually 10 non-inoculated or 10 surviving inoculated plants are transplanted to the field and evaluated for various horticultural characteristics and disease resistances. The plants receive



2. Location of University of Florida Agricultural Research Centers cooperating in tomato variety development.

routine sprays of fungicide and insecticide twice weekly until a good fruit set is obtained. Then fungicide sprays are stopped, and the plants are later evaluated for resistance to various foliage diseases. Seed is saved from one to all ten plants of the F_1 depending on homogeneity of the population. Sometimes an outstanding plant is seen in the F_1 and it is selected on the basis of bush and fruit character and freedom from disease. Seed from single or bulk F_1 selections is grown out the next season to produce the F_2 population.

The size of the F_2 population evaluated in the greenhouse varies from 50 to 5,000. The size of an average F_2 is about 500 plants in the greenhouse, of which about 150 (diseasefree) aretransplanted to the field. Screening techniques in the greenhouse and field are the same as for the inoculated F_1 populations. Single plant selections are made in the F_2 populations in the field. The basis of selection is the same as that practiced in the F_1. Normally, about 5 to 10 single plants which possess the desirable recombinant characters are selected from an F_2 population. Usually about 1/2 to 3/4 of the F_3 populations produced from F_2 plants contain no plants deemed worthy of selection, and these populations are discarded completely whereas an F_1 is rarely without, a selection being made.

F_3 populations, which vary from 50 to 2,500 plants, are produced from the

singleplant F_2 selections and screened in the greenhouse. About 50 to 75 disease-free or symptomless plants are transplanted to the field. At this time it is possible to determine which populations have homozygous resistance to the various pathogens, since the F_3 is actually a progeny test of the F_2 when conducted in this manner. Again the superior plants in the F_3 are harvested as single plants. More than 50 percent and sometimes as high as 90 percent of the F_3 populations are discarded. The remainder are maintained by selecting the superior plant or plants within the F_3.

These singleplant F_3 selections are used to produce the F_4 populations. If a particular F_3 selection was classified as outstanding, it would normally be entered in an observational nursery and compared with standards. Only in very rare instances is an F_3 selection evaluated in the Replicated Yield Nursery. Normally the F_4 population evaluated in the greenhouse is about the same size as the F_3. The F_4 field populations are about 1/2 - 2/3 the size of F_3 populations. Single plants are selected from the F_4 to produce the F_5 and often from the F_5 to produce the F_6. Quite often F_5 populations are bulk-harvested to provide large quantities of seed for widescale testing; however, breeder's seed is always produced from screened single plant selections.

At no time during the development of an inbred line or of a candidate for variety release is a bulk selection made with the exception of the F_1 population. Bulk selections are made for seed increases for testing purposes. The maintenance of crosses and lines by single plant selections provides two distinct advantages to the plant breeder:

1) homozygosity and uniformity is attained as rapidly as possible, and
2) each generation is a progeny test of the previous generation for all characters concerned.

As presented above, it appears that each crop season is devoted to a single generation. This is not the case. Rather, crosses are made each season and evaluated, and F_1, F_2, F_3, F_4, F_5, F_6 and advanced lines are also evaluated each crop season. Approximately40 percent of the plot acreage each season is planted to F_2 populations, 15 percent to F3 populations, and the remainder to F_1, F_4, F_5, F_6 and advanced generation lines.

Three hundred to five hundred crosses are made each season, resulting in nearly this many F_1 and F_2 populations to be evaluated. Selection in the F_2 is very rigorous. If the segregates do not measure up in both horticultural characteristics and disease resistance, the population is dropped. The most common reason for discarding a population in the field is failure to attain adequate fruit size or to maintain a high proportion of superior marketable fruit.

Disease screening with various pathogens is conducted on all populations except for those few which have special characters that render them otherwise valuable. Usually these lines are valuable for only one reason or characteristic, and in this sense the line itself is not valuable; only the gene or very few genes controlling a characteristic are considered valuable. When a line with a particularly valuable characteristic is discovered, the line is not incorporated per se into the breeding program. Rather the line is crossed with other lines in the program which have desirable characters, and the desirable character of the first line is combined

with the desirable characters of the second line. The resistant progeny which possess all the desirable characters are then maintained in the program. The original line carrying the desirable character is stored and usually not utilized again. This provides the fundamental basis for the gene pool method of breeding as devised by John B. Sieglinger at Oklahoma State University which has been used extensively in the University of Florida tomato breeding program.

The disease screening tests in the greenhouse are normally quite precise and accurate. Screening tests in the field are another matter. Tests can be effectively conducted in the field for a few diseases but the field method is unsuitable for testing resistance to a number of important diseases. Resistance to bacterial leafspot and gray leafspot can be evaluated quite effectively in the field. Often a large number of susceptible plants escape disease with one population or generation, but these are located and identified in the next generation, using the progeny test as described earlier.

Several screening tests cannot be conducted at AREC-Bradenton, including that for southern bacterial wilt, old land syndrome, and Septoria leafspot because of environmental conditions. In these cases the resistant breeding lines and populations must be evaluated by establishing small scale nurseries in areas where the disease is a problem. In these studies the objective is to establish as soon as possible if resistance exists, how it is inherited, and how feasible field screening is for the disease. The results of these studies are then incorporated into the major breeding program, and progeny must be evaluated not only at AREC-Bradenton but in the locale where the disease is a problem. In this way multiple diseaseresistant varieties with wide ranges of adaptation are developed.

The development of F_1 hybrid varieties is a very important part of the program, and the potential use of male sterility in F_1 hybrid production is being investigated. The University of Florida malesterile tomato was discovered in February 1969 in a commercial field owned by Mr. Lewis Rauth of the Flavor-Pict Corporation at Delray Beach, Florida. Additional malesterile plants were obtained from various tomato growers' fields in Florida, and from Dr. T. O. Graham of the University of Guelph. All were evaluated and found to be lacking in one or more qualities.

The steriles evaluated were of three apparent types:

1) anthers and pollen present but pollen nonfunctional (most likely these types were aneuploids or cytoplasmic in nature),

2) anthers and pollen both present and functional, but the stigma extended above the anther column so pollination could not occur (commonly referred to as functional sterility), and

3) anthers reduced to vestigial remnants and no pollen produced (commonly referred to as genetic sterility or stamenless).

The type3 sterility was found to be highly desirable for F_1 hybrid seed production. This naturally occurring 'Floradel' mutant (Figure 3) has not been characterized except that its expression is apparently controlled by a single recessive gene. The mutant appears very similar to stamenless one (sl_1,) and flower structure appears different from sl_2.

Present male-sterile lines bear little resemblance to the original 'Floradel' mutant. The original plant was a tall, rank indeterminate. Pollination of the first

3. An individual flower from a plant homozygous for the Type 3 male sterility obtained from Mr. Lewis Rauth, Falvor-Pict Corporation, Delray Beach. Florida. Vestigial remnants of the anther column are collapsed around the style and partially cover the stigma.

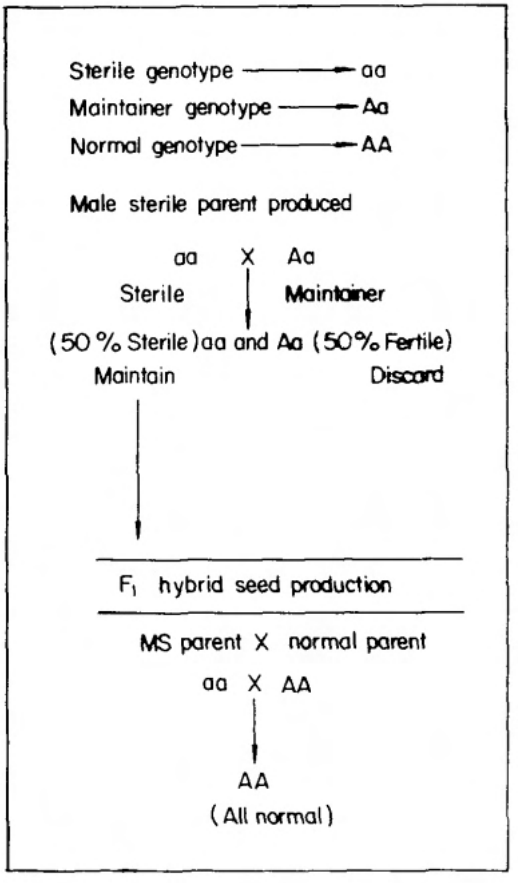
flowers produced extremely deformed and very unattractive fruit. On some flow ers the anther vestiges were developed to such a degree pollen was produced, but it was not ascertained to be either viable or non-viable.

The immediate goal of the University of Florida tomato breeding program is to develop male-sterile 'Walter' and 'Florida MH-1' lines and to convert those breeding lines of high general combining ability to steriles for use in developing F_1 hybrids.

The mechanics of seed production are described in Figure 4. The sterile parent for seed production is derived from two lines: a sterile line and a maintainer line. Presumably the sterile line would differ from the maintainer line only for the character controlling sterility, with all other characters being identical.

The pollinator parent is no different from the usual pollen parent of conventional F_1 hybrids. The same techniques of pollen collection are employed and pollinations are made in the same manner except for emasculations which are done genetically.

The advantage of the Florida fresh market tomato industry relying on F_1 hybrid varieties is the speed with which the breeding programs can respond to industry needs. For example, if a new disease or new race of a present pathogen was to occur in Florida, it should be detected with the present survey system for tomato diseases. An immediate screening program for resistance would be initiated, and a resistance gene or genes would presumably be located. Intensive evaluation efforts should yield enough information within a year to determine if production of an F_1 hybrid with the available resistance is feasible. If such is the case, a suitable



4. Schematic flow-sheet for production of F_1 hybrid tomato varieties using derivatives of the Rauth sterile.

F_1 hybrid could be synthesized quite rapidly, and the hybrid would be recommended for use only where the new disease is a particular problem. Such a procedure quickly provides a usable variety, so growers can remain in the business of producing tomatoes. Since the F_1 is produced using a multiple disease resistant parent of high combining ability in conjunction with the parent carrying resistance to the new disease, the resultant hybrid variety should be acceptable in most instances.

This advantage will not exist for some new diseases. In general, resistance to most tomato pathogens is controlled by a single gene, and in most instances this gene is dominant. With all such cases it is only necessary to have the resistance gene in one parent. If the gene controlling resistance is recessive, then the gene must be present in both parents for resistance to be expressed. If more than one gene is involved, as with polygenic tolerance mechanisms, the level of tolerance in the F_1 will depend upon how many different tolerant genes can be inserted into

each parent. Obviously, tolerance mechanisms are not nearly as desirable as monogene resistances from the breeding and management aspects of tomato variety development.

It has been demonstrated in numerous host-pathogen interactions, although not in tomato, that resistance genes against different races may be allelic and also that multiple loci may occur. The method of control which has been recommended by plant pathologists for such cases in which host resistance is utilized, is use of polygenic tolerance, sometimes referred to as horizontal resistance. This is the easiest way out for the plant pathologist, but sometimes does little good for the grower. In areas of highly specialized agriculture, the use oftolerance or horizontal resistance is completely unacceptable when manageable monogenic resistance is available. Routine surveys for diseases conducted by plant pathologists, if properly managed, contain enough information to permit epidemiologists to predict the occurrence and severity of any disease reasonably well. Such predictions are qualified by environmental factors such as rainfall, temperature, pathogen race and host variety. If it is observed that a certain race of a pathogen is becoming increasingly prevalent, then an epidemiologist could recommend the planting of a variety resistant to the particular race. The pathogen population would then be forced to evolve on the resistant line, and the race capable of attacking the resistant variety would become less prevalent or extinct. If the pathogen population evolves a new race, capable of attacking the resistant variety, it would be detected by the disease survey, and the breeders could substitute an alternate variety which would be resistant to the new race.

If such a system were effected, the plant breeder's job would be made much easier in that the effects of multiple loci and allelic resistance genes would be eliminated by plant pathologists in annual pest management recommendations for a crop production system. A system of this type would eliminate those losses which the farmer would endure if polygenic tolerance or horizontal resistance were selected by the plant breeder and pathologist for disease control.

The Ronda tomato breeding program is not confronted with such a problem of multiple alleles, and evidently multiple alleles are not common in most tomato disease resistances. If such a problem in developing diseaseresistant varieties does arise, then the F_1 hybrid program coupled with an extensive statewide tomato disease survey should provide adequate protection for the industry.

SUMMARY

The University of Florida tomato breeding program was initiated in 1922 by George F. Weber and brought to fruition by James M. Walter. Under the leadership of these two plant breeders-pathologists, the florida tomato variety development program became recognized as one of the world's classic plant breeding programs. Tomato varieties developed by the University of Florida tomato breeding program have been the primary factor in maintaining the viability of the Florida tomato industry. Tomatoes are the second most important crop in Florida, ranking only behind citrus, and contribute greatly to maintaining a healthy econ omy within the state. In addition, Florida varieties have been utilized in most other

states as well as various countries in the tropics and sub-tropics. It is impossible to place a monetary value upon the contributions of the University of Florida tomato breeding program to the economic well-being of the World. The 1974-75 tomato crop produced exclusively with University of Florida varieties in Florida and Mexico and marketed in the U.S. was nearly 62 million cartons. The average on-farm price for this crop was in excess of $6.00 per carton, which results in over $375,000,000 on-farm sales by tomato farmers. It is also significant that tomato growers the world over recognize Florida varieties by name and use them because of their multiple disease resistance and the fact that they result in a high quality product for the consumer.

ACKNOWLEDGEMENTS

In a manuscript such as this it is not possible to give credit to those who originated most of the ideas presented and discussed. The authors make no claim for originating any of the ideas or philosophies presented in this paper. Literature citations have been kept to a minimum as we felt it was appropriate with a subject such as this. It is unfair to give credit with a single citation, and omit all the others which have contributed to the understanding of a basic concept. It becomes prohibitive to tabulate and publish all of the references which are available for all of the ideas presented in this paper. The contribution of original ideas and concepts from R. A. Peck, F. F. Davies, J. B. Sieglinger, E. W. Hanson, D. J. Hagedorn, P. H. Williams, R. H. Painter, S. Diachun, R. R. Nelson, J. W. Strobel, and J. C. Walker are gratefully acknowledged.

The University of Florida tomato breeding program has been a long-term and continuing effort on the part of many people. The current program represents our efforts to utilize new methods and build upon the tremendous mass of tomato germ plasm which has been collected, selected, and developed by G. F. Weber, A. L. Harrison, J. M. Walter, J. W. Strobel, R. E. Stall, and others. Future successes in the Florida tomato breeding program will be based upon the past efforts of these people in collecting and maintaining a broad base of germ plasm. Likewise, one of the important aspects of maintaining a viable tomato breeding program for the future will be to increase and maintain a broad-based gene-pool from which characteristics can be drawn for use in future varieties.

In most successful research programs the technicians contribute considerable information and ideas as well as manage and conduct the routine affairs of the program. This has certainly been true with the University of Florida tomato breeding program, and the assistance and expertise of Senior Technicians Tommy H. Cline and John Pressley, Jr., who have a combined total of 60 years experience with the tomato breeding program, are gratefully acknowledged.

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An assessment of stabilizing selection in crop variety developmen[1]

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INTRODUCTION

There is a limited amount of published information on the subject of stabilizing selection. The authoritative treatises on stabilizing selection do not, for the most part, contain data collected from experiments designed to test the validity of the hypothesis (44, 49, 50, 52, 58-60). Papers that do deal directly with the subject have been limited to studies of one host and one pathogen (5, 12, 14-18, 32, 33, 36-38, 41, 46, 47, 53, 54, 57), from which it is difficult to draw conclusions with general applicability to the majority of host-pathogen interactions.

Based solely upon proven scientific importance, there is inadequate information to justify the preparation of a review article on the subject of stabilizing selection. One has only to refer to the publications of Nelson (44, 45), Van der Plank (58-60), and Horsfall (31) to be familiar with the bulk of the information in print concerning stabilizing selection. The concept of stabilizing selection deals directly and intimately with the production of food through the utilization of new varieties. The world press (television, radio, magazines, newspapers) has popularized these subjects indirectly through news stories that deal with food production, and especially with the failures of food production systems around the globe. Every person exposed to the free-world press was made aware of the achievements of Norman Borlaug when he won the Nobel Peace Prize for developing new crop varieties. These new varieties provided the basis for the "green revolution," which was intended to make the famine-stricken people of underdeveloped countries self-sufficient in food production. Likewise, people everywhere were madeaware of the sales of American grain to Russia when the Russian small grain crop production failed, and of the resultant rise in the price of wheat and bread in

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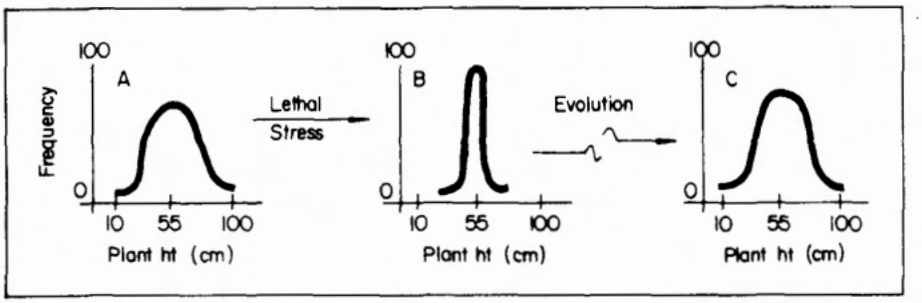
the USA. More recently, we were made very aware on a daily basis of the effects of the drought in Europe during the summer of 1976, and the effects of this on world food production. We are continuously bombarded with news reports of the rise in the cost-of-living index and of the increased price of various key foods. Today, consumers are more conscious of food costs and food production disasters than ever before. Because the consuming public is aware of these facts, the politicians are aware of them, and the politicians exert considerable effect on research through their control of funds. J. B. Kendrick, Jr., vice-president of the university of California, summarized this situation when he stated that "public policy and research funding are affected by the opinions, sentiments and fears in the public mind, legislative halls and governmental agencies" (35). He concluded, "Simplistic solutions and distrust of science can only result in losses for the farmer, consuming public and the hungry world." Abelson (1) and Kantrowitz (34) also provide interesting insights into the politics controlling research. In addition to these political and crop production events, three primary factors are responsible for the attention currently being focused on stabilizing selection and genetic vulnerability: (a) the publication of the revised concept of stabilizing selection as it pertains to host-pathogen interactions (59), (b) the occurrence of the corn leaf blight epidemic in the United States (31), and (c) the publication of *Genetic Vulnerability of Major Crops* by the National Academy of Science (31). If any one of these events had not occurred, or if they had not occurred in such a short timespan, this topic probably would not have been considered by Annual Reviews. This review offers an analysis of stabilizing selection as it pertains to the development of crop varieties. It may appear that this paper has to do more with plant breeding than plant pathology, but the reader should keep in mind that the "new" concept of stabilizing selection was coined, published, and promoted by plant pathologists and was presented as a criticism of plant breeders' past efforts (59). What follows, therefore, is not a review in the usual sense, but rather an assessment of the concept of stabilizing selection and of the ways it can be used in crop variety development to best utilize host resistance as a means of disease control.

CLASSICAL CONCEPTS OF STABILIZING AND DIRECTED SELECTION

Stabilizing Selection

Stabilizing selection is a relatively old concept of classical evolution which explains why species under natural conditions remain more or less constant and relatively unchanging. This concept was first hinted at by Charles Darwin in 1859 (20), supported with experimental evidence by Bumpus in 1899, and is generally accepted today (19, 24). Present-day theorists of evolution have considered stabilizing selection to be the mechanism whereby a species maintains its status quo within environmental extremes (40).

Any population of organisms can be analyzed for various specific characters, and the data for any single character quantified and plotted in graph form. A hypothetical example is diagrammed in Figure 1 using plant height as the character being measured. The frequency of distribution within the population for



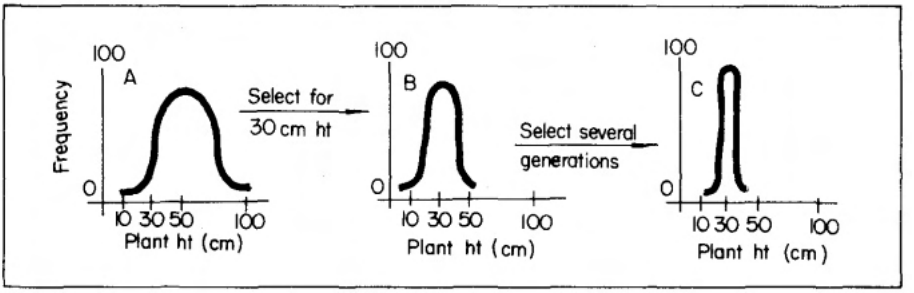
1. Hypothetical occurrence of stabilizing selection in a naturally occurring plant population subjected to environmental stress. A. Frequency distribution curve illustrates plant height of the individuals comprising the population. B. individuals surviving the stress exhibit the fewest deviations from the mean plant height of 55 cm. C. Through evolution the population eventually returns to the original distribution with a mean plant height of 55 cm.

plant height results in a normal distribution curve, that is, the bulk of the population centers around a mean (55 cm) and only a small percentage is found at the extremes (10 and 100 cm) (Figure 1A). A similar normal distribution curve will result when any character is measured for which selection pressure is not being exerted. When such populations are subjected to lethal stress, and the majority of the individuals of the population are killed and the survivors measured, the survivors are without exception derived from the mean or near the mean value of the original population (Fig 1B). Individuals that survive the near-lethal environmental stress are those that exhibited the fewest deviations from the original population means for all measurable characters. This basic concept of stabilizing selection has long been accepted by evolutionists and cannot be seriously challenged (26, 40).

Directed Selection

Directed, or directional, selection is a concept closely associated with that of stabilizing selection. Directional selection is the mechanism whereby the population of a species responds to environmental extremes by phenotypic shifts (40). For the most part, there are probably very few alleles gained or lost from the total population in this process. The phenotypes change because of the pressure of directional selection, but the amount of genetic material within the population remains relatively constant. The Hardy-Weinberg law (55) states that the relative frequency of a gene within a population will remain constant unless there is selection, either for or against those traits governed by the gene. Therefore, the relative frequency of any given gene in a population is dependent upon selection pressure. If the population of any organism has evolved to approach maximum adaptability within its environmental extremes, there will be little or no change in gene frequency. The dominant selection pressure that is operative in this instance is stabilizing selection. If the population is in an evolutionary state such that deviant phenotypes have a selective advantage, then directional selection probably will be operative.

Directed selection can be diagrammed starting with the Same initial hypothetical population which was used to illustrate stabilizing selection (Fig. 2). The wild



2. Hypothetical occurrence of directed selection in a naturally occurring population when selection pressure favors plants 30 cm high. A. Frequency distribution curve illustrates plant height of the individuals comprising the original population. B. The frequency of 30 cm tall individuals is increased with positive selection pressure. C. With continued selection pressure for plant height of 30 centimeters almost all individuals in the population are 30 centimeters tall.

population depicted (Fig. 2A) varies in plant height from 10 to 100 cm with the mean height being 55 cm. If for any reason environmental stress is applied which favors plants that are at least 30 cm tall but not more, there will be a sudden shift in the mean plant height of the population (Fig. 2B). If selection pressure is maintained to favor plants which are only 30 cm tall, the frequency distribution of the population for plant height will eventually appear as in Figure 2C.

THE NEW CONCEPT OF STABILIZING SELECTION

In 1968, Van der Plank applied the terms stabilizing selection and *directional selection* to host-parasite interactions (59). He was very critical of the past efforts made by plant pathologists and plant breeders in utilizing host resistance to control plant diseases. The basis for his criticism was that there are two types of host resistance:vertical and horizontal. He contended that the success of either of these two types of resistance is due to stabilizing selection, and that failures of diseaseresistant varieties in the past have been due to failure of the plant breeders in manipulating the host resistance genes so that stabilizing selection would operate favorably.

Stabilizing Selection with Vertical Resistance

"When a variety is more resistant to some races of a pathogen than to others the resistance is called 'vertical' or 'perpendicular' (59). Vertical resistance reduces the effective amount of initial inoculum from which the epidemic starts and thereby delays the observed start (59). A feature of vertical resistance is that the infection rate is as fast in the vertically resistant as the completely susceptible variety after the initial infection has occurred (59). Most vertical resistance apparently is controlled by single genes (23, 50, 61). Van der Plank assessed vertical resistance as follows (59): "The effects of vertical resistance are strong in seasons of little disease when resistance is not very important, but weaker in seasons of much disease when resistance is most needed. It is a matter of history that all the great disappointments with vertical resistance have been in seasons of unusually high infection rate." Thus, vertical resistance is portrayed as the basis for a "boom

and bust" cycle of variety production (59). The plant breeder develops varieties which on leaving the nursery are resistant because there are no prevalent races that can attack them. The vertical resistance delays the epidemic for such a long time that no disease develops in the new variety. If the variety is agronomically successful, it will become more popular each year until it occupies 100% of the acreage, and at this time vertical resistance fades away (59). Van der Plank concludes (59) that the plant breeder is in a ludicrous position. The breeder hopes his new variety will be popular among farmers and that it will maintain its resistance, but his two hopes are likely to be mutually exclusive.

Vertical resistance in the host dictates which races of the pathogen survive. If the pathogen does not possess the genes required to incite disease in the host population, negative selection pressure occurs against the nonpathogenic portion of the population. This is directed selection pressure as illustrated in Figure 2 and discussed previously.

Van der Plank (59) states that when a vertical resistance gene is present in the host, the pathogen must be able to develop races with virulence to match this gene if it is to survive. He further states that when the vertical resistance gene is absent from the host, and a matching virulence in the pathogen race is not necessary, then stabilizing selection will operate in favor of races of the pathogen that have the fewest genes for virulence. Thus, the races of the pathogen that survive are those that have the minimum number of virulence genes required to infect the host. Stabilizing selection in the pathogen then is portrayed as the basis of effective vertical resistance in the host (59).

The effectiveness of stabilizing selection in the pathogen population is determined by the relative strength of the vertical resistance gene of the host (59). The strength of a vertical resistance gene is in turn defined in terms of the strength with which stabilizing selection acts against the complementary pathogen race (59). "The stronger the gene for vertical resistance, the stronger the pressure of stabilizing selection" (59).

Stabilizing Selection with Horizontal Resistance

When host resistance is equally effective against all races of a pathogen it is termed horizontal or lateral (59). The action of horizontal resistance is to slow down the epidemic after it has started (59). Horizontal resistance can vary from zero or no resistance at all to "practically absolute immunity" (59). Horizontal resistance (59) is portrayed to include a whole array of resistance mechanisms including klenducity, field resistance, and all of those factors formerly considered to be disease-tolerance mechanisms (52).

Van der Plank (59) states, "Underlying horizontal resistance is a stabilizing selection operating against extremes: A genetic homeostasis. This stabilizing selection differs in origin from, but is as real as, the stabilizing selection on which vertical resistance rests." Stabilizing selection in the pathogen population when horizontal resistance is used in the host is attributed to the stability of the races of the pathogen. This stability does not preclude the appearance of new races or the disappearance of old races, but maintains a stable balance among all the various races (59).

Mechanism of Action in Stabilizing Selection

As previously stated, Van der Plank believes that stabilizing selection with horizontal resistance differs from stabilizing selection with vertical resistance. If properly managed, however, both sources of resistance should be stable and enduring (59). Stabilizing selection in favor of pathogen races without unnecessary virulence is considered to be the "force" behind vertical resistance. Those races of the pathogen with excess virulence genes are less fit to survive and will therefore disappear from the pathogen population with time. The logic for this according to Van der Plank is that the pathogen is forced to develop a substitute metabolic pathway for each additional virulence. The degree of inferiority of the substitute metabolic pathway employed by the pathogen to overcome the vertical resistance of the host gene then determines the strength of the resistance gene (59, 61). If such is the case, it is obvious that some pathogens (such as the cereal rusts) are quite adept at shifting from one metabolic pathway to another with little change in effectiveness. Because of this, Van der Plank feels that vertical resistance must be ephemeral and therefore inferior to horizontal resistance which is more stable and longer lasting.

Stabilizing selection in pathogen races without unnecessary virulence is considered also to be the "force" behind horizontal resistance. The mechanism of action is not the same as for vertical resistance, but rather is ascribed to the polygenic inheritance of aggresiveness (59). This is just a restatement of the classical theory of stabilizing selection as illustrated in Figure 1. During each crop season the pathogen population will be exposed to a uniformly susceptible, but horizontally resistant, variety and the survivors of the pathogen population will be all from the mean of the population. The intermediates of the population survive and reproduce while the extremes are lost. As Van der Plank states it, races that survive are those with intermediate aggresiveness, not the extremely aggresive or unaggressive (59).

CRITICISMS OF THE NEW CONCEPT

The concept of stabilizing selection as proposed by Van der Plank in 1968 has been challenged and criticized by plant breeders and plant pathologists (12, 14-17, 23, 32, 33, 44-47, 53, 54). The only lengthy treatise on the subject is that of Nelson (44). All other criticisms of the theories and applications of stabilizing selection are based upon limited data collected from experiments involving for the most part only one host and one pathogen. It seems significant, however, that some of these studies involved the same host and pathogen that provided Van der Plank with much of the data used in formulating his concept of stabilizing selection (12, 14-16, 23, 33).

Acceptance of the New Concept

Van der Planks thesis in 1968 (59) would have probably passed from consideration as a worthy topic of scientific investigation by this time if it had not been for the corn blight epidemic of 1970 and the subsequent events that have been pre viously discussed. The primary message that plant breeders received from this

thesis was that because of stabilizing selection, horizontal resistance is stable and long lasting while vertical resistance is ephemeral and worthless. Because horizontal resistance is quantitative and vertical resistance qualitative (61), horizontal resistance is much more difficult for the plant breeder to manage. Coyne & Schuster (11), Crill et al (l6, 18), and Duvick (22) have discussed in some detail the problems that plant breeders face in developing multiple disease-resistant varieties using only horizontal resistance. It is virtually impossible to manage as many as 25-30 quantitatively inherited disease resistances and incorporate all of them into a single variety. It is also difficult to manage this many qualitatively inherited characters in a breeding program, but at least it is feasible (1 6). Primarily because of this difficulty in managing horizontal resistance, most plant breeders have not used horizontal resistance even though it was portrayed as more stable and longer lasting.

Genetic Vulnerability

Because of the corn blight epidemic in the United States in 1970 and publication of the report Genetic Vulnerability of Major Crops in 1972, the concept of stabilizing selection received considerable impetus (31). The National Academy of Science Committee on Genetic Vulnerability of Major Crops even went so far as to support Van der Planks (59) proposal for federal control of host resistance genes (31).The concept of genetic vulnerability has never been concisely defined, even though the original publication on the subject was 307 pages in length. The concept of genetic vulnerability is very closely associated with the concept of stabilizing selection. The National Academy of Science Committee on genetic vulnerability concluded that plant breeders had used only a very small portion of the available germ plasm, and with respect to disease resistance, only a relatively few monogenes for vertical resistance were being used to confer resistance to the major diseases of important food crops (31). Because most of the varieties within a given crop possessed the same monogenes conferring vertical resistance, they concluded that this comprised a monoculture with respect to disease resistance and rendered the crop genetically vulnerable. Their conclusion (31), as well as that of others (21, 29, 30), was that plant breeders should use more diverse sources of germ plasm and utilize horizontal instead of vertical resistance to reduce the threat of genetic vulnerability.

The situation with respect to stabilizing selection and genetic vulnerability had now polarized. On the one hand was the group (31, 59-61) who maintained that vertical resistance is worthless because it is mismanaged so that stabilizing selection is not operative; and at the other extreme was the group (16, 44, 45) who maintained that vertical resistance is the only feasible way for economical control of the major diseases of crop plants and that there is no such thing as stabilizing selection.

VERTICAL RESISTANCE: SUCCESSES AND FAILURES

The failures of vertical resistance have been extensively enumerated (59). Listed as failures are vertical resistance to stem rust of wheat, crown and stem rust of

oats, late blight of potato, and numerous others (59). The successes with vertical resistance are not so well documented and deserve a brief discussion at this point. Several examples can be cited but only three are discussed here: the nailhead spot disease of tomato, the *fusarium* yellows diseases of cabbage, and then the milo disease of sorghum.

Nailhead Spot of Tomato

Nailhead spot, a fruit, stem, and leaf spot disease of tomato, was quite prevalent throughout the United States and in the late 1920s was considered so serious that predictions were made that the tomato industry would be eliminated if some means of control was not implemented (65). G. F. Weber identified a source of resistance which was incorporated into the currently used varieties and the causal agent, *Alternaria tomato,* apparently was eradicated (12). There has been no bona fide report of nailhead spot for many years, and the pathogen apparently is scarce or possibly extinct. Tomatoes are grown today on vast acreages with no thought given to this disease which once threatened the very existence of the crop. Tomato breeders are even unable to screen their varieties and breeding lines for resistance since cultures of the pathogen are no longer available.

Milo Disease of Sorghum

A second example is the milo disease or Periconia root rot of Sorghum caused by the fungus *Periconia circinata.* The "combine" grain sorghums were developed by John B. Sieglinger, beginning in 191 9 as a new grain-feed crop which could be harvested mechanically (42). He released the first varieties, Beaver and Wheatland, respectively, in l 928 and 1931 ; these were followed shortly with additional improved ones (42). The fledgling industry became widespread in the great plains with hundreds of thousands of acres in production when the milo disease appeared. All varieties were susceptible, and where the disease occurred, entire fields of grain sorghum were killed. It appeared that the newly developed industry was to be eliminated by this new disease. Through perseverance and continuous observation of field after field of disease plants, one field was found with three healthy, normal plants. Seed was saved from these and one plant was found to be homozygous-resistant to the milo disease (56). This single plant contained the source of resistance which is present in all the varieties developed and released since then.

Fusarium Yellows of Cabbage

The third and most widely cited example is the yellows disease of cabbage caused by *Fusarium oxysporum* f. sp. *conglutinans.* In 1930, Walker isolated a vertical gene for resistance to this pathogen which was incorporated into cabbage varieties (62). To date, there has been no report of a new race which can attack the vertical-resistant varieties. It is interesting to note that Walker and associates had access to a good source of horizontal resistance which was not widely used because it was not as stable as the vertical resistance (62).

These three examples represent one extreme of a true situation with respect to stabilizing selection and genetic vulnerability. At the other extreme are the exam

ples cited by the "prophets of doom" who advocate replacement of successful breeding programs which have been based on monogenic (vertical) resistance for programs based upon unproven polygenic (horizontal) resistance. In most instances, the best answers to any problem are to be found between the extremes, and this is probably true also with respect to the use of host resistance for controlling plant diseases.

HOST RESISTANCE IN INTENSIVE AND SUBSISTENCE AGRICULTURE

Crill et a1 (18) have described two types of agriculture, intensive and subsistence. Intensive agriculture is characterized by the utilization of all modern technology to maximize production. Very often pest control, fertilization, irrigation, and other cultural practices are carried beyond economic justification in intensive agriculture. All available technology is used to maximize yields and quality, and many times excessive protective sprays, fertilizer, and irrigation are applied when not necessary. Such agriculture is characteristic of crop production in many areas of the developed agriculture world. In subsistence agriculture, crop production is not nearly so technical and the objective is not the maximum production possible per unit area of land utilizing all of modern technology, but rather to produce merely enough for subsistence. The primary goal of subsistence agriculture is to fill the needs of local markets whereas intensive agriculture is called on to supply not only local but also export markets. It generally is acknowledged that agricultural production must become more intensive each year if the ever-increasing world population is to receive adequate food.

Vertical Resistance in Intensive Agriculture

Crill et a1 (18) also characterized the contrasting requirements for varieties with respect to intensive versus subsistence agriculture. Among the variety requirements for intensive agriculture were maximum yields of a high quality product based upon a good response to fertilizer and irrigation using monogenic disease resistance (vertical resistance). In subsistence agriculture, production of maximum yield of high quality is of less concern. Thus varieties are utilized that can tolerate drought as well as excessive water and can produce a crop with suboptimal plant nutrition and are only disease tolerant (horizontal resistance). The reason for using vertical resistance in systems of intensive agriculture is that disease losses are minimized or even eliminated within a given crop season, whereas, when horizontal resistance is used, some disease losses usually occur each season. When deciding between vertical-resistant and horizontal-resistant varieties, the farmer is actually making one of the following choices: (a) to grow a vertical-resistant variety that may result in no disease loss at all or may be subject to epidemic disease if the appropriate race develops, or (b) to grow a horizontal-resistant variety and contend with an endemic disease problem but not with a potential epidemic. Varieties with vertical resistance react differentially to races of the pathogen, whereas varieties with horizontal resistance react uniformly to all races of the pathogen. If the appropriate race is not present, there is no disease in a variety with vertical resistance. If any race is present there will be some disease with

horizontal-resistant varieties. Historically, farmers have opted for no disease and this is especially true with the modern farmer in intensive agriculture. Each year plant pathologists' disease surveys become more and more inclusive and refined so that farmers can make more intelligent decisions as to which varieties will fit best into their individual schemes of crop production and disease control.

Corn breeders and the corn industry are justifiably proud of the way in which the 1970 epidemic was managed (22). In 1970, US corn losses were estimated at 15% because of the blight epidemic (31). In 1971, significant amounts of seed of resistant hybrids were deployed into the areas of greatest need and in 1972, the nation's farmers were provided a full supply of resistant seed (22). This single example illustrates the rapidity with which an industry can shift to other types of resistance in intensive agriculture. Such is not the case with farmers in subsistence agriculture. The technology simply is not available to these farmers whereby they can produce the maximum potential yields. Furthermore, if they did produce the maximum yields possible, they would probably suffer economic disaster because one of the characteristics of subsistence agriculture is an inadequate storage and marketing system (18).

Horizontal Resistance in Subsistence Agriculture

It therefore appears that horizontal resistance is of much greater benefit to farmers in subsistence agriculture because stabilizing selection will operate to maintain yield losses at a tolerable level and prevent catastropic epidemics. Most of the examples of vertical resistance cited as failures have been taken from subsistence agriculture. At the time these epidemics occurred, the system of agriculture in which they occurred was considered to be intensive. Compared with today's modern agriculture, however, the systems in which they occurred would be less than intensive and in many instances subsistent by present standards. Only one such example (oat breeding) is discussed here. It was chosen because it is the same example used to illustrate the problems of disease resistance when vertical resistance was utilized (59). It was noted that oats have been widely cultivated since before the Christian era and that before 1940 the crop survived without any vertical resistance (59). In the decade prior to the time plant breeders began to incorporate vertical resistance into oat varieties, the combined losses of oats to all diseases in the United States was estimated to be 2.8% of the crop. In the decade after oat breeders had incorporated vertical resistance to stem and crown rust, a new variety was needed every 4 or 5 years to counteract changes in the races of the pathogen. It was also pointed out that these vertical-resistant varieties provided significant increases in yield during the same time period, but since new varieties were needed periodically it was implied that the crop had deteriorated because of the oat breeders' ineptness (59).

To appreciate what happened in US oat production from 1930-1950, it is necessary only to note that oat cultivation changed from a system of subsistence production to one of intensive production. During this relatively brief time, several major changes in crop production occurred. Initially, oats were sown broadcast with low plant populations; this was changed to drill planting in rows to achieve uniform stands of higher plant populations. The method of harvesting changed

from hand cutting with a cradle, scythe, or the mowing-bundling-shocking-stationary threshing system to mechanized combine harvesting. Water management was introduced through water conservation practices of contour farming, terracing, supplemental irrigation, and drainage systems. The acreages planted changed from small blocks to large fields. Grain storage facilities changed from small farm granaries to large, complex elevator systems. Significant advancements were made in fertilizer technology which was rapidly adopted by oat producers. Likewise, important advances were made in farm machinery; the tractor eliminated the horse and it became possible to do a better job of plowing, seed-bed preparation, planting, cultivating, and harvesting. These changes, whereby oat production was converted from a subsistence form of agriculture to a more intensive form, were accompanied by and in part were made possible by the development of new varieties that would respond to these technological advances. Oat farmers still had access to the older varieties and no one forced them to grow the new varieties. They grew the new varieties because they would yield more and better quality grain than the older varieties in the new intensive production scheme that had evolved.

Such changes would not have been possible without vertical resistance. The old horizontal-resistant varieties were not sufficiently resistant when used in a scheme of intensive production and therefore would not produce acceptable yields.

THE ENIGMA

It is generally accepted that our major food crops produced in a system of intensive agriculture may be susceptible to as yet unknown new diseases or new races of old pathogens (8, 27, 31, 66). It is also generally accepted that plant breeders will continue to utilize vertical resistance wherever possible to control diseases of crops grown in a system of intensive agriculture (16, 18). In a system of intensive agriculture, varieties change periodically regardless of the disease situation. They must change to keep abreast of the continuous improvements in crop production technology. Therefore, it is not necessary or usual for a variety to have a long life expectancy in intensive agriculture. Plant breeders, plant pathologists, the seed industry, and farmers all have demonstrated how quickly they can effect changes in a given crop (22). Also, food production must not only be maintained, but increased in the future if people are to be fed. The population explosion has occurred in part because food produced in countries with intensive agriculture production was exported to developing countries which have only a subsistence agriculture. It seems only reasonable that variety development programs directed toward areas of subsistence agriculture should explore in depth the value of horizontal resistance and attempt to capitalize upon stabilizing selection as a means of restricting the proliferation of pathogen races. Conversely, to recommend that variety development for intensive agriculture proceed in the same manner, especially when the past successes so clearly exceed the past failures, appears to court disaster.

Coyne & Schuster (11), Duvick (22), Leonard (37-39), Browning (9, 10), Nelson (44, 45), Hooker (29, 30), Borlaug (6, 7), Roane (51), McNew (43), Walter (63, 64),

Goth (25), and Crill and coworkers (12, 14, 16, 18), and others (24) have all commented recently upon specific techniques and problems in managing host resistance genes to control plant diseases. There is no central theme obvious from their individual statements, and it is difficult to formulate any kind of cohesive and workable strategy of disease control with stabilizing selection based upon their comments. In fact, Nelson (45) states that, with respect to vertical resistance, stabilizing selection is not even a valid concept.

MANAGEMENT OF STABILIZING SELECTION WITH VARIETY DEVELOPMENT

Some plant pathologists often lose sight of the fact that host resistance is worthless unless it is combined with other desirable crop plant characters. Likewise, plant breeders are often unable or unwilling to utilize plant pathological methods that have been devised for their use (13). In most cases the development of successful diseaseresistant varieties has been accomplished through the joint efforts of both the breeder and pathologist. Similarly most of the strategies of disease control with host resistance that merit discussion have been developed in cooperative programs between plant breeders and pathologists.

Some of the strategies currently in favor among plant pathologists, which supposedly rely on stabilizing selection as their basis for success, are multiline varieties, gene deployment, and gene pyramiding (6, 9, 10, 29, 30, 45). The proposed basis of success of these strategies is the introduction of vertical resistance genes into the host population in such a way that they will function as horizontal resistance. In this way stabilizing selection in the pathogen population will mimic the stabilizing selection which would occur with a naturally horizontal resistant host population. This has in fact been demonstrated (9), and limitations of the various strategies have been discussed (30). Each of these strategies has been used effectively with only a very few crops and pathogens. It is not possible to select any single strategy and generalize it to the point where it would be suitable for all or even for most crops. It probably could not even be generalized to where it would work against all pathogens of just one crop. Furthermore, use of anyone of these disease-control strategies results in a crop that is genetically vulnerable. This is true in multilinevarieties because some portion of the variety will be susceptible each year. With gene deployment, crops in a specific geographic area are susceptible and when vertical resistance genes are pyramided, the possibility also exists that the pathogen can pyramid enough virulence genes to overcome the hosts' resistance genes. The purpose of the first two strategies is to mimic horizontal resistance, but in effect what will happen is that races will be selected on the basis of the number, location, and manner in which the vertical-resistant genes are deployed and their effectiveness will be lost.

MINIMIZING GENETIC VULNERABILITY WITH VERTICAL GENE DEPLOYMENT

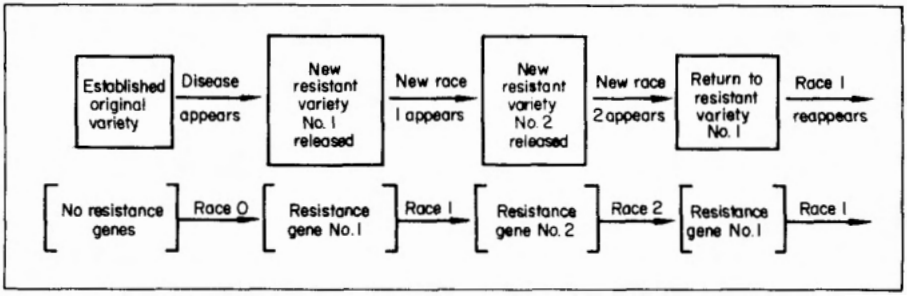
Stated in the simplest terms, horizontally resistant varieties are genetically vulnerable. This is true because any pathogen, regardless of race, can cause disease in a nondifferentiating method on horizontal-resistant varieties. For genetic vulnerabil-

ity to be realized with vertical resistance, a specific pathogen race must exist or evolve that is capable of inciting disease in the host population.

Atheoretical plan of minimizing genetic vulnerability in any crop is for the plant pathologist, plant breeder and crop production specialists to capitalize on the fact that naturally occurring stabilizing selection as well as artificially controlled directed selection is operative within pathogen populations as is diagrammed in Figures 1 and 2. Through directed selection in the host crop, the plant breeder can make rapid changes in the pathogen population by use of vertical resistance (Fig. 3). It must be recognized that races that predominate in a crop production area are the result of specific disease resistances or susceptibilities unique to the crop varieties grown in the area. When a variety is introduced into such a crop production area and it is vertically resistant to the pathogen race in question, the pathogen population will decrease rapidly in the case of obligate parasites and at a slower rate for facultative saprophytes because of directed selection. The new variety can be grown successfully in the area until a "new" race of the pathogen appears. As soon as the new race is identified, the plant breeder should be able to provide the farmer with a variety that is resistant to the new race as was done for corn resistant to race T of *Helminthosporium maydis* and for tomatoes resistant to race 2 of *Fusarium oxysporum* f. sp. *lycopersici.* This new variety should be grown until another new race appears and then the whole procedure can be repeated.

Such a plan would be effective only in areas of intensive agricultural production where plant pathologists maintain adequate and intensive disease surveys in cooperation with plant breeders. The plan probably would be disastrous if attempted in an area of subsistence agriculture. Figure 4 represents an enlargement of the basic ideas presented in Figure 3 and outlines a scheme of rotation of vertical resistance genes. Regardless of whether the new concept of stabilizing selection is valid, such a scheme should be workable in disease control because the original concepts of directed selection and stabilizing selection are valid. The initial variety grown would not necessarily have to have any particular vertical or horizontal resistance and could be grown until a pathogen race appeared which would attack it severely enough to warrant development of a resistant variety. The plant breeder then would identify and incorporate vertical resistance to the pathogen into a new variety agronomically similar to the initial adapted variety. The new vertical-resistant variety No. 1 would be introduced into the area and grown for as long as the resistance was effective. When a new pathogen race developed which could successfully attack vertical-resistant variety No. 1, the plant breeder would go through the same procedures and develop vertical-resistant variety No. 2 to replace vertical-resistant variety No. 1. The same sequence would occur which would result in the development of vertical-resistant variety No. 3. Three hypothetical vertical-resistant varieties would now have been developed, each identical with and as desirable as the initial variety except that each of the three contains a single, separate vertical-resistance gene, and each of the varieties is resistant to only one race.

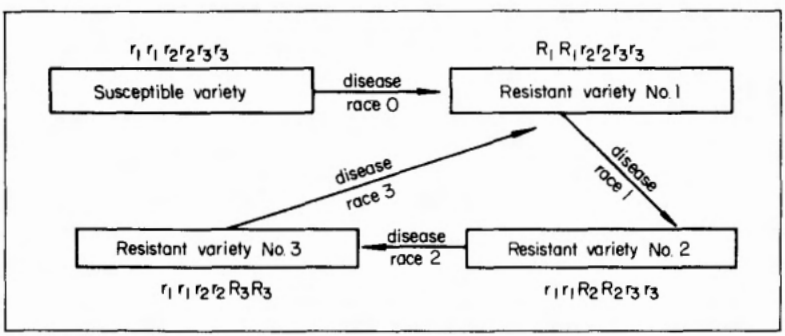
Vertical-resistant varieties such as these have been developed in several crops for resistance to several pathogens. As Van der Plank has pointed out (59, 61),



3. Proposed breeding system to minimize genetic vulnerability by manipulation of host resistance genes to create directed selection in the pathogen.

This scheme is based upon the following suppositions: 1. The original variety has no monogenic resistance. 2. Resistance gene No. 1 confers resistance only to race 0. 3. Resistance gene No. 2 confers resistance only to race 1.4. Pathogen race 0 can attack only varieties without resistance genes. 5. Pathogen race 1 can attack only original variety and resistancevariety No. 1.6. Pathogen race 2 can attack only original variety and resistant variety No. 2. 7. Resistant variety No. 1 is resistant to races 0 and 2. 8. Resistant variety No. 2 is resistant to races 0 and 1.

The scheme is compatible with the classical concepts of the gene-for-gene hypothesis and the onegene-oneenzyme hypothesis. It is also compatible with the classical evolutionary concepts of stabilizing selection and directed selection as well as Van der Plank's concept of stabilizing selection, with respect to horizontal resistance.



4. A three-gene system of variety rotations to minimize genetic vulnerability by utilizing directed selection in the host to produce directed selection in the pathogen.

these have not been effectively utilized and the resistance has been ephemeral and useless. When a pathogen evolves that can attackvertical-resistant variety No. 3, the farmer should be able to return to -vertical-resistant variety No. 1 and the process recycled indefinitely if the plant breeder and plant pathologists have conducted their variety development program properly. Person (48) has discussed the fate of host genes for resistance and pathogen genes for pathogenicity in "natural occurring disease systems." He did not consider that plant breeders and pathologists could exert directed selection on the pathogen population each crop season through manipulation of host resistance genes. Because of this directed selection in the host by the plant breeder using vertical resistance, directed selection occurs in the pathogen population. By the time race 3 has

evolved, directed selection produced by the plant breeder coupled with naturally occurring stabilizing selection in the pathogen population against races with excess genes for pathogenicity should have eliminated race 1 from the natural population; therefore, vertical-resistant variety No. 1 would be resistant to the resulting pathogen population whose evolution was controlled by the plant pathologists and plant breeders. With this system for use of varieties, the farmer could continue to rotate plantings of vertical-resistant varieties indefinitely, based upon the genotype of the pathogen population with reduced likelihood of an epidemic. It is obvious that this proposed strategy has greatest potential with diseases caused by obligate parasites or pathogens that are unable to survive for long periods of time in the environment without a susceptible host. For such diseases, a modification of the gene rotation strategy maybe required. A possible system would be to pyramid two genes only in a variety and rotate them in the following manner. For example, resistant variety 1 would contain gene R 1 ;variety 2, R genes 1 and 2; variety 3, R genes 2 and 3; variety 4, R genes 3 and 4, and so on.

There are obvious difficulties with such a gene rotation system which any serious student of plant pathology or plant breeding can visualize. With some pathogens, the time required for evolution of new races might be so short that such a system would not be feasible. Also, the screening program might be so slow that it would not be feasible to incorporate resistance after it was located. With some crops, the life span is obviously too lengthy to fit into such a breeding system. Despite these obvious objections, and perhaps several others, such a system would offer a plant breeder and plant pathologist an opportunity to control some diseases in some crops very effectively. This system does provide answers and remedies to some of the criticisms which have been brought against other breeding systems using vertical resistance. Some of the advantages are (a) genetic vulnerability is limited or reduced, (b) development or occurrence of pathogen races is controlled by plant breeders and pathologists through directed and stabilizing selection, (c) vertical resistance is deployed and effectively managed, and (d) strong vertical-resistance genes are conserved and can be used repeatedly over a long time. Some of my colleagues have suggested to me that legal mandates or laws might be required to implement such a strategy. I strongly oppose all such suggestions from both scientific and political viewpoints and rest my case with the industry's response to the corn blight epidemic. As discussed earlier, new resistant varieties were available the first year after the occurrence of the epidemic and adequate seed was available the second year to plant the entire US corn acreage. It is quite unlikely that a federal bureaucracy could have managed the response to the corn blight epidemic nearly as well as did the corn industry itself.

SUMMARY

As Harlan (28) has so aptly stated, "A fully domesticated plant cannot survive without the aid of man, but only a minute fraction of the human population could survive without cultivated plants." This single statement sums up the basis of

concern that scientists, politicians, sociologists, and food producers all express concerning the world's future food supply. They apparently do not understand, however, that our present-day crop varieties could not survive in a wild or natural state or that varieties of yesteryear cannot be utilized without significant decreases in yield and quality in present-day intensive crop production. Until the majority of concerned people do comprehend and appreciate Harlan's statement, controversies such as that on stabilizing selection will continue.

There is little doubt that stabilizing selection in favor of pathogen races without unnecessary virulence does occur with use of horizontal resistance. All evidence, including the original theory of stabilizing selection, indicates this to be true. Such is not the case for stabilizing selection with vertical resistance. Nelson's arguments against stabilizing selection with vertical resistance (44, 45) are just as logical and valid as Van der Plank's were when he proposed the concept (59). Van der Plank's (59) criticism of plant breeders' usage of vertical-resistance genes appears to be a valid one. It remains to be seen whether this misuse was due to stabilizing selection, however.

A new system has been proposed for deployment of vertical-resistance genes in plant breeding programs. This system has the potential for conservation of vertical-resistance genes in a number of host-pathogen interactions and will function regardless of the new concept of stabilizing selection. The proposed gene deployment system should allow for much better management of vertical-resistance genes. It appears that vertical resistance is generally more desirable in crops grown in intensive agriculture. Horizontal resistance is preferred in subsistence agriculture because stabilizing selection is operative.

There are numerous formats which could have been used to present this concept. No doubt, I have not included some aspects which certain people think are significant and have included some which certain people may consider superfluous. I did not present any of the specific charges which have been made against the new concept of stabilizing selection because those who are doing research in this area of plant pathology are quite familiar with the charges. Instead, 1 chose to treat stabilizing selection in a very general manner which I hope will be of more value to the general reader.

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Effective and stable control of rice blast with monogenic resistance[1]

J. P. Crill and G. S. Khush[2]

FOREWORD

Numerous practices for reducing rice blast disease development have been developed; however, epidemic of the disease still occurs in most of the rice producing countries. Plant breeders are of the opinion that the development of resistant rice varieties can be the only feasible recourse for control of the disease.

The author discusses the two types of resistance - horizontal and monogenic - and compares the merits of the two. Horizontal resistance serves only to slow down a blast epidemic after it has started whereas monogenic resistance serves to prevent the Occurrence of an epidemic. Therefore, incorporation of horizontal resistance in crop varieties is not worthwhile as a primary means of disease control. As implied by the concept 'for every gene for resistance in the host, there is a corresponding gene for pathogenicity in the parasite', it appears that the first line of genetic defense or resistance in a host are major genes or monogenes for resistance. The author points out that monogenes are usually dominant, and when present in the host, result in no disease or no symptom development.

Such a complex topic as presented in this bulletin is discussed by the authors in very simple language that any layman may understand. This paper was presented by the senior author in the lecture meeting on 'Rice blast disease' held in Suweon, Korea, on July 23 to 29, 1979.

INTRODUCTION

It has been generally assumed by most rice researchers that the most effective and economical way to control the blast disease of rice is by development and use

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of resistant varieties (8). Rice blast, caused by the fungus *Pyricularia oryzae,* can be chemically controlled with various fungicides; however, this practice is expensive and often uneconomical in some parts of the world. Other problems with chemical control besides those of an economic nature include uncertain availability of chemicals, methods and machinery for application are not understood or not available, environmental pollution, etc. It has also been long recognized that cultural practices influence the occurrence and severity of the blast disease. Numerous practices designed to reduce the inoculum potential of the blast disease have been developed. These include practices of exclusion, eradication, sanitation, etc. In some instances certain of these practices may be effective in reducing disease development, however none of them will prevent the occurrence of an epidemic. The only feasible recourse for control of the blast disease has been development of rice varieties which are resistant to the disease.

Resistance is a term of ancient origin, considerably older than the science of plant pathology itself. The generally accepted usage of the word resistance implies that a specific plant, variety or crop has less disease than another. Such a definition is acceptable to a layman or novice plant pathologist or plant breeder, but wholly unacceptable to those who are intimately acquainted with plant diseases, their occurrence, and the genetic and environmental interactions among various hosts and pathogens. Within the rice - rice blast system, host refers to a rice plant which is infected or has been exposed to the pathogen, a fungus, taxonomically characterized with the scientific name *P. oryzae,* within environmental conditions suitable for infection and disease development to occur. The development and expression of the blast disease of rice is much more complex than the above simple description implies.

A susceptible rice plant or variety is one which develops symptoms when exposed to a pathogenic race of the blast fungus within an environment suitable for disease development. A resistant rice plant or variety is one which does not develop symptoms under the same environment as the susceptible one. Resistance is then absolute as measured by symptom development. Plants with symp toms are susceptible and those without symptoms are resistant. Susceptibility is not absolute. Rather, susceptibility varies from slightly susceptible to very highly susceptible. In recent times it has become popular to refer to disease reactions which are characterized by slight to moderate susceptibility as a type of resistance such as horizontal resistance, field resistance, non-specific resistance, etc. In practical terms, slight to moderate susceptibility is much preferred to high susceptibility, and by terming such reactions as resistant a great deal of confusion is injected into the picture, especially from the standpoints of plant breeding and development of blast resistant varieties.

The expression of resistance or susceptibility in the rice plant with regard to the blast fungus is genetically controlled. With respect to specific isolates or races of the blast fungus, resistance in the rice plant (as measured by absence of symp toms) is controlled by a single dominant gene while susceptibility (as measured by presence of symptoms) is governed by the recessive condition of the same gene. The genetic control of the different degrees of susceptibility which are referred to as horizontal resistance or non-specific resistance is much more complex and

considered to be polygenic in nature. Likewise, pathogenicity or the ability of an isolate or race of the fungus to incite disease on a specific host plant or variety is genetically controlled. The expression of pathogenicity of the blast fungus is apparently controlled by a single gene. In all probability pathogenicity is expressed in a recessive condition and nonpathogenicity in a dominant condition. The disease severity, or severity of symptoms, produced by a given isolate or race of the fungus on a specific host is termed virulence. Virulence is apparently controlled by polygenes in *P. oryzae* and little is known concerning the inheritance of virulence.

It therefore becomes impossible to study the genetics of resistance to blast in rice without studying the genetics of pathogenicity. Such studies form the basis of the gene-for-gene concept, which with rice blast can be stated as follows: 'For every gene for resistance to blast in the rice plant, there exists in the blast fungus a complementary gene for pathogenicity to the rice plant.' This concept can be extended to a genes-for-genes situation as well which can be stated as follows: 'For those genes governing horizontal resistance to blast in the rice plant, there exists in the blast fungus complementary genes for virulence to the rice plant.' From these two statements it is obvious that at least two distinct host-pathogen systems are operative within the rice-rice blast host-pathogen interaction. The host resistance base of these two systems may be referred to as monogenic resistance (vertical resistance, specific resistance, etc.) and polygenic tolerance (horizontal resistance, field resistance, non-specific resistance, etc.). The effectiveness and stability of either type of resistance are determined by the dynamics of pathogen variation. Thus, the problem of controlling the blast disease of rice with resistant or tolerant varieties is dependent upon a thorough and complete understanding of the mechanisms of variability which are present in P. oryzae.

DYNAMICS OF PATHOGEN VARIATION

When a host plant is exposed to a pathogen and environmental conditions are suitable for infection and disease development to occur, there are two possible results; either, a) symptoms develop or b) symptoms do not develop. If symptoms develop, the extent of development is influenced by three factors: the genotype of the host, genotype of the pathogen and environment. When a series of host plants or varieties are exposed to the same genotype of the pathogen within the same environment, susceptible varieties would exhibit: 1) more lesions; 2) larger lesions; 3) earlier lesion development; 4) less vigorous growth, or 5) more severe symptoms in general. On the other hand, some varieties would exhibit: 1) fewer lesions; 2) smaller lesions; 3) late lesion development; 4) more vigorous or robust growth. or 5) less severe disease development and these varieties are said to exhibit tolerance. general resistance, partial resistance, horizontal resistance, non-specific resistance, slow disease development, etc.

It is generally assumed, but rarely documented, that such reductions in disease severity are quantitative in nature, and therefore polygenically controlled. As such, it is further assumed, but rarely documented, that such host responses, since they are polygenically. and not monogenically controlled, are effective against all races

of the pathogen. Because of this effectiveness, non-specific resistance or horizontal resistance has been portrayed as uniform, long lasting and stable.

HORIZONTAL RESISTANCE

Such resistance was first described specifically by Helen Hart (6) for stem rust in wheat although many general reports for various diseases were made earlier. Since then, tolerance or general, non-specific, or horizontal resistance has been the subject of numerous research investigations, reports and review papers. In most instances, such resistance is expressed on a plant population rather than single plant basis using an arbitrary scale or disease index.

Loegering (7) has expressed such general resistance on a single plant basis and developed disease indexes for the reaction of single plants to a population of plant pathogens. The objective of the 'Loegering Method' is to develop crop plant populations from single plant selections which exhibit general resistance. Using Loegering's technique enables plant breeders to rapidly establish plant populations or varieties from single plant selections which exhibit 'good or 'high-levels' of general resistance.

Assumptions of Horizontal Resistance

The underlying assumptions which a plant breeder and plant pathologist make when they select for general, nonspecific or horizontal resistance to a pathogen in a plant breeding program are: 1) environmental conditions are uniform in the nursery where selections are made; 2) the plants or lines in the nursery are uniformly exposed to all the various genotypes of the pathogen which exist in the pathogen population and; 3) the genetics of the pathogen, with respect to pathogenicity and virulence will remain static and unchanging from season to season and year to year. The first two assumptions are probably, but not necessarily, valid in most nurseries. The third assumption has been portrayed as the basis of stability of horizontal resistance which is attributed to stabilizing selection because of the polygenic nature of inheritance in the pathogen (1 0). The key to the success of tolerance, horizontal or general resistance which the plant breeder incorporates into the new variety is, therefore, based upon assumption number 3, i.e. genetics of pathogenicity and virulence are static when specific or vertical resistance is removed from the host population and only non-specific, general or horizontal resistance remains. It is unfortunate that specific experimental evidence is so inadequate to evaluate this assumption. However, numerous evolutionary studies of various organisms indicate the assumption to be false.

Analysis of Pathogen Variation and Horizontal Resistance

Van der Plank stated: 'Increased horizontal resistance in the host can be matched by increased aggressiveness in the pathogen,' (10) which is just an extension of the gene-for-gene concept applied to polygenes. Crill et al. (2) demonstrated in the tomato-Fusarium wilt host-pathogen interaction that virulence of the pathogen, as measured by disease severity and controlled by polygenes, was influenced and regulated by the polygenes controlling tolerance or non-specific resistance in the

host. The evolution of virulence in the tomato wilt pathogen is apparently directly controlled by the level of tolerance, general or horizontal resistance present in the host tomato varieties. As more and more polygenes for horizontal or non-specific resistance were incorporated into the varieties, the pathogen responded by accumulating more and more polygenes for virulence. Evidence for the occurrence of this phenomenon was obtained by inoculating the tomato variety Missouri Accession No. 160 (Mo. 160), with various isolates of the pathogen. When it was first reported, the resistance in Missouri accession 160 was so strong against all the known isolates of the fungus that disease development did not occur or was very rare. In fact, the resistance was so strong that the investigators who first studied the tomato-Fusarium host-pathogen interaction considered the resistance to be immunity and designated a gene called I (for immunity) which controlled the disease reaction.

This source of resistance was widely used by tomato breeders and in a short period of time, it was incorporated into all varieties grown in the U.S. Plant breeders, plant pathologists and seed producers rapidly recognized that the only way such resistance could be maintained in a variety was through a continuous screening and selection program. It was quite common for a small percentage of plants in a field to become infected with the pathogen and the only way a suitable level of resistance could be maintained was by screening plant populations using isolates of the pathogen obtained from diseased plants. From such screening trials, seed was produced only from those plants which did not show disease symptoms. In this way, continuous selection pressure was maintained for the polygenes controlling resistance in the host. When these host plant varieties were grown by farmers, the polygenes for resistance resulted in selection pressure being applied to the pathogen population to acquire a higher level of virulence. The pathogen population responded, presumably by the incorporation of more and more genes for virulence into more and more individual propagules within the pathogen population and these were the forms which persisted and increased in frequency in the pathogen population. As a result, the pathogen population became more virulent each cropping season and more and more polygenes for resistance had to be incorporated into the newer varieties to maintain the original level of general resistance in Mo. 160. It is now generally believed that the resistance which was originally identified as being controlled by the dominant gene I, was in fact not monogenic resistance but rather polygenically controlled general or horizontal resistance and the experimental evidence supports such a conclusion.

Seed of Missouri Accession No. 160 tomato had been carefully maintained with a minimum number of seed increases and there is no reason to doubt that the present stock is in any way different from the original. In fact, the purpose of maintaining the stock seed was to keep Missouri Accession 160 in its original genetic form. Unfortunately, there are no isolates of the tomato wilt pathogen which have been maintained from the same era in the same way. Therefore, the only possible comparison to determine if changes in pathogen virulence had occurred was to evaluate the disease resistance of Missouri Accession 160 and the presently grown varieties against prevalent, present isolates of the pathogen.

This was done by inoculating Missouri Accession 160, Bonny Best, a variety known to possess no recognizable resistance, and various varieties which had been developed using Missouri Accession 160 as a donor source of resistance with specific amounts of inoculum under controlled conditions, with field isolates of the wilt pathogen and observing disease development. The susceptible Bonny Best was immediately killed, Missouri Accession 160 was severely diseased and the other varieties exhibited disease in various degrees. In general, the more recent the date of development and release, the more field resistant or tolerant the variety was to the field isolates of the pathogen. These results clearly indicate that the virulence of the pathogen in the field had been changed by growing the more tolerant varieties. Also, when the screening data from various tomato breeding and screening programs were compared, virulence in the pathogen was seen to increase as varieties were developed with higher levels of tolerance, general or horizontal resistance. In the early studies where Mo. 160 showed a very high level of resistance, Bonny Best developed disease symptoms at a much slower rate and with less severity than it does now, even when identical inoculation techniques are used. This is a further indication that present isolates of the Fusarium wilt pathogen have more genes for virulence (not more genes for pathogenicity) than was present when Mo. 160 was first identified as being resistant.

There is still some controversy as to whether the resistance attributed to the I gene in tomato was actually due to a single monogene. There is, however, little question that the resistance in the original Missouri Accession 160 was general or horizontal resistance rather than specific or vertical resistance. When disease reactions of Fusarium wilt occurring on Bonny Best are compared with those on Missouri Accession 160, the reaction on Mo. 160 fits the classical pattern described for horizontal or general resistance. In fact, the resistance in Mo. 160, when first reported, was so strong that it was assumed to be 'immunity'. Subsequent studies showed that it was not immunity but rather a very good, or strong source of general or horizontal resistance which gradually became ineffective because of virulence changes in the pathogen.

Stabilizing Selection and Horizontal Resistance

The same phenomenon of increasing virulence in the pathogen when varieties with increased general or horizontal resistance are introduced is well documented in the watermelon - Fusarium host-pathogen interaction and various others. The stability that is claimed for horizontal or general resistance does not exist.

General, non-specific, or horizontal resistance is not any longer lasting or any more stable than is monogenic, specific or vertical resistance. The stability of resistance, which directly controls how long lasting resistance will be, is not a function of the resistance genes in the host, but rather a function of the genes in the pathogen which control pathogenicity and virulence. The evolution of the pathogen is controlled by two forces, directed selection and stabilizing selection. It is commonly, but erroneously thought that general or horizontal resistance in the host is stable and unchanging because stabilizing selection is presumed to operate in the pathogen population and functions to maintain a uniform, unchanging level of pathogenicity and virulence.

Every plant pathogen in existence has evolved to its present level, because of its ability to compete and survive. The high-type parasites, such as rusts and mildews, have evolved to the point they are obligate parasites. If the host becomes extinct, such pathogens also become extinct. Within such pathogens, a protective mechanism has also evolved which prevents the pathogen from causing disease to the extent that the host population is eliminated. Such pathogens are usually well adapted to be transported and spread over large areas and distances. Similarly, the low-type pathogens such as *Rhizoctonia* have also evolved in such a way that they are adapted to survive. Such lowtype pathogens are relatively non-discriminatory and have a wide host range and attack and kill their hosts. When the host is killed or eliminated as a food source, the pathogen lives saprophytically in the soil or exists as sclerotia in a resting stage waiting for another host to appear as a food source. Such pathogens are not adapted to rapid or widespread dispersal. All plant pathogenic populations exist within such a large evolutionary framework and balance. It is a corollary of evolution that if such balance is upset or changed too far in either direction, the pathogen will become extinct and cease to exist. Every plant pathogenic population must exist within such a balance and it is because of this that each pathogen may be characterized by its severity or effect on the reproductive capability of the host, i.e. high-type or lowtype parasites.

The evolution of plant pathogens, within the already established evolutionary confines, is controlled by directed selection and stabilizing selection. At any given time, both of these evolutionary forces are in operation for all populations of organisms and the evolution of the population is controlled by both forces, never by one alone. At some times or stages in the evolutionary development of a population, the effects of directed selection on a single characteristic may be much greater than those of stabilizing selection or conversely, but never does one totally replace the other for all characteristics of the population. It is because of these evolutionary laws that stabilizing selection cannot function to maintain a population of plant pathogens in a static state with respect to virulence when the host population is changed.

When plant varieties with high levels of general or horizontal resistance are introduced into the cropping system, the effect on the pathogen population is immediate and direct. Selection pressure is immediately applied to the pathogen population and the population responds with the direct selection of those individuals which have adequate virulence genes to overcome the general or horizontal resistance. These individuals are the ones which survive and reproduce to form the future generations of the pathogen and the frequency of virulence genes in the pathogen population increases correspondingly. In this instance, direct selection pressure for virulence has a considerably greater effect than does stabilizing selection, although stabilizing selection will be operative for all other characters of the pathogen population to insure that those individuals which survive are the ones best fitted for the environment.

All arguments supportive of the stability of horizontal resistance have two features in common: they are based on very short term results and specific races identified by their reaction on hosts with specific R genes are used as sources of inoculum to inoculate hosts with and without specific R genes. The disease

reaction of the host with R genes is compared with that of the host without R genes. In many instances, the host with no known R genes will exhibit less disease or reduced disease development when compared with the host with specific R genes. This is interpreted to mean that the host without specific R genes has general or horizontal resistance and it is incorrectly assumed that this resistance will be stable and long lasting because it is polygenically controlled. In those few cases where the breeding programs have emphasized such general or horizontal resistance, it has not been stable or long lasting.

Consider the late blight disease of potato. After the destructive famines of the 1840's in Ireland and elsewhere in Europe, the variety Magnum Bonum was introduced which had a high level of general or horizontal resistance to the late blight pathogen, *Phytophthora infestans.* This was nonspecific, general or horizontal resistance which was quite effective in controlling the disease initially. As the variety, and others of this type were grown on a wider scale, the resistance gradually became less and less effective. Such a loss of resistance was due to the evolution of more virulent genotypes of the pathogen which were able to overcome the general resistance. Evidence to support such a statement has been provided by Reddick and Mills (9) who observed that the pathogen became increasingly virulent on such horizontally resistant varieties throughout the grow ing season. They were able to demonstrate when wild type isolates were cultured on horizontally resistant varieties for several generations that virulence (not pathogenicity) was increased in each generation up to the point where the horizontally resistant variety was no longer resistant. These results explain very clearly that what happened to Magnum Bonum and other horizontally resistant varieties used in Europe, and especially Germany, was a change in the virulence of the pathogen. As varieties were developed which had higher and higher levels of general or horizontal resistance, the pathogen population responded by accumulating more and more genes for virulence.

A very high level of general or horizontal resistance was known to exist in the cultivated potato, *Solanum tuberosum* and no monogenes for resistance were ever reported to be present. All of the original varieties were susceptible, but varieties differed greatly in susceptibility and exhibited marked degrees of nonspecific, general or horizontal resistance to the late blight fungus. Potato breeders in Europe and America combined the various sources of non-specific or horizontal resistance which were for the most part considered to be polygenically controlled and additive and numerous varieties were released which had very high levels of horizontal resistance. Walker stated that numerous varieties had been developed by hybridization and selection which showed marked resistance (horizontal resistance) to late blight, but in the main, they needed fungicidal protection under severe epidemic conditions (11).

EFFECTIVENESS OF HORIZONTAL RESISTANCE

The Fusarium wilt of tomato, Fusarium wilt of watermelon, late blight of potato and numerous other host pathogen interactions provide classical examples of horizontal resistance, yet this resistance became ineffective and was not stable or

long lasting when introduced into crop varieties that were grown over large areas for a period of time. Horizontal resistance is described as polygenic in nature and effective against all races or isolates of the pathogen. Likewise, it is described as stable and long lasting. Horizontal resistance is measured or determined by the lack of a differential interaction. If the resistance in a host is evenly spread against all races of a pathogen, it is called horizontal. The degree or amount of horizontal resistance is measured by disease development or symptom production. For example, when two potato varieties, Kennebec and Maritta, are grown side by side, Kennebec succumbs faster to blight than Maritta. Maritta is said to have more horizontal resistance than Kennebec. The stability of such horizontal resistance and the reason for its long-lasting nature is attributed to the phenomenon of stabilizing selection (1 0). The horizontal resistance of the host is assumed to be stable, because of stabilizing selection which occurs in the pathogen. Such stabilizing selection is presumed to operate in the pathogen population because of the polygenic inheritance of virulence. To understand the nature of stabilizing selection, it is necessary to study the population interactions which occur when a host with strong horizontal resistance is introduced into a cropping system.

EFFECT OF HORIZONTAL RESISTANCE ON PATHOGEN VIRULENCE

When a new crop variety with a higher level of general or horizontal resistance than previously existed is introduced into a cropping system, the effect on disease incidence is readily obvious and seen to decrease. This is true regardless of whether the pathogen is a 1) high-type or low-type parasite; 2) soil-borne or air-borne; or 3) foliage or vascular pathogen. Each of these factors, in conjunction with environmental factors which affect disease development, will affect the length of time that disease incidence is decreased. As previously indicated, the disease severity, or effect of the pathogen on the host is controlled by stabilizing and directed selection. When disease development is mild or slight, directed selection operates to select out those individuals of the pathogen population which are capable of causing disease and reproducing to produce the future generation. As noted previously, pathogens have at their disposal numerous and varied means of reproduction and genetic recombination. Those individuals within the population that are best adapted to the environment, in this instance, those capable of causing infection on the horizontally resistant new variety and reproducing either asexually or sexually, are the individuals which survive. Those which could not attackthe new host variety and reproduceare lost, or at least diminish in frequency of occurrence to a very low level. Those that are weakly virulent will survive and reproduce, but their frequency of occurrence within the population will also be low. The greatest change in the frequency of various virulence types in the pathogen population will be toward increased virulence. As farmers continue to plant the horizontally resistant variety on larger and larger areas, direct selection pressure is applied to the pathogen population for more and increased virulence within the pathogen population. The selection and establishment of such virulent pathogens is said to be due to directed selection.

Eventually, forms of the pathogen will occur which are 'supervirulent'. Such

forms can be selected and maintained in an artificial system by plant pathologists and plant breeders, but they do not become established in nature because of stabilizing selection. Such highly virulent forms are discriminated against in nature because they have no mechanism of survival, and this is what stabilizing selection is all about. The highly virulent form, when it evolves through mutation, genetic recombination or any other process does not become established as a part of the naturally occurring pathogen population because it is unable to reproduce and maintain itself. When a highly virulent pathogen attacks a host, the host may die or its growth may be considerably reduced and the reproductive capability of the pathogen is greatly restricted simply because it has destroyed its food source. This is what is meant by the evolutionary balance of host-pathogen interactions and the statement that every plant pathogen in existence has evolved, or is evolving, to a level of adaptation based upon its ability to compete and survive in nature. Furthermore, this level of adaptation is maintained by stabilizing selection.

Consider also that when a highly virulent pathogen occurs in nature, it must compete with all of the other individuals within the pathogen population for survival. The number of infective propagules produced by a pathogen population which are capable of inciting disease are nearly infinite when compared to those which actually do cause disease. For any given situation, there are only a limited number of host plants to be infected and only a limited number of infection sites available on each host. Furthermore, the rate of survival of the progeny produced by a pathogen population is extremely low. For infection to occur, the infective propagule must be in an environment which has the proper host and appropriate climatic conditions. The pathogen progenies which survive are the ones most adapted to the environment. Those progeny, which in turn produce tremendous numbers of progeny by coexisting with the host in a way that maximizes progeny production with the least damage to the growth and survival of the host will appear in the highest frequency in the population. A highly virulent or super virulent form of the pathogen will be immediately lost in nature because it kills or reduces the capability of the host to support it. The super virulent form cannot reproduce adequate progeny to become established in natural conditions and is said to have been curbed by stabilizing selection.

The proponents of non-specific, general and horizontal resistance to plant pathogens will argue that the desirable feature of such resistance is stability and that such stability is due to stabilizing selection. Such an argument is valid and cannot be questioned. What is questionable is the effectiveness of such resistance and how long the original level of horizontal resistance will remain economically effective before direction selection in the pathogen for increased virulence is able to overcome the resistance as in the case of Fusarium wilt of tomato and watermelon, late blight of potato and various other host-pathogen systems. The real question at issue is: are plant breeders and plant pathologists wasting their time by looking for horizontal resistance and incorporating such resistance in crop varieties. Past experience with several diseases on several crops indicates they are. Furthermore, the application and study of the concepts of stabilizing and directed selection in host-pathogen systems also indicate that incorporation of

such resistance is not worthwhile as a primary means of disease control. The assumption that virulence of the pathogen population remains static and unchanging when varieties with increasing levels of horizontal resistance are introduced into the cropping systems does not appear to be valid.

MONOGENIC RESISTANCE

The gene-for-gene concept as advanced by Flor (5) can be briefly stated as follows: For every gene for resistance in the host, there is a corresponding gene for pathogenicity in the parasite. The first line of genetic defense or resistance in a host then is major genes or monogenes for resistance. These genes are usually dominant, and when present in the host, result in no disease or no symptom development. When such genes are absent, the pathogen infects the plant. symptoms are produced and disease development occurs. Since the first report by Biffen in 1905, (1) the literature abounds with numerous studies demonstrating that resistance to disease, more specifically, resistance to a particular pathogen is controlled by dominant monogenes. Likewise, many but not nearly as numerous, reports are available indicating that the ability of the pathogen to incite or cause disease in the host is controlled by monogenes as well, although these are usually recessive. Such studies have been conducted with all classes of plant pathogens including viruses, bacteria, fungi, nematodes and parasitic phanerogams with the results generally confirming that such gene-for-gene relationships exist. In many, the evidence is indirect; however, the specificity of genetic relationships are clearly indicated and indirectly supports the gene-for-gene concept.

Stated in the simplest terms, horizontally resistant or field resistant varieties are susceptible. This is true because any race of *Pyricularia oryzae* can cause blast on any rice variety which has no monogenes for resistance. Horizontal resistance is evenly spread and acts against all races of the pathogen which supposedly serves to slow down an epidemic after it is in progress. For susceptibility to be realized with monogenic resistance, a specific race of the blast fungus must exist or evolve which is capable of overcoming the specific monogene conferring resistance before an epidemic can occur. Horizontal resistance serves only to slow down a blast epidemic after it has started whereas monogenic resistance serves to prevent the occurrence of an epidemic.

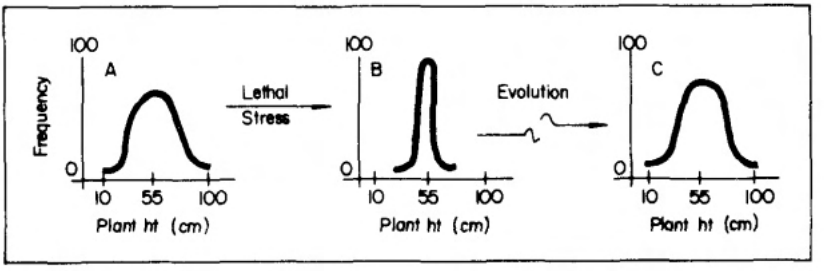
It is a well documented fact in Korea that horizontal resistance in rice to blast was ineffective in controlling the disease during the 1978 epidemic. Such horizontal resistance may have been effective in slowing down the epidemic but it was not effective in controlling the epidemic. With such conditions as existed in the 1978 Korean epidemic it makes little difference whether the epidemic was slowed down or not when the end result was total loss due to neck blast

Numerous varieties and lines of rice had been grown in Korea from 1975-77 which had high levels of horizontal resistance as measured by seedling blast ratings of 3,4 and 5 and only slight neck blast at maturity. The response of these varieties and lines was essentially the same in the blast nurseries, breeders plots, test plots and farmers fields under both inoculated and naturally-infected conditions. In 1978, under epidemic conditions, these same varieties and lines which

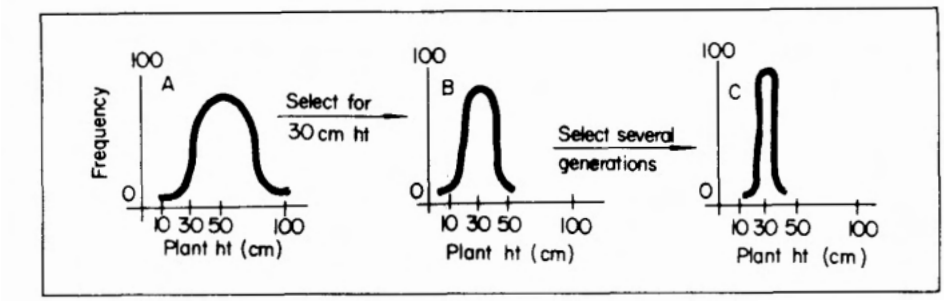
had given scores of 3,4 and 5 to blast were seen to be highly susceptible with scores of 7, 8 and 9 and high levels of neck blast. The inherent horizontal resistance was not effective in preventing very significant losses even though it may have been effective in slowing down the epidemic. The argument that if the entire country of Korea had been planted with horizontally resistant varieties the epidemic would not have been so severe, is not entirely without merit but can not be experimentally supported. In fact, much of experimental evidence available tends to be non-supportive of such an argument.

Obviously it is much preferred to prevent an epidemic from occurring with monogenic resistance than it is to slow down an epidemic with horizontal resistance after the epidemic has started. In those areas of the world where blast is a major limiting factor in stable rice production, the major problem which rice scientists must contend with is how can blast be controlled consistently and economically. If monogenes for resistance are effective in preventing the occurrence of a blast epidemic for one or two years until a new pathogenic race of *P. oryzae* arises which can overcome such resistance, rice scientists should be conceptually clever enough to devise techniques aimed at thwarting the pathogen so that farmers can be indefinitely assured of having varieties which will not succumb to blast.

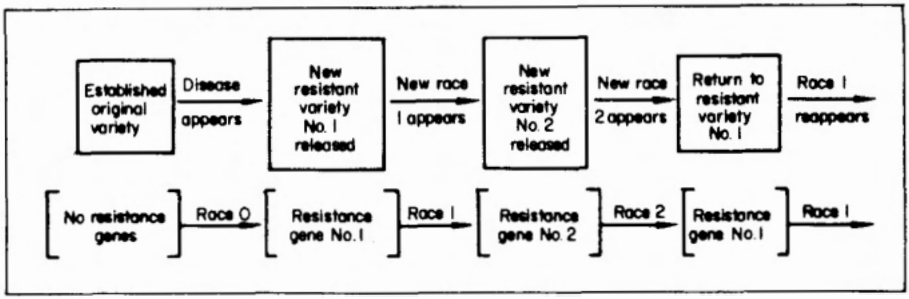
A theoretical plan of minimizing susceptibility to blast in rice is for the plant pathologist, plant breeder, and crop production specialists to capitalize on the fact that naturally occurring stabilizing selection as well as artificially controlled directed selection is operative within pathogen populations as is diagrammed in Figures 1 and 2. Through directed selection in the host crop, the rice breeder can make rapid changes in the pathogen population by use of monogenic resistance (Fig. 3). It must be recognized that races that predominate in a crop production area are the result of specific disease resistances or susceptibilities unique to the crop varieties grown in the area. When a variety is introduced into such a crop production area and it is monogenically resistant to the pathogen race in question, the pathogen population will decrease rapidly because of directed selection. The new variety can be grown successfully in the area until a 'new' race of *P. oryzae* appears. As soon as the new race is identified, the rice breeders should be able to



1. Hypothetical occurrence of stabilizing selection in a naturally occurring plant population subjected toenvironmental stress. A. Frequency distribution curve illustrates plant height of the individuals comprising the population. B. individuals surviving the stress exhibit the fewest deviations from the mean plant height of 55 cm. C. Through evolution the population eventually returns to the original distribution with a mean plant height of 55 cm.



2. Hypothetical occurrence of directed selection in a naturally occurring population when selection pressure favors plants 30 cm high. A Frequency distribution curve Illustrates plant height of the individuals comprising the original population B. The frequency of 30 cm tall individuals is Increased with positive selection pressure. C. With continued selection pressure for plant height of 30 centimeters almost all individuals in the population are 30 centimeters tall.



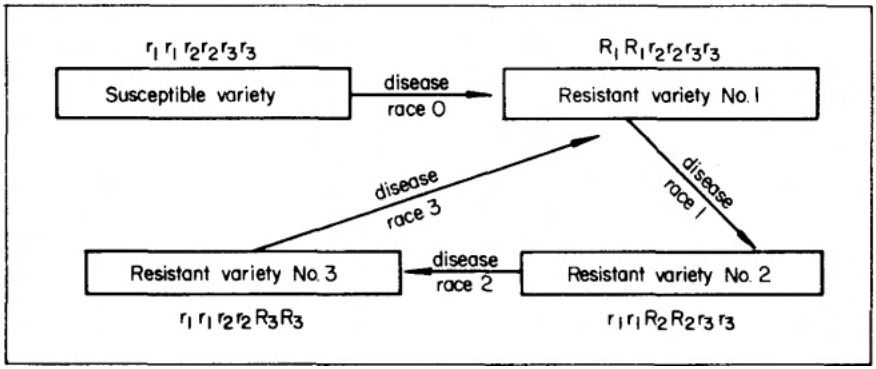
3. Proposed breeding system to minimize genetic vulnerability by manipulation of host resistance genes to create directed selection in the pathogen

This scheme is based upon the following suppositions: 1. The original variety has no monogenic resistance. 2. Resistance gene No 1 confers resistance only to race O. 3. Resistance gene No. 2 confers resistance only to race 1 4 Pathogen race 0 can attack only varieties without resistance genes. 5. Pathogen race 1 can attack only original variety and resistance variety No. 1. 6. Pathogen race 2 can attack only original variety and resistant variety No 2. 7 Resistant variety No. 1 is resistant to races 0 and 2 8 Resistant variety No 2 is resistant to races 0 and 1.

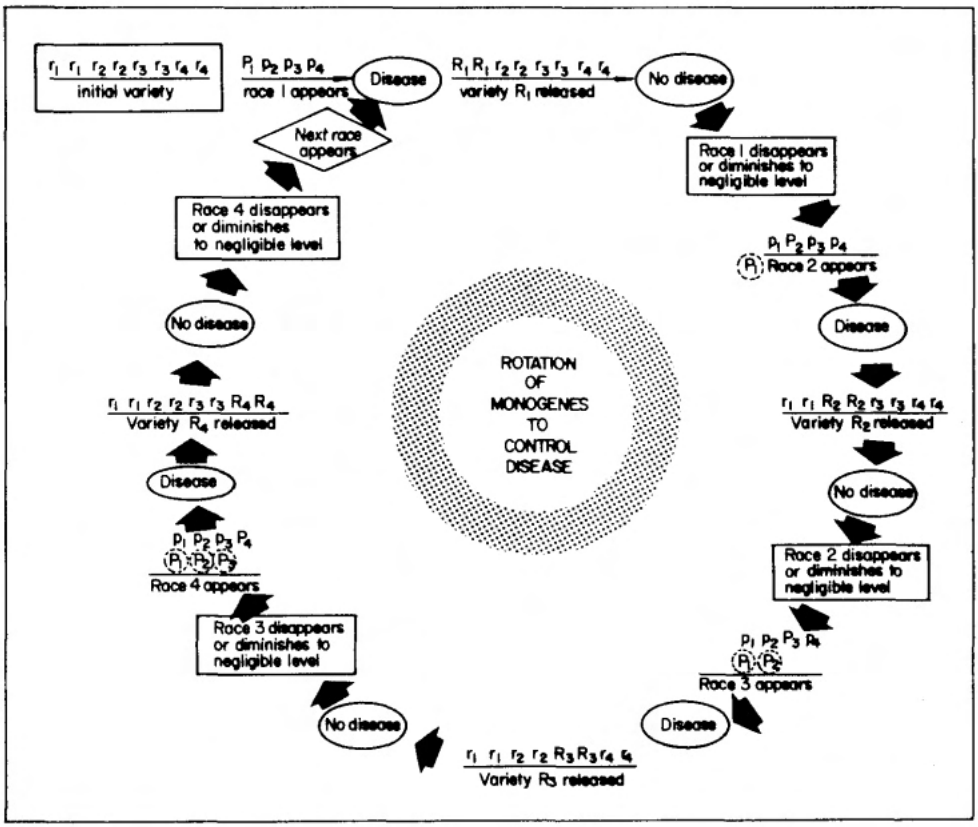
The scheme is compatible with theclassical concepts of the gene-for-gene hypothesis and the one gene-one enzyme hypothesis. It is also compatible with the classical evolutionary concepts of stabilizing selection and directed selection as well as Van der Plank's concept of stabilizing selection, with respect to horizontal resistance.

provide the farmer with a variety that is resistant to the new race as was done for corn resistant to race T of *Helminthosporium maydis* and for tomatoes resistant to race 2 of *Fusarium oxysporum* f. sp. *lycopersici.* This new variety should be grown until another new race appears and then the whole procedure can be repeated.

Such a plan would be effective only in areas of intensive rice production such as Korea where plant pathologists maintain adequate and intensive disease surveys in cooperation with plant breeders coupled with a strong and respected extension service. The plan probably would be disastrous if attempted in an area of subsistence agriculture. Figure 4 represents an enlargement of the basic ideas presented in Figure 5 and outlines a scheme of rotation of monogenes controlling



4. A three-gene system of variety rotations to minimize genetic vulnerability by utilizing directed selection in the host to produce directed selection in the pathogen.



5. Rotation of monogenes to control disease.

resistance. The initial variety grown would not necessarily have to have any particular monogenic or horizontal resistance and could be grown until a patho-

gen race appears which would attack it severely enough to warrant development of a resistant variety. The rice breeder then would identify and incorporate monogenic resistance to the pathogen into a new variety agronomically similar to the initial adapted variety. The new monogenic-resistant variety No. 1 would be introduced into the area and grown for as long as the resistance is effective. When a pathogen race develops which could successfully attack monogenic-resistant variety No. 1, the plant breeder would go through the same procedures and develop monogenic-resistant variety No. 2 to replace monogenic-resistant variety No. 1. The same sequence would occur which would result in the development of monogenic-resistant variety No. 3. Three hypothetical monogenic-resistant varieties would now have been developed, each identical with and as desirable as the initial variety except that each of the three contains a single, separate monogenic-resistance gene, and each of the varieties is resistant to only one race.

Monogenic-resistant varieties such as these have been developed in several crops for resistance to several pathogens. As Van der Plank (10) has pointed out, these have not been effectively utilized and the resistance has been ephemeral and useless. When a pathogen evolves that can attack monogenic-resistant variety No. 3, the farmer should be able to return to monogenic-resistant variety No. 1 and the process recycled indefinitely if the plant breeder and plant pathologists have conducted their variety development program properly. Person has discussed the fate of host genes for resistance and pathogen genes for pathogenicity in 'natural occurring disease systems'. He did not consider that plant breeders and pathologists could exert directed selection on the pathogen population each crop season through manipulation of host resistance genes. Because of this directed selection in the host by the plant breeder using monogenic resistance, directed selection occurs in the pathogen population. By the time race 3 has evolved, directed selection produced by the plant breeder coupled with naturally occurring stabilizing selection in the pathogen population against races with excess genes for pathogenicity should have eliminated race 1 from the natural population; therefore, monogenic-resistant variety No. 1 would not be susceptible to the resulting pathogen population whose evolution was controlled by the plant pathologist and plant breeders. With this system for use of varieties, the farmer could continue to rotate plantings of monogenic-resistant varieties indefinitely, based upon the genotype of the pathogen population with reduced likelihood of an epidemic.

There are obvious difficulties with such a generotation system which any serious student of plant pathology or plant breeding can visualize. However, some of the advantages are: 1) genetic vulnerability is limited or reduced; 2) development or occurrence of pathogen races is controlled by plant breeders and pathologists through directed and stabilizing selection; 3) monogenic resistance is deployed and effectively managed; and, 4) strong monogenes for resistance are conserved and can be used repeatedly over a long time.

To successfully implement such a system of blast control in Korea should not be overly difficult. All of the necessary ingredients, including an excellent forecasting and race identification system are present. The plant breeders and plant pathologists cooperate well and through the strong system of the Provincial Office

of Rural Development (PORD), rice farmers can be rapidly and adequately informed of the control plan. What remains to be done is the ORD plant breeders and plant pathologists must identify the specific resistance gene (or genes) which is effective against the present race(s) of blast prevailing in Korea, identify these races on the basis of genes for pathogenicity, and release varieties immediately which possess the monogene for resistance.

When the new variety or varieties which possess the monogene(s) for resistance to the prevalent blast races now in Korea are released, they should be introduced in a thorough and complete manner with the present susceptible varieties virtually being eliminated from use. The reason for introducing the new resistant variety (varieties) on a large scale basis is to avoid and/or discourage the development of adapted mutants which are capable of attacking the new variety.

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The role of varietal resistance in disease management[1]

Pat Crill, F. L. Nuque, B. A. Estrada, and J. M. Bandong[2]

THE RATIONALE FOR HOST RESISTANCE

Rice yields have not increased as much as they were expected to when IRRI was established. Initial yield increases were phenomenal, especially with the release of IR8 and the generation of supporting technology. That these initial successes have not been enlarged upon is a concern to many. Numerous explanations have been offered. The only factor of significance, however, is the co-evolution of diseases and pests on new varieties grown with the new technology.

The reason for IRRI's initial success was that IR8 was a new plant type with a new germplasm. The pathogen populations were not capable of attacking IR8; they had to evolve and develop forms capable of overcoming the new plant types. They had to adapt to new varieties and new crop production practices before they were capable of existing at the present optimal level.

The evolution of pathogens is directly controlled by the varieties and cropping production practices used by farmers in a given area. IR8 was greatly different from the traditional japonica and indica varieties. When new varieties, especially ones that differ greatly from the traditional varieties, are grown, the pathogen populations are confronted with a totally new environment and cannot incite disease at an optimum level.

When pathogen populations are evolving and adapting, new varieties and new crop production practices are used successfully. This was the basis of the success of the green revolution in rice. The green revolution has not progressed at the same rate it was initially expected to because the pathogen populations have adapted to the new varieties and new crop production practices (6).

Evidence of the adaptation of pathogen populations to new varieties is readily

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[1]Presented at the 1981 Annual Meeting of the Pest Control Council of the Philippines, University of the Philippines at Los Baños, College, Laguna, 13-16 May 1981.

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available from experiments at the IRRI experimental farm. Yields of IR8 at the IRRI farm, which exceeded 10 t/ha in 1966, now rarely exceed 3-4 t/ha. Yields of new varieties may approach the 10-t/ha benchmark, but they have not been attained, primarily because the new varieties are similar to IR8. When released they bring about an environment similar to that brought about by IR8. Therefore they are incapable of expressing their maximum yield potential on the IRRI farm or in farmers' fields. This major constraint affects rice yields in Asia.

A specific, documented case that supports this concept is the sheath blight disease of rice. Before the release and adoption of the IR8-type varieties, sheath blight was not a problem in rice production in Asia, especially Southeast Asia. The IR8 plant type is a significant departure from the plant type of traditional varieties. The IR8-type plant is short statured, early maturing, high tillering, and nitrogen responsive. Farmers have used these plant characteristics by increasing the plant population densities, applying more fertilizer, and growing more crops of rice per unit of time (6).

Sheath blight was never a problem before the new varieties were introduced. The traditional varieties have fewer, well-separated tillers. Unlike the short plants with high numbers of tillers, they do not provide a favorable growth environment for the sheath blight pathogen. Farmers created an ideal environment for the sheath blight pathogen when they adopted the new varieties, began to increase the plant population per hectare, and grew more crops per unit of time.

Since the introduction of the IR8-type varieties in the early 1960s, sheath blight disease has changed from a minor disease, only occasionally observed and considered a scientific curiosity, to a disease of major importance. It now probably causes more losses than any other fungus disease of rice.

The value of the new, improved varieties grown with the new technology is beyond measure. The pertinent question is how to minimize the effect of sheath blight on the new varieties. How can plant pathologists protect and safeguard the yield advances made by rice breeders and agronomists? Only a novice or the ignorant would suggest that the advances in rice variety development and rice production be discarded in favor of a return to old varieties and old production methods.

All research programs that result in progress always produce some unexpected side effects, which are never known in advance, but whose presence is always anticipated. This major consequence can be expected from any active, progressive, and successful variety development program. Disease management and control practices must be formulated specifically for new varieties as they are developed and released. If the control strategy is resistance, breeders and pathologists must be confident that the new variety will perform under favorable epidemic conditions. If resistance is not available or cannot be incorporated into varieties, plant pathologists must devise control strategies to protect the new varieties when used.

Disease control is a critical element of modern intensive crop production. In intensive crop production the occurrence of crop plant diseases often results in significant economic losses. The modem farmer who uses the best adapted high

yielding varieties, good water control, adequate to maximum fertilizer, and good weed and insect control cannot afford to ignore plant disease control. If disease control is ignored, crop production likely will be reduced, and all the inputs for varieties, irrigation and water control, fertilizer, and weed and insect control will be lost.

The following measures can be used to control plant diseases in specific situations.

- regulatory controls such as quarantines, embargoes, and inspections:
- cultural controls, including the manipulation of various farming practices that favor the crop at the expense of the pathogen;
- chemical control such as systemic and nonsystemic fungicides and chemical control of vectors; and
- host resistance, including the specific and nonspecific disease-resistance mechanisms.

When it is available and can be used, host resistance is generally regarded as the most economical and effective plant disease control measure. It can be classified and categorized in a number of contexts. Epidemiologists consider disease resistance in a totally different way than biochemists, who in turn view disease resistance much differently than plant breeders. Disease resistance is effective and has value only when it is used in a crop variety to eliminate or reduce disease losses. Therefore plant breeders who incorporate disease resistance mechanisms into crop varieties are the first and primary group to be concerned with disease resistance.

From a plant breeding viewpoint, host resistance mechanisms can be classified as 1) those controlled by single or monogenes, and 2) those controlled by multiple or polygenes. Such a simple classification is not only justified but also essential to the development of plant breeding methods. The breeding, screening, and selection methods used for monogenically controlled characters are totally different from the methods used for polygenically controlled characters.

One of the most significant plant diseases in history is the blast disease of rice (*Oryza satiua*) caused by *Pyricularia oryzae* (22). The rice blast disease symptoms occur on the leaves, nodes, peduncles, panicles, and grains of the rice plant. The most severe losses occur when the nodes or peduncles of the plant are attacked and grain does not form in the panicle. Neck rot is the common name of the disease caused by *P. oryzae* when it attacks the peduncle. Early neck rot infections may result in complete yield loss.

For many years, rice breeders and pathologists have attempted to control blast disease by incorporating resistance into agronomically acceptable rice varieties. Thus far, any control achieved has been only temporary and totally unsatisfactory (1). The control of rice blast with disease-resistant varieties remains one of the major challenges to rice researchers.

Two types of resistance to rice blast that potentially can be used by rice breeders and pathologists to reduce losses have been identified. One type is polygenically controlled and is termed horizontal resistance. The second type is monogenically controlled.

HORIZONTAL RESISTANCE

Horizontal resistance is considered as disease rate reducing; varieties that have only horizontal resistance become diseased in environmental conditions conducive to disease development. Horizontally resistant varieties are susceptible in that they become diseased, but disease development is limited or is less than that on highly susceptible varieties (5,10). From a varietal development standpoint, horizontal resistance is desired because so many genes contribute to resistance that the pathogen cannot mutate sufficiently to overcome all of them (4). This reasoning is based upon two attributes claimed for horizontal resistance:

- Horizontal resistance is stable and unchanging.
- Horizontal resistance is equally effective against all races of a pathogen (25).

Neither is true.

Van der Plank (26) later defined horizontal resistance as resistance not lost through adaptation by the pathogen. He stated further that if resistance is lost by parasitic adaptation, then the resistance is not horizontal. For these statements to be true, the pathogen must remain a static, nonchanging entity incapable of evolving types that can cause more or less disease. Definitions do not alter biological facts. The mass of evidence indicates that when a pathogen population confronts a variety with resistance that is characterized as ratereducing and polygenic (horizontal resistance?), adaptation occurs and the diseaseinciting capacity of the pathogen is altered. Horizontal resistance, as defined by Van der Plank, probably does not exist. The first definition (25) is used in this paper.

Stability of horizontal resistance

As early as 1952, Walker (28) noted that although many potato varieties with polygenically controlled resistance to late blight had been developed, the resistance was not effective under epidemic conditions. Ten years later, Thurston et al (24) reported that the relative general levels of such resistance, compared with those of standard varieties did not change from year to year. In epidemiological terms, with low levels of inoculum or in conditions less than optimum for an epidemic, such resistance may be termed stable. Such polygenically controlled horizontal resistance however, is not effective in preventing crop losses in epidemic conditions. The reaction of crops with horizontal resistance to disease is dependent on environment and cannot be considered stable.

The stability of horizontal resistance has been attributed to stabilizing selection (25) because of the interaction of a large number of genes. Stabilizing selection is a counterpart of directed selection (5). Whenever any population is confronted with selection pressure, measurable phenotypic changes occur because of directed selection. Stabilizing selection does not operate for a specific character, e.g. disease resistance, when directed selection pressure is being applied for the same characteristic of the population. Resistance will not remain stable when varieties with horizontal resistance are introduced into cultivation because selection pressure to overcome such resistance is applied on the pathogen population. The pathogen population responds to the horizontal resistance in the varieties by accumulating genes for virulence, not genes for pathogenicity. This proposition

was evaluated at IRRI using the sheath blight disease, which is incited by *Rhizoctonia solani*, and for which, in rice, no monogenes for resistance are known.

More than 20,000 entries from the IRRI rice germplasm bank have been evaluated for their reaction to the sheath blight disease. Among the least affected by sheath blight were Tapoochoz, Bahagia, and Laka. A standard susceptible variety is IR1487-372-1-1. The reactions of these four varieties have been confirmed in numerous experiments. They were grown in the field and artificially inoculated with a single isolate of the pathogen, which was originally isolated from the susceptible variety IR1487-372-1-1. Inoculum production and inoculation procedures have been described (14). The fungus was isolated from plants of each variety, inoculum was produced, and healthy plants of the same variety were reinoculated. At the same time several cultures of each isolate were prepared and stored. The fungus was cultured on each of the 4 varieties for 7 successive crops; 28 isolates were produced from the original. Isolates from each of the crops were simultaneously inoculated onto healthy plants of the same varieties on which they were cultured (Table 1). In another experiment, isolates obtained from a single variety for seven crop cycles were inoculated onto all four varieties (Table 2). Disease incidence was measured as the percentage of infected leaf sheaths 3 weeks after inoculation.

Obviously the sheath blight pathogen can adapt to varieties that have horizontal resistance to the disease (Table 1). The isolates from all three horizontally resistant varieties caused increased disease severity on the varieties from which they were isolated. Isolates from the susceptible variety caused decreased disease severity after adaptation. When isolates from a single variety were inoculated on all varieties, the pathogen responded by producing more disease on the horizontally resistant varieties than on the susceptible variety (Table 2). Disease severity caused by isolates obtained from the susceptible variety either did not change or slightly decreased on the horizontally resistant varieties as well as on the susceptible variety (Table 1, 2).

The sheath blight pathogen responded to selection pressure applied by the

Table 1. Comparison of the mean percent infected leaf sheaths 3 weeks after inoculation with Rhizoctonia solani isolates obtained from 7 crop cycles (C1-C7) on 4 varieties with the original (Co) isolate.

R. solani isolates from different crop cycles	Infected leaf sheaths (%)			
	Tapoochoz (horizontally resistant)	Bahagia (horizontally resistant)	Laka (horizontally resistant)	IR1487-372-1-1 (susceptible)
Co	34.7	39.6	30.8	55.1
C1	50.0	33.1	38.6	35.6
C2	30.4	37.1	31.2	46.2
C3	31.2	32.9	29.1	32.3
C4	32.7	40.7	36.3	65.4
C5	52.0	33.8	16.7	49.3
C6	27.9	74.3	31.2	42.8
C7	62.6	60.7	36.5	40.7

Table 2. Comparison of the mean percentage of infected leaf sheaths of 3 horizontally resistant rice varieties and a susceptiblevariety inoculated with *Rhizoctonia solani* isolates obtained by successive isolation and reinoculation of 7 consecutive crop cycles of the same 4 rice varieties.

Variety	Crop cycle	Infected leaf sheaths (%)			
		Horizontally resistant to *R. solani*			Susceptible
		Tapoochoz	Bahagia	Laka	IR1487
Tapoochoz	C1	28.6	32.9	25.9	39.3
	C2	32.1	35.6	37.0	53.9
	C3	37.2	34.6	35.4	46.7
	C4	24.1	25.0	23.1	33.7
	C5	30.9	30.5	32.5	47.4
	C6	19.7	33.7	43.2	70.5
	C7	38.0	37.2	37.7	58.9
Bahagia	C1	28.6	23.8	23.4	35.4
	C2	28.2	34.2	35.5	54.2
	C3	40.2	40.0	38.7	68.8
	C4	25.0	32.9	33.3	51.5
	C5	31.7	30.5	29.1	36.4
	C6	42.5	86.9	54.5	71.2
	C7	44.9	54.7	59.4	81.4
Laka	C1	34.7	31.7	40.3	45.7
	C2	36.4	40.5	35.5	71.4
	C3	42.3	46.7	41.0	62.8
	C4	20.5	32.0	22.8	26.0
	C5	32.9	34.1	35.4	38.3
	C6	26.0	32.0	28.8	54.8
	C7	27.3	34.2	35.1	36.8
IR1487	C1	29.3	30.9	36.8	33.8
	C2	22.5	33.8	25.0	28.4
	C3	22.6	21.7	26.0	27.3
	C4	29.6	29.6	27.5	45.2
	C5	32.9	37.7	21.5	20.2
	C6	32.0	30.0	32.0	54.2
	C7	30.4	26.9	24.6	29.7

horizontal resistance, causing increased disease severity. This increase must be presumed to result from gradual accumulation of polygenes which, collectively, govern virulence. It is clear that the pathogen responded to selection pressure induced by horizontal resistance, and adapted to the horizontally resistant varieties. The pathogen is not stable, as assumed by the proponents of horizontal resistance, but is dynamic, depending upon the selection pressures applied.

Directed selection is the evolutionary force acting upon a population of a species that results in the incorporation of new characteristics into the population (5,10). Directed selection occurs in pathogen populations when the pathogen is confronted with resistance, either monogenic or polygenic. Buddenhagen and Reddy (3) stated “It is difficult to conceive of a single ‘pathogen’ . . . as representing pathogenic populations of many billions of individuals . . . and hard to realize that none of the individuals present today existed in last year‘s breeding

plots nor will they be present in next years screening experiments.'' Those individuals that do not reproduce are lost while those that do, survive. Those that reproduce the most survive the best; the result is a dynamic pathogen population that is adapting to the crop varieties in the field.

In a study of horizontal resistance to leaf blast in rice, Villareal (27) identified seven genetic components that affect disease development:

1. Number of spores required to cause infection.
2. Latent period of the pathogen in the host.
3. Proliferation of the pathogen in the host.
4. Number of lesions produced per unit of spores.
5. Sue of lesions.
6. Lesion growth and expansion rate.
7. Number of spores produced per lesion.

Changes in any one of these components result in a corresponding change in disease severity. Rice breeders and pathologists must consider at least four factors when they contemplate the use of horizontal resistance to leaf blast:

1. Each of the seven genetic components of horizontal resistance probably functions independently of the others and results in the expression of a single measurable character, e.g. horizontal resistance to blast. Each component is controlled by the interaction of a minimum of one gene in the rice plant and one in the blast fungus.
2. All seven components directly affect the reproductive capability of the pathogen. As survival ability is directly related to the reproductive capability, each component has the potential to determine which individuals in the population survive.
3. Selection pressure is immediately applied on the pathogen population when a variety with horizontal resistance consisting of the seven components is introduced. Each component applies direct selection pressure on the individuals of the population.
4. Directed selection of individuals within the pathogen population occurs immediately. Those variant individuals within the pathogen population that can successfully overcome the effects of the most components are immediately selected. These individuals are the ones that reproduce first and the most rapidly thereby providing the genetic base for succeeding generations of the pathogen.

In brief, the seven genetic components of horizontal resistance operate independently, each directly affecting the reproductive capability of the pathogen. Selection pressure on the pathogen is immediate. Those pathogen individuals that can overcome the most resistance components become the genetic base for succeeding pathogen generations.

Effectiveness of horizontal resistance

The second attribute of horizontal resistance (that it is equally effective against all races of a pathogen) is a myth. In 1978, IRRI began evaluating rice varieties from Japan that possess high levels of field resistance (horizontal resistance) to blast (Table 3). About 20 varieties and lines with horizontal resistance to blast were

evaluated using about 40 isolates of the pathogen (15, 16). Many of them exhibited high levels of horizontal resistance to specific races but were highly susceptible to other races (Table 4).

More importantly, horizontal resistance is ineffective when environmental con ditions favorable for epidemics are present (10). The development of the rice blast epidemic in Korea provides ample evidence of the ineffectiveness of horizontal resistance to blast. The variety Milyang 30 has more horizontal resistance to leaf blast than any other variety evaluated at IRRI. The high level of horizontal resistance was not effective in controlling the blast disease, even in years during which the environment was not particularly favorable for blast development. in 1977, the environmental conditions in Korea were not especially favorable for blast, but were highly favorable in 1978, especially in the southern part of Korea, as reflected by the disease reactions on Milyang 30 (Table 5). Conditions for a blast epidemic were less favorable in 1979 and 1980 than in 1978. In many locations, however (Table 5), blast was a problem on Milyang 30 in 1979 and 1980 when it comprised

Table 3. Reaction of 9 Japanese field-resistant rice varieties to different blast isolates (1978).

Variety	Isolates (no.) with	
	Resistant (0-2) reaction	Susceptible (5-9) reaction
Ou 247	35	4
Chiyohikari	28	8
Kogane-Masari	27	10
San-in 63	25	6
Harima	21	7
Sensho	21	11
Tokai 26	18	22
Tangin Bozu	18	13
Suzuhara-mochi	5	30

Table 4. Effectiveness of Japanese field resistant rice varieties against Philippine isolates of the blast fungus (1979).

Japanese varieties (no.)	Philippine isolates (no.)
20	16
19	8
18	5
17	4
16	6
15	1
13	2
12	1
9	2
8	2
6	1

Table 5. Leaf blast reactions of the rice varieties Milyang-30 and Tongil at 17 sites in Korea over a 4-year period.

Site	Leaf blast reaction[a]							
	1977		1978		1979		1980	
	M-30	Tongil	M-30	Tongil	M-30	Tongil	M-30	Tongil
Suweon	1	1	2	5	4	6	5	9
Icheon					5	9	4	8
Hwaseong					6	8	4	9
Chuncheon					3	9	4	9
Cheolweon					0	0	1	0
Cheongju					4	9	4	8
Yuseong					1	4	4	8
Iri (HCES)					4	7	4	8
Iri (PORD)					4	8	3	7
Imsil	2	3	3	7				
Gwangju					3	7	5	5
Jangseong					5	8	4	8
Chilgog					4	7	5	9
Milyang					2	5	4	8
Jinju					3	8	4	9
Jinyang	2	7	6	9				
Sacheon					4	8	5	9

[a] Based on the IRTP blast nursery instructions.

about 40% of the area planted to rice. The blast disease could be controlled only with fungicides when the predominant variety was Milyang 30 (8). The situation is basically the same in Japan where farmers plant field-resistant varieties of rice but are prepared to spray with fungicides when surveys indicate they are necessary.

It appears that horizontal resistance, as portrayed with the sheath blight and blast diseases of rice, is no more and may be less stable than monogenic resistance. There are a few reports, mostly unverified, of varieties considered to have horizontal resistance that supposedly have been grown over large areas for many years. They are often cited as evidence to support plant breeding programs designed to capitalize upon horizontal resistance. No single variety of any major crop with resistance incorporated through a specific screening program for horizontal resistance has been grown successfully over a large area in intensive crop production.

MONOGENIC RESISTANCE

Pros and cons of monogenic resistance

Monogenic resistance has been referred to as vertical resistance, racespecific resistance, true resistance, immunity, etc. By *monogenic resistance* we mean the resistance expressed by the lack of macroscopic symptoms in the host when it is inoculated with a pathogen that produces symptoms on a check variety inoculated under the same conditions. Such reactions are termed monogenic resistance when they are controlled by single genes (either dominant or recessive,

nuclear or cytoplasmic) and are directly transferable from one variety to another through plant breeding methods. We recognize that many hypersensitive reactions are controlled by single genes; for practical purposes of variety development, we include them in the broad grouping of monogenic resistance. Reactions that appear controlled by a single gene but are affected by modifier genes are not considered monogenic resistance. The ultimate test for monogenic resistance is the demonstration of a gene-for-gene relation between the host and pathogen, in which susceptibility is characterized by the production of symptoms, and resistance by the absence of symptoms.

Most of the major successes in breeding disease-resistant varieties have been achieved with monogenic resistance. That is not surprising because 1) in a plant breeding program, monogenically controlled characters are much easier to manipulate than polygenically controlled ones, and 2) plant pathologists have only recently been able to distinguish polygenically controlled diseaseresistance mechanisms and develop methods for evaluating them. In contrast, sources of monogenic disease resistance are relatively easy to detect and identify. Disease-screening techniques to detect monogenic resistance are simpler than those required to detect horizontal resistance. Monogenic resistance is most widely used because it is easier to manage in a disease resistance breeding program.

The use of monogenic resistance by plant breeders has been criticized (13,25) because monogenic resistance has failed to control diseases in intensive crop production and has led to epidemics with disastrous consequences. The argument is that because monogenic resistance has failed, plant breeders should rely upon horizontal resistance.

Monogenic resistance has failed and costly epidemics have occurred. Two classical examples are the wheat rust epidemics in 191 6 and 1935-37 and the corn leaf blight epidemic in 1970-71, in North America. In those years, varieties with monogenic resistance were widely cultivated. The general assumption is that such varieties are more susceptible to epidemics. This does not mean that an epidemic is more likely to occur when a variety that has monogenes for resistance to a specific disease is widely grown.

Two of the most disastrous epidemics in history, the Irish potato famine and the Bengal rice famine, occurred on crops that had no monogenic resistance. The crops in the field were probably quite diverse genetically because all farmers produced their own planting stock each year. It may be argued that, potatoes were not a native crop to Ireland and the pathogen was introduced after the crop became established. Although the argument has some merit, it can not be made with respect to the Bengal famine. Rice and its pathogens were well known and established in Bengal at the time of the epidemic in 1942 (22). In fact there had been considerable effort to develop rice varieties resistant to the brown spot disease, which was the principal factor of the Bengal famine. The varieties involved probably possessed a high level of horizontal resistance to the brown spot disease (22). They most certainly were not monogenically resistant to brown spot. Susceptible varieties, horizontally resistant varieties, and varieties with monogenic resistance rendered ineffective by a new pathogen race will succumb when conditions are optimum for the development of an epidemic.

The recent rice blast epidemic of 1978 in Korea is a well-documented example (8, 10). A new race, first detected in 1977, was capable of attacking all the high yielding indica-japonica varieties (IJV's) that had the same monogenic resistance as Tongil. In 1978, Tongil and its derived varieties were highly susceptible to the new race of blast in most parts of Korea; they still are (Table 6). The contributions of Tongil and related IJVs to increased rice production in Korea were significant (Table 7). The monogenic resistanceto blast, which was incorporated into Tongil, provided essentially blast-free crops from 1972 through 1977. When the monogenic resistance failed in 1978, yield losses were significant and disrupted the Korean economy (8). The economic development plans of the country had to be revised because suddenly Korea was no longer self-sufficient in rice production. The cost of the actual yield losses is insignificant compared with the costs of the fungicides imported to control the disease, the cost for equipment and labor to apply them, and the rice that had to be imported.

Monogenic resistance to rice blast had failed in Korea. There had been no significant planning to develop alternative diseasecontrol strategies. Korean scientists almost immediately located sources of monogenic resistance to the new race, but the resistance had to be incorporated into acceptable varieties and seed

Table 6. Comparison of average yields of milled rice of indica japonica varieties (IJV's) with those of all varieties and increase in production of milled rice due to the IJV's over a 9-year period in Korea. (After Crill et al [8]).

Year	Mean yield (t/ha)		Total yield (million tons)	Increase due to IJV's (thousand tons)	Area planted to IJV's (%)
	IJV's	All			
1971	-	3.4	4.00	-	-
1972	3.9	3.3	3.98	116.2	16
1973	4.8	3.6	4.23	116.0	10
1974	4.7	3.7	4.42	217.1	15
1975	5.0	3.9	4.63	416.6	23
1976	4.8	4.3	5.18	442.5	44
1977	5.5	4.9	5.97	858.1	54
1978	4.9	4.7	5.78	474.0	85
1979	4.6	4.5	5.46	194.0	61

Table 7. Predicted and actual 1978 yields of milled rice for IJV's and traditional varieties in Korea and losses attributed to the rice blast disease (Office of Rural Development, Suweon Korea).

Year	IJV's		Traditional varieties		Total production (million tons)
	Mean yield (t/ha)	Area (%)	Mean yield (t/ha)	Area (%)	
1978 predicted	5.5	85	4.2	15	6.36
1978 actual	4.9	85	4.4	15	5.78
Actual losses in 1978 due to blast epidemic					0.58

multiplication of the new variety had to be completed before farmers could use the new source of resistance. A significant amount of seed of a resistant variety could be supplied to farmers, at the earliest in 1981. In the meantime, losses to blast continued although they were not as great as those in 1978 (8).

The rice blast epidemic in Korea in many ways is typical of other historical epidemics in that no contingency plan was available to be put into effect when the monogenic resistance failed. It is atypical in that researchers were able to generate new resistant varieties rapidly. The rice blast epidemic in Korea clearly illustrates that monogenic resistance is not completely safe and that it must be properly managed if yield losses are to be avoided when new pathogen races develop.

Managing monogenic research

All strategies using monogenic disease resistance require knowledge of genetics of resistance and pathogenicity. To be successful, they require the monitoring of pathogen races throughout the cropping area concerned.

Gene pyramiding, The pyramiding of successive genes for resistance into an adapted variety has been the most widely used strategy (18, 20, 21). A resistant variety is developed and grown until a new race of the pathogen that can cause disease on the resistant varieties appears. A monogenic source of resistance is identified and incorporated into the adapted, but susceptible, varieties, usually by a modified backcross method. New varieties resistant to the new race can be developed relatively rapidly in this manner.

The advantage of this strategy is that when new varieties are developed by backcrossing a resistant-source variety into the currently adapted varieties, the new resistant variety differs very little from the adapted one. The relative potential and performance of the new resistant variety should not be much different from those of the recurrent parent. This means extensive testing for yield, quality, and other factors is not as time consuming. Furthermore, the breeding and back-crossing can be initiated as soon as a new race is identified. In most instances, it is unnecessary to specifically identify the monogene for resistance to the new race, which is being transferred to the old adapted variety. The genetics of resistance and pathogenicity can be studied simultaneously during the variety development and breeding process. The method can be used in developing pure-line varieties or parental lines for synthesis of hybrid varieties. If the monogene for resistance to the new race is dominant, it may be possible to generate resistant F_1 hybrid varieties for temporary use by using male or female lines homozygous for the monogenic resistance, but still heterozygous and unfixed for certain other characteristics.

The supposed disadvantage of gene pyramiding is that the number of mono genes for resistance is limited; after all have been pyramided, the pathogen could evolve a superrace that would overcome all of them. This is a strictly hypothetical situation. All monogenes for resistance have not yet been pyramided in a single variety to determine whether a superrace could be induced to evolve on it A more real disadvantage is that as the number of monogenes for resistance are increased, it is more difficult to incorporate the next one identified. The reason is that the variety or line, which has the gene for resistance to the new race, may have

no other monogenes for resistance to other races. When the adapted variety (with multiple monogenic resistance genes) is mated with the source variety (with the monogene for resistance to the new race), all of the monogenes become unfixed in the segregating populations derived from the cross. All the specific races must be screened to identify the recombinant progeny that contain the monogenes controlling resistance to all races. In general, as the number of monogenes for resistance increases arithmetically, the number of progeny that must be evaluated increases geometrically. This problem can be alleviated somewhat by using F_1 hybrids as varieties, and distributing the resistance monogenes between the parental lines. Other problems with gene pyramiding include the possibility of multiple alleles for resistance at the same locus, pleitropic effects, etc. (19).

Multiline varieties. A multiline variety is made up of several component lines. Each line is as genetically similar to every other line as possible, except for monogenes for resistance to a specific disease. The use of multiline varieties as a disease control strategy has been discussed in detail (2). Multilinevarieties of oats and wheat have been developed and successfully used for disease control (2).

The major advantage of multiline varieties is that they confront the pathogen with several monogenes for resistance simultaneously (2). Each monogene for resistance is present in a separate component line. Multilines provide insurance against total crop failure because some of the components should be resistant to certain of the pathogen races. A further advantage is that the component lines with the individual monogenes for resistance have a dilution effect on the inoculum potential. If five monogenic resistant component lines make up a variety and four of them are resistant to all the races prevalent in the inoculum, the inoculum potential is reduced by 80%. This serves to decrease disease severity.

The primary disadvantage of multiline varieties is the time required for their synthesis. It is necessary that all the component lines be nearly identical in intensive crop production where uniform maturity, plant height, and crop quality are essential. Before component lines with different monogenes for resistance can be synthesized, they have to be genetically identified and isolated into separate breeding lines. This essential time-consuming process requires major inputs and research facilities. A theoretical disadvantage is that the deployment of several resistance monogenes at one time generates selection pressure for the formation of a superrace, as with gene pyramiding. The evolution of such a superrace would cause all components of the multiline to become susceptible. This should not occur if pathogen race surveys are conducted and only component lines resistant to the prevalent races are included.

It is possible to use F_1 hybrid varieties in a disease control strategy with multiline varieties. If the monogenes for resistance are dominant, they need to be present in only one parent - either the pollen or seed parent. If the seed parent is selected as common for all components of the F_1 hybrid multiline, the individual monogenes may be incorporated into the common pollen parents through backcrossing. The pollinator lines would not have to be nearly as uniform as when used directly as varieties. After such a series of lines is established, it should be possible to generate new multiline varieties as F_1 hybrids quite rapidly, and at the same time improve yield potential and quality. Used in this way, multiline varieties should be

more acceptable in intensive crop production. The objection to them has been the time required to develop component lines and their not being uniform.

Gene rotation and race prediction. The origin of the concept of crop rotation as a disease control measure is lost in antiquity (9). Stevens (23) discussed the possibility of replanting discarded varieties as a means of disease control. Van der Plank (25) suggested that resistant genotypes could be planted in rotation as a disease control strategy, but did not specify how that could be accomplished. Kiyosawa apparently made a similar suggestion for rice blast control (1 7).

The concept of rotating disease resistance monogenes based upon the prediction of new races of the pathogen was first suggested by Crill et al (11); it was later refined by Crill (5) and Crill and Khush (10). The rotation of monogenes for resistance is based upon the concept that races of a pathogen indigenous to a cropping area are a result of the monogenes for resistance present in the varieties being grown in the area (5). The evolution of pathogen races with monogenes for pathogenicity is controlled by the resistance monogenes present in the varieties being grown.

The primary advantage of the strategy of rotating monogenes for resistance is that resistance genes are deployed only when needed, and a means of conserving and reusing genes is established. The strategy assumes that new genes for pathogenicity will arise in the pathogen population and the strategy is designed to control them before they become important (8,10); yield losses due to the new races should be nonexistent to minor. Superraces will be prevented from developing and the problems of multiple alleles, etc. associated with gene pyramiding should be negated. The strategy also provides a means of using multiple disease resistance in variety-development programs much better than before. The problem of incorporating numerous genes for disease resistance into a single line (7) is eliminated. The strategy is highly amenable to breeding programs concentrating on the development of F_1 hybrids as varieties.

The major disadvantage of gene rotation based upon race prediction is that extensive disease and pathogen race surveys are required for optimal effectiveness. Such surveys have been done for intensively grown crops. The strategy would be difficult to implement when information from such surveys is not available. Genetic studies of resistance and pathogenicity are required, as in gene pyramiding and multiline varieties.

To be effective, the strategy requires a vigorous plant breeding and crop variety development program. Few public plant breeding ‘institutions are capable of developing satisfactory new resistant varieties to be used in a gene rotation program. Gene rotation based upon race prediction is a sophisticated and exacting strategy. It is being used successfully with rice in Korea and with certain high-value crops in North America. As crop production becomes more intensive and the price of food increasingly higher, gene rotation will become increasingly popular as a disease control strategy.

FACTORS AFFECTING DISEASE RESISTANCE RESEARCH

A number of factors dictate disease control strategies. Foremost is the value of the

crop to a country's economic well-being and the potential losses that may be caused by a specific disease. For example, rice is the major crop in most Asian countries, and its adequate supply at a reasonable price makes for a contented populace, which is required for a stable government. It then becomes easy for Asian politicians to support rice research and production. The government indirectly controls research by providing or not providing funds for specific projects.

The type of research, especially disease control, is affected next by the availability of resources such as the equipment and facilities required. If one or several specific diseases have the potential to significantly reduce yields, and a history of yield reduction by specific disease(s) is known, research funds are made available for these specific problems. Research for minor diseases of minor crops is almost never funded.

Monogenic resistance research programs generally require considerably more facilities and inputs than do horizontal resistance programs. With monogenic resistance, it is necessary to maintain laboratories for: 1) culture collections of isolates of different pathogen races and 2) seed-storage facilities for collections of varieties and breeding lines that possess known genes for resistance or serve as sources of genes for resistance to specific races. In addition, genetic studies of pathogenicity and resistance must be in progress continuously, requiring more manpower and facilities.

In contrast, horizontal resistance research can be done with only limited facilities in the field with simple experiments. Specific genetic cultures of the pathogen and elaborate seed-storage facilities are not required. This significant difference in requirements for funds and facilities has stimulated research on horizontal resistance and discouraged the use of monogenic resistance.

SELECTION OF DISEASE RESISTANCE STRATEGIES

Van der Plank (25) stated that there are two types of pathogenic races, two types of pathogenicity, and two types of resistance. This is apparently the first time that these important observations were so succinctly noted. The significance of this observation remains unclear to many who are attempting to control crop diseases with host resistance. Because of this confusion, progress in many disease resistance breeding programs was generally slow. Many repeatedly confuse one type of resistance with another or one type of pathogenicity with another, and devise disease rating schemes that integrate, but do not separate, two types of races. In addition to the three traits of two types listed by Van der Plank, here are two types of resistance genes.

Two types of pathogenicity and pathogenic races

The terms for *pathogenicity* are *pathogenicity* for type 1, and *virulence* for type 2. These terms have been used in the literature for many years, often interchangeably, and with many different meanings. In a strict sense, and for purposes of this discussion, pathogenicity is the property of a pathogen that allows it to incite disease (29). Pathogenicity is controlled by monogenes, and in many pathogens the ability to incite disease is inherited as a recessive characteristic. The type 1 race

of a pathogen then is controlled by monogenes. Pathogenic races differ from one another in single genes that determine whether the pathogen can incite disease on a particular host variety - a qualitative character (monogenically controlled) expressed as either the *presence or absence of disease.*

Virulence (or type 2) is the property of the pathogen that determines the severity of disease. Virulence is controlled by polygenes. It is never expressed unless the diseaseinciting individual of the pathogen population has expressed the property of a pathogenicity (ability to incite disease). The polygenes that control virulence are expressed only after the property of pathogenicity has been expressed. Virulence is measured by differences in disease severity, which are quantitative and polygenically controlled.

Two types of resistance (and resistance genes)

The terms used by Van der Plank (25) to refer to the two types of host resistance were *vertical resistance* and *horizontal resistance.* They were coined in an epidemiological context and have been widely used. As genetic terms for use by plant breeders, they are confusing and less than acceptable. Vertical resistance is best referred to as monogenic resistance (type 1) because it is controlled by single genes. These monogenes quite often are dominant and inherited qualitatively. Monogenes for resistance in the host are recognized by the presence or absence of disease symptoms.

Horizontal resistance is expressed only in the absence of monogenic resistance, and is polygenically controlled. In general, horizontal resistance generally appears to be incompletely dominant and inherited, with additive effects. Horizontal resistance has an effect only when monogenic resistance is absent or does not prevent pathogenesis from occurring. Polygenes controlling horizontal resistance in the host are recognized by a decrease in disease severity. In their absence, disease severity is increased.

Monogenes that control monogenic resistance in the host plant interact directly with monogenes that control pathogenicity in the pathogen. This interaction between monogenic resistance and pathogenicity is expressed and measured only by the presence or absence of symptoms, which reflect disease occurrence. This host-pathogen interaction is the gene-for-gene relation, as postulated by Flor (12). Pathogen races are identified by single gene differences and monogenic resistance is recognized by single gene differences.

Polygenes that control horizontal resistance in the host plant interact directly with polygenes that control virulence in the pathogen. The interaction between horizontal resistance and virulence is expressed and measured by disease severity. Horizontal resistance and virulence are most likely due to the interaction of single genes in the host and pathogen. The combined effect of all single-gene components in the expression of disease severity as a unit character is polygenic and quantitative. This host-pathogen interaction is a genes-for-genes relation (6). Virulence is not discreet, but variable and continuous. Pathogen populations cannot be categorized into specific groups on the basis of virulence as they can be on pathogenicity. Likewise, horizontal resistance is variable, continuous, envir-

onment responsive, and can not be classified into discreet groups.

Horizontal resistance

On many occasions horizontal resistance has not been a suitable disease control strategy. Most of its failures have occurred in intensive crop production; they are less likely to occur in subsistence crop production.

Horizontal resistance is environment responsive and is ineffective under epidemic conditions created by intensive crop production. When epidemics occur, protective sprays are required to maintain stable crop production. Furthermore, horizontal resistance is not stable over time since the pathogen populations are selected for their ability to overcome it. In subsistence crop production, horizontal resistance may remain effective longer.

Horizontal resistance is polygenic, requiring the plant breeder to manipulate many genes. It is not as complex as it may first appear because no genetic studies are required and record keeping is minimal. All research is done in the field. Because it is assumed that the variety responds similarly to all races, no special inoculum production or inoculation procedures are required. The inoculum occurring in the field is often adequate to detect differences in horizontal resistance. Because horizontal resistance to one disease is not effective against a second disease (26), it may be difficult to generate varieties that have multiple-horizontal disease resistance. Furthermore, certain components of horizontal resistance may control one phase of a disease but actually increase severity at another stage of growth - effects similar to those of monogenic pleiotropism.

Horizontal resistance appears to be most suitable for use in subsistence crop production where there is only one major important disease on the crop. It is generally not effective in epidemic conditions. When it is used as a disease control strategy, alternative disease control plans should be formulated for use when epidemics occur.

Monogenic resistance

Monogenic resistance has been ineffective as a disease control strategy on many occasions. Disease-management techniques have been devised for its better use. Each technique has specific advantages and disadvantages that need thorough evaluation before it is selected. Each technique also requires specific breeding methods and relatively extensive laboratories, greenhouses, and fields. Monogenes for resistance to many diseases have not yet been identified, and sources may never be found.

Host resistance is frequently cited as the most economical method of controlling crop diseases. Because of that, administrators often assume that disease resistance breeding programs are inexpensive. They are not! All successful disease resistance breeding programs are expensive. For the most part, they are underfunded at public, tax-supported research institutions. Each year, the private research sector assumes more and more responsibility for developing disease-resistant crop varieties.

Monogenic resistance, primarily because of its effectiveness, will continue to be

the most widely used disease control strategy in crop production. Its advantages are far greater than its disadvantages. The three primary methods for using monogenic resistance may be adapted to numerous variations. These variations offer more potential for successful disease control than has ever before existed.

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The rice blast disease in Korea and its control with race prediction and gene rotation[1]

Pat Crill, Y. S. Ham, and H. M. Beachell[2]

INTRODUCTION

Rice has been cultivated in Korea for about 3,000 years. It is the country's most important food crop.

Throughout the Japanese occupation, especially during World War II, Korea experienced Severe food shortages. From the liberation in 1945 until the end of the Korean War in 1953, Korea depended upon US food grants and imports.

At the end of the Korean war, agricultural production was chaotic. The enactment of US Public Law 480 in 1954 stabilized Korea's food supply. By 1972 the country imported 3 million tons of grain annually, most of it rice. Food importation policies severely depressed the local grain market and debilitated the rural economy. Korean food production stagnated because farmers had less incentive to increase production; a chronic dependence on the overseas food economy developed (5).

INTENSIFICATION OF RICE PRODUCTION IN KOREA

The Korean Government in 1962 established the Office of Rural Development (ORD) to develop agricultural technology and increase food production to the level of national self-sufficiency. Research to increase rice production through the development of new varieties and manipulation of cultural practices was emphasized. By 1964 ORD scientists were training at the International Rice Research Institute (IRRI) and being exposed to the rice breeding philosophies there. An ORD-IRRI cooperative research group, established in 1964, has emphasized the development of high-yielding rice varieties for intensive production. This coopera-

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tive rice research program has achieved notable successes in improved crop production.

Initial efforts to develop improved rice varieties for Korea consisted of introducing rice varieties and breeding lines from around the world. These were evaluated in large-scale adaptation nurseries. The most promising introductions were released as varieties in Korea. Early in the program it was obvious that introducing varieties developed elsewhere would not provide Korea with the kind of varieties required. Characteristics identified as desirable for future Korean varieties were:

1. High grain yield. Grain yield was considered as a function of plant type. The most desirable plant type was a semidwarf, lodging-resistant plant with a high number of tillers and upright or erect leaves to maximize photosynthetic potential.
2. High grain quality. Grain quality is measured primarily by cooking or eating quality. Koreans prefer a rice that has an amylose content of less than 20% and an alkali digestion value between 5 and 7.
3. Cold tolerance. Korea has a typical northern or temperate climate. Rice varieties must be tolerant of cold if they are to grow and yield well.
4. Resistance to rice blast. The only rice disease that had ever caused any serious problem in Korea was rice blast caused by *Pyricularia oryzae.* Blast had reduced rice yields and epidemics occurred periodically.

A rice breeding program to incorporate these four characteristics into one variety was launched in 1965 as a part of the ORD-IRRI cooperative project. The best japonica varieties, which were grown exclusively in Korea, were crossed with various semidwarf lines. The progeny of these crosses were critically evaluated by Korean plant breeders who soon found that all the desirable characteristics had been recombined in IR667-98. Various lR667-98 lines were evaluated in yield trials throughout Korea in 1970 and 1971. Their yields exceeded by 50% those of the best adapted varieties. In 1972, IR667-98 was released as 'Tongil' and planted on more than 180,000 ha, about 16% of the rice land in Korea. The significant increase in rice production that resulted was due not only to the new plant type but to Tongil's resistance to blast (Table 1).

Table 1. Yield comparison of traditional and indica-japonica varieties (IJV's) in Korea.

Year	IJV's		Traditional varieties		Total		Increase due to IJV's (1000 t)[a]
	Yield[a] (t/ha)	% of area	Yield[a] (t/ha)	% of area	Mean yield[a] (t/ha)	Production[a] (million t)[a]	
1971	-	-	3.4	100	3.4	4.00	-
1972	3.9	16	3.2	84	3.3	3.98	116.2
1973	4.8	10	3.4	90	3.6	4.23	116.0
1974	4.7	15	3.5	85	3.7	4.42	217.1
1975	5.0	23	3.5	77	3.9	4.63	416.6
1976	4.8	44	4.0	56	4.3	5.18	442.5
1977	5.5	54	4.2	46	4.9	5.97	858.1
1978	4.9	85	4.4	15	4.7	5.78	474.0
1979	4.6	61	4.4	39	4.5	5.46	194.0

[a] Milled rice.

While plant breeders continued their efforts to improve Tongil, other ORD scientists explored ways to increase rice production through improved soil fertility, irrigation and water control, insect control, mechanization, and various other cultural practices.

During the period from 1962-72 the federal government of Korea had not been idle; numerous projects had been initiated to intensify rice production. Except for disease control, all cultural practices considered essential for intensive crop production were implementea (4).

Tongil was soon joined by a number of other new, improved indica/japonica hybrid rice varieties (IJV's). Each found a special area of adaptation or use as improvements were made in yield, grain quality, and cold tolerance. These concerted efforts resulted in a continuous increase in Korean rice production. The IJV's remained resistant to blast while the old japonica varieties continued to suffer yield losses caused by blast.

Primarily through the intensification of rice production, Korea had become self-sufficient in food and had exported rice since 1975. It appeared that the Green Revolution in Korea had been accomplished with rice, and the major problems of intensive rice production were under control.

DEVELOPMENT OF THE RICE BLAST EPIDEMIC IN KOREA

By 1977 IJV's were grown on about 54% of the rice land in Korea. In September 1977, blast was found on the IJV's in three, very small, isolated areas in the southern part of Korea. Until then rice blast had never been observed on any of these varieties in farmers fields. Just before the infected plants were discovered, two typhoons had struck Korea. Both entered the country from the south; one proceeded north-northeast and the other north-northwest. These isolated pockets of blast disease caused little concern; in 1977 Korea established a world record yield for milled rice with an average of 4.9 t/ha on 1.2 million hectares.

It became obvious in 1978 that rice blast resistance in the IJV's was no longer effective. The 1977 typhoons had spread the new blast race throughout Korea and the 1978 crop was severely infected. The Green Revolution in Korea was about to succumb to a plant disease epidemic just as previous intensive crop production schemes had succumbed when disease control was ignored.

The 1978 rice blast epidemic was totally unexpected; Korean scientists and the government were not prepared to cope with it. Research in rice pathology, especially rice blast control, had been deemphasized after the development and release of the blast-resistant Tongil and other IJV's. No rice blast chemical control studies had been conducted in Korea for nearly 10 years and relatively little research had been done to identify new sources of blast resistance, especially resistance to new races which might develop on the IJV's.

RICE BLAST CONTROL IN 1978-79

The first noticeable effect of the new rice blast race was the early Occurrence and widespread development of leaf blast. Neck blast later became especially severe

in a few local areas. Total yield losses were not disastrous; the average milled rice yield in 1978 was 4.7 t/ha, the second highest yield in Korean history. However, 1978 total rice yields were anticipated to be much higher than in 1977 because in 1978 IJV's were planted on 85% of the rice land compared to only 54% in 1977. From this viewpoint the 1978 yields were disastrous; the government's plans for food production had to be drastically altered and rice exports were discontinued.

Because of the sudden and widespread Occurrence of the epidemic, little could be done to reduce the 1978 damage, but blast control plans for 1979 were formulated even before the 1978 crop approached maturity. Among the disease control measures planned for 1979, two were implemented in 1978. One was the identification of germplasm resistant to the new race(s) and the other was introduction of disease management practices.

In September 1978 approximately 1500 rice blast samples were collected from the diseased IJV's for pathogenicity evaluations on advanced ORD rice breeding lines. Each isolate was inoculated onto about 30 varieties and elite breeding lines. All of the recommended varieties were susceptible, but at least three of the elite breeding lines were resistant to all 1500 isolates. Seeds of the resistant lines were sent to IRRI for winter increase so they could be widely evaluated in Korea in 1979. Many of the 1500 isolates were also identified to the specific race level by Korean plant pathologists.

A strict program of sanitation was practiced by farmers throughout Korea to reduce the inoculum potential of the new race. This included burning straw and chaff after harvest, early plowing of paddies to facilitate decomposition of infested straw, composting straw that was not burned or plowed down in the fall, and late fall and early spring flooding of paddies to aid straw decomposition. Much of the 1979 crop was treated with systemic fungicides that protected against *P. oryzae* from seeding until harvest. The same basic procedures were used for rice blast control in 1980.

EVOLUTION OF A LONG-TERM RICE BLAST CONTROL PROGRAM

With the appearance of the new race of blast, Korean scientists began an evaluation of possible long-term rice blast control programs. At an international symposium held in Suweon, Korea, all the known alternatives to rice blast control were explored, among them cultural practices, chemical control, the use of horizontal or field resistance, and the use of monogenic resistance.

Chemical control

The need for short-term chemical control of rice blast to protect high-yielding varieties until resistant ones could be developed was generally recognized. Chemicals were ruled out as the primary control measure because of their cost and the absence of a selection of several fungicides with different mechanisms.

Cultural practices

Numerous cultural practices reduce disease incidence as well as disease severity. Among these are various exclusion and eradication practices directed toward the

pathogen with emphasis on sanitation. Crop production practices such as manipulating soil fertility, plant population densities, and water management affect disease development. In general, those practices which decrease the incidence and severity of blast also reduce rice yields, so cultural practices could not be considered as the primary solution for long-term rice blast control.

Horizontal or field resistance

Horizontal resistance to rice blast, characterized by disease ratings of type 4 and 5 based on the international standard evaluation system, had been studied in Korea since 1970. In the non-epidemic years, some varieties and breeding lines consistently exhibited horizontal resistance in trials, other varieties remained blast-free, and others were destroyed. In the 1978 epidemic all varieties considered to have horizontal resistance suffered severe blast symptoms; their horizontal resistance did not provide adequate protection under environmental conditions suitable for epidemic disease development. Horizontal or field resistance as a long-term rice blast control measure was rejected.

Monogenic resistance

The monogenic resistance to blast that was incorporated in Tongil and the subsequent IJV's controlled blast for six years (1972-1977). In the winter 1978 blast screening nurseries, three elite breeding lines and several source varieties resistant to the 1500 isolates of the "new" race were identified. This resistance, later demonstrated as monogenic, resulted in the absence of disease just as the IJV's were disease-free until the new blast race developed in 1977. Monogenic resistance was considered to be the most desirable alternative for long-term rice blast control because of

- the structure of the breeding program,
- the capability to rapidly develop and introduce new varieties into the intensive rice production areas of Korea, and
- the absence of disease on resistant varieties.

MANAGEMENT OF MONOGENIC RESISTANCE TO CONTROL RICE BLAST

Monogenic resistance to blast had not been properly managed in the IJV's; no solution was immediately available to prevent blast epidemics from continuing. The only hope of curtailing epidemics in 1979 and 1980 was the extensive use of chemicals. To maintain adequate yields in 1979, more than $350,000,000 of blast control fungicides were imported. A considerable, although lesser expenditure, was also made in 1980.

In early 1979 a rice blast control program based upon the concepts of race prediction and gene rotation described by Crill (2) and proposed by Crill and Khush (3) was adopted by Korean scientists following approval by the government. The blast control procedures using monogenic resistance and race prediction follow.

- Monogenic resistant sources and lines are identified annually in 15 national blast screening nurseries.

- Breeding lines that express monogenic resistance reactions in the field screening nurseries are evaluated for resistance to specific races in greenhouse trials at three Korean sites during the winter.
- Lines identified in the winter greenhouse screening as monogenic-resistant to specific races are field evaluated for agronomic acceptability the following summer. All lines identified as potential variety candidates are seed-increased in the Philippines during the winter.
- The variety candidates are evaluated in national yield trials conducted by ORD at several geographic sites, and their reaction to blast is evaluated at all the 15 disease screening nurseries. Also, all the potential variety candidates are evaluated at more than 4,400 farmer demonstration trials conducted by ORD. This is the first opportunity for Korean rice farmers to observe the new varieties; they are encouraged to observe them several times during the season.
- A variety or varieties may be selected for release based upon performance in yield and disease nurseries and demonstration trials. Varieties selected for release in Korea during the summer are seed-increased in the winter in Philippines. The increased seed is managed as foundation stock seed.
- After a variety is selected for release to Korean farmers, it is evaluated for resistance to blast in a number of countries including Nepal, India, and the Philippines. If the variety is susceptible to blast at any site, particular attention is paid to varieties in the same nursery that express monogenic resistance. These varieties are immediately utilized as parents to cross with the newly released Korean variety. Progeny from the crosses are evaluated in the blast nursery where the new variety was susceptible and the resistant source variety was located. Progeny exhibiting monogenic resistance are selected and returned to Korea for agronomic and disease resistance evaluations. Furthermore, they are evaluated in the foreign blast nursery where the new variety was susceptible.
- The race of blast attacking the new variety at the foreign site will have the same gene for pathogenicity as any race capable of attacking the new variety that evolves in Korea. Screening of the new variety for susceptibility to blast at the foreign sites predicts which race or gene for pathogenicity is required for the blast fungus to attack the new variety.
- After the new race or gene for pathogenicity is identified, it is relatively easy to locate a monogenic source of resistance and incorporate it into desirable germplasm.

At this stage of the variety development program, it has been determined which gene for pathogenicity is required for the fungus to overcome the monogene for resistance in the new variety. A source of monogenic resistance has also been identified. The plant breeder now has the option of pyramiding this new resistance monogene into the new variety through a recombination or backcrossing pro gram. A second option is to introduce the new gene for resistance into a different germplasm source to develop a monogenic resistant, high yielding variety to use n a gene rotation program.

The source of primary inoculum for blast disease development on rice in Korea

has never been adequately determined. A number of weedy grasses may serve as hosts of *P. oryzae;* they may be the source of primary inoculum (1). Other sources may include infested seed, poorly composted and infested stubble, and infested straw.

Thevariety development procedures used in the gene rotation program and the presumed fate of the genes for pathogenicity in the blast fungus population have been described (3). Genes for pathogenicity in the fungus are assumed to be recessive, but there is no evidence to support or deny this assumption. The fate of genes for pathogenicity when resistance genes are rotated will be the same regardless of dominance.

Because the biology and life cycle of P. oryzae are obscure, Korean scientists have opted to rotate two resistance (R) genes at once. The first variety released will possess only R_1. The second variety will have R_1 plus R_2. The third, R_2 plus R_3, etc. This system of R gene management places extreme selection pressure on the fungus population for about twice as long a period as the rotation of single R genes. The longer period of selection pressure reduces the possibility of genes for pathogenicity surviving in the pathogen population. The longer period may be necessary to eliminate the nonfunctional genes for pathogenicity from the blast fungus population. If it were possible to simultaneously replace all susceptible varieties with new resistant varieties, such nonfunctional genes for pathogenicity could survive only on weed hosts. The rotation of two R genes at a time appears to provide a mechanism for overcoming this problem in Korea.

Rice variety development, just as any other plant breeding program, is an intensified evolutionary process oriented to serve man's needs. Race prediction and gene rotation likewise represent an intensified evolutionary process, guided by plant pathologists and plant breeders to serve the needs of crop production rather than the natural need of the pathogen for survival. Race prediction and gene rotation provide the crop production scientist with a means to intervene in the natural processes of directed and stabilizing selection, and to guide the evolution of pathogen races to effect minimal loss to blast in rice.

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