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Genetics of the Pathogen–Host Association

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I. Introduction

This treatise deals with the cereal rust diseases, not the cereal rust fungi or their cereal hosts alone. For this reason this chapter considers the interorganismal genetics of the association of the cereal rust pathogens and their hosts rather than their intraorganismal genetics. The inheritance of reaction in the host and of pathogenicity in the pathogen have been studied extensively. These studies have shown that inheritance is usually simple with low reaction and low pathogenicity usually dominant, whereas high reaction and high pathogenicity are usually recessive. It is common to find allelism for reaction; however, allelism for pathogenicity has seldom been observed. There are numerous reports of interactions among genes for reaction and a few for

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pathogenicity. These reports were either wrong or the definitive studies to demonstrate their validity were not made. The observed interactions could just as well be at the interorganismal level and perhaps are.

Interorganismal genetics, the genetics of symbiosis, has its foundations in Flor's gene-for-gene concept (1971). Pathogen–host associations are considered to be symbiotic. The aegricorpus (Loegering, 1966) is the result of such a symbiosis and is defined as the living manifestation of the genetic interactions in and between pathogen and host. In the cereal rusts, the infection type (IT) is the phenotype of the aegricorpus, not that of the pathogen or host. Thus the central concept of interorganismal genetics is that the genotypes are of the symbionts, but the phenotype is of the symbiosis.

II. The Origin of the Gene-for-Gene Concept

H. H. Flor (1946, 1947) pioneered the study of interorganismal genetics using flax rust (*Melampsora lini* (Pers.) Lev.–*Linum usitatissimum* L.) as his model. Based on these studies he developed the gene-for-gene concept (Flor, 1971). In his initial studies he dealt with a gene pair in the pathogen corresponding to a gene pair in the host—the corresponding gene pairs (CGP). It should be noted that CGP is plural. The expression "gene-for-gene" is used to designate a concept and should never be translated literally.

To demonstrate the gene-for-gene relationship the ideal model would involve two symbionts in which classical diploid inheritance occurs and that can be propagated as clones. This would make it possible to observe the phenotype of all possible combinations of the F_2 individuals of both symbionts. This has never been done, and no such combination of symbionts is available at present. Of the models worked with, the one that best approximates the ideal is flax rust. *M. lini* has diploid inheritance, and uredial cultures are clones. It is genetically stable and is easily maintained. Flax is self-pollinated and has normal diploid inheritance but is difficult to propagate as a clone. Sequential inoculation of a given plant with different cultures of the pathogen, however, is relatively easy to accomplish. Flor found that the IT was consistent as the plant aged. Often this is not true for the hosts of the cereal rust fungi. It must be remembered that Flor did not set out to demonstrate the gene-for-gene relationship. He developed it as a result of the characteristics of his model, the creative analysis of his data, and the development and use of special methods.

Flor demonstrated more than 25 gene-for-gene relationships in flax

Table I

Inheritance of Pathogenicity in a Cross of Cultures of Race 22 and 24 of *Melampsora lini* When the F_2 Population of Cultures Was Used to Inoculate the Ottawa 770B Cultivar of Flax[a]

Cultivar	Cultures[b]				
	Race 24	Race 22	F_1	F_2	
Ottawa 770B	I	S	I	I	S
				101	32
				Ratio 3:1	
				$\chi^2 = 0.06$; $p = .95–.99$	

[a]Data from Flor (1946).
[b]I, Immune; S, susceptible.

rust. Data (Flor, 1946, 1947) for two will be used for illustration (Tables I–VIII). The symbolization he used for genes and phenotypes will be used in the first tables to illustrate some of the problems encountered with the presentation of the data.

He crossed a culture of race 24 with one of race 22 of *M. lini* and used the F_2 progeny to inoculate the flax cultivar Ottawa 770B (Table I). The cultivar was immune (I) with race 24 (IT 0) and susceptible (S) with race 22 (IT 4). Segregation was 3:1 I:S, indicating that recombination for a single gene pair in the pathogen occurred and immunity was dominant. This gene was designated *AlAl*. He then crossed the Ottawa 770B and Bombay flax cultivars and inoculated the F_2 progeny with the culture of race 24 (Table II). Ottawa 770B was immune (I = IT 0), and Bombay was susceptible (S = IT 4). Segregation was 3:1 I:S, indicating

Table II

Inheritance of Reaction in a Cross of Ottawa 770B and Bombay Flax Cultivars When Inoculated with a Culture of Race 24 of *Melampsora lini*[a]

Cultivars	Culture race 24[b]
Ottawa 770B	I
Bombay	S
F_1	I
	Ratio 3:1
F_2	I 142
	S 52
	$\chi^2 = 0.34$; $p = .50–95$

[a]Data from Flor (1947).
[b]I, Immune; S, susceptible.

Table III

Tables I and II Combined with Genotypes and Infection Types[a] Added[b]

Cultivar	Genotypes	Cultures[c] Race 24 *AlAl*[d]	F_1 *Alal*[d]	Race 22 *alal*[d]
Ottawa 770B	*LL*	I (0)	I (0)	S (4)
F_1	*Ll*	I (0)		
Bombay	*ll*	S (4)		

[a]Genotypes and infection types from Flor (1946, 1947).
[b]F_2 data has been omitted.
[c]I, Immune; S, susceptible.
[d]Infection types are in parentheses.

that recombination for a single gene pair occurred in the host and immunity was dominant. This gene was designated *LL*. Because Ottawa 770B and race 24 were common to both studies, the data are combined in Table III with the genotypes added and the ITs placed in parentheses after Flor's I–S symbolization.

As part of the experiment just described, Flor inoculated Bombay and the F_2 plants with the culture of race 22 by using sequential inoculations and inoculated Bombay with each of the F_2 cultures at the time he inoculated Ottawa 770B. Bombay with race 22 was immune (I = IT 0), and Ottawa 770B was susceptible (S = IT 4). The segregation in both organisms was 3:1 I:S (Tables IV and V), although exactly the same number of individuals were not found in each class as in the work with

Table IV

Inheritance of Pathogenicity in a Cross of Cultures of Race 22 and 24 of *Melampsora lini* When the F_2 Population of Cultures Was Used to Inoculate the Bombay Cultivar of Flax[a]

Cultivar	Cultures[b] Race 24	Race 22	F_1	F_2	
Bombay	S	I	I	I	S
				105	28
				Ratio 3:1	
				$\chi^2 = 1.10$; $p = .50–.95$	

[a]Data from Flor (1946).
[b]I, Immune; S, susceptible.

Table V

Inheritance of Reaction in a Cross of Ottawa 770B and Bombay Flax Cultivars When Inoculated with a Culture of Race 22 of *Melampsora lini*[a]

Cultivars	Culture race 22[b]
Ottawa 770B	S
Bombay	I
F_1	I
	Ratio 3:1
F_2	I 153
	S 41
	$\chi^2 = 1.54$; $p = .20–.50$

[a]Data from Flor (1947).
[b]I, Immune; S, susceptible.

Ottawa 770B. Because Bombay and race 22 were common to both tests, the data are combined in Table VI. Following this report of Flor's data may be somewhat confusing, because in using I and S he sometimes meant immunity and susceptibility of the host, and sometimes he used I and S as symbols for IT. He evidently was aware of the problem, because in his 1959 review the footnote to Table 2, which reports data on the results of inoculating the F_2 plant populations, reads: "I = immune; S = susceptible," whereas in Table 3, which reports the result of using the F_2 cultures to inoculate the two cultivars, the footnote reads: "I = immune (avirulent); S = susceptible (virulent)." The confusion arises from utilizing host-oriented genetic concepts in pre-

Table VI

Tables IV and V Combined with Genotypes and Infection Types Added[a,b]

		Cultures[c]		
Cultivar	Genotypes	Race 24 *anan*[d]	F_1 *Anan*[d]	Race 22 *AnAn*[d]
Ottawa 770B	*nn*	S(4)		
F_1	*Nn*	I(0)		
Bombay	*NN*	I(0)	I(0)	S(4)

[a]Genotypes and infection types from Flor (1946, 1947).
[b]F_2 data has been omitted.
[c]I, Immune; S, susceptible.
[d]Infection types are in parentheses.

senting the data and failure to recognize that the IT is not the phenotype of either symbiont but of the aegricorpus. To produce the IT 0, a particular genotype must be present in both organisms, whereas IT 4 is the result of the alternate genotype in at least one of the organisms. To avoid the potential confusion of the I–S symbolization, the actual phenotypes (ITs) will be used hereafter.

The two studies just reported showed that there were two genes segregating independently both in the F_2 population of the cross of Ottawa 770B and Bombay flax and of the cross of cultures of races 22 and 24 of *M. lini*. Actually, the two studies were part of a single experiment in which the same F_2 populations were used. Ottawa 770B, Bombay, and their F_2 were inoculated with the cultures of race 22, race 24, and their F_2. Thus the two sets of data can be combined. Table VII is made up of the data much as presented by Flor (1959). This demonstrates that the two genes in each of the organisms were inherited independently. Table VIII presents the same data in another manner based on what has been learned since Flor published the data in 1946 and 1947.

Table VIII

Infection Types Observed by Flor for All Possible Genotype Combinations of Two Independent Genes for Reaction (*L* and *N*) and for Pathogenicity (*Al* and *An*) in *Melampsora lini*

Host genotypes	Ratios	Pathogen genotypes			
		*Al_An_*	*Al_anan*	*alalAn_*	*alalanan*
		9	3	3	1
*L_N_*	9	0	0	0	4
L_nn	3	0	0	4	4
*llN_*	3	0	4	0	4
llnn	1	4	4	4	4

It is clear in Table VIII that IT 0 developed only when either or both *Al_*/*L_* or *An_*/*N_* came together, but in no other combinations. *Al_* and *L_* corresponded, and *An_* and *N_* corresponded; thus there are two sets of corresponding gene pairs. This correspondence of dominant genes giving low-infection type was the origin of the expression "gene-for-gene." Later, the expression came to refer to the gene pairs. It is important to understand that the phenotype (IT) is not a genetic character of the host or the pathogen, but is the result of the genotype of both host and pathogen.

From the accumulation of data such as those in Tables VII and VIII

Table VII

Relationship between the Segregation of the F_2 of the Cross of Ottawa 770B and Bombay Flax Cultivars, and of Cultures of Race 22 and 24 of *Melampsora lini*[a]

		Cultures[b]							
						F_2			
Cultivars	Genotypes	Race 24 *AlAlanan*	Race 22 *alalAnAn*			*Al__An__*	*Al__anan*	*alalAn__*	*alalanan*
Ottawa 770B	*LLnn*	0(I)	4(S)			0	0	4	4
Bombay	*llNN*	4(S)	0(I)			0	4	0	4
				O[e]		78	23	27	5
					C[c]	9	3	3	1
							p = .5–.7		
	L__N__	0	0	110	9				
	L__nn	0	4	32	3				
F_2	*llN__*	4	0	43	3				
	llnn	4	4	9	1				
				p = .3–.5					

[a]Data from Tables I–VI combined. Adapted from Flor (1959).
[b]0 and 4 are ITs; I, immune; S, susceptible.
[c]O, Observed plants and cultures; C, calculated ratios.

came the concept of gene-for-gene. Based on these data there emerged several general principles concerning the genetics of symbiosis.

1. The phenotype (IT) is of the aegricorpus, not of host or pathogen; but the genotypes are of the symbionts.
2. The difference between a gene and the organism that has the gene became clear from Flor's work. While a cultivar or culture may have genes for "resistance" and "avirulence," it may be "susceptible" or "virulent," respectively. Thus we are alerted to be careful in how we use such terms as resistance, susceptibility, avirulence, and virulence.
3. In Flor's work, although he discussed race 22, race 24, and so on, he was very careful always to use the same culture of these races and understood why it was important. A race is an unofficial taxon and often is made up of many genotypes. In genetic studies the pathogen unit is the culture, not the race.

Present concepts concerning interorganismal genetics will certainly become modified with time and additional principles developed.

III. The Gene-for-Gene Model

A model (Fig. 1) has been developed as a generalization of the gene-for-gene concept. [This model is sometimes erronelusly referred to as the "quadratic check"; however, this term was originally published by Rowell *et al.* (1963) as the name for a suggested experimental design for biochemical studies.] The symbols *P* and *H* are assigned to the gene pair in pathogen and host, respectively. The model is derived from the phenotypes of the nine possible combinations of the homo- and heterozygotes of the two symbionts as follows:

	PP	*Pp*	*pp*
HH	0	0	4
Hh	0	0	4
hh	4	4	4

This is then reduced to the idealized gene-for-gene model (Fig. 1). The combination of genotypes *PP/HH, PP/Hh, PpHH,* and *PpHh* all give IT 0 and are grouped in the upper left-hand corner of the model; *pp/HH* and *pp/Hh* give IT 4 and are grouped in the upper right-hand corner; *PP/hh* and *Pp/hh* give IT 4 and are grouped in the lower left-hand corner; and *pp/hh,* which also gives IT 4, is placed in the lower right-

Host genotype	Pathogen genotype P_	Pathogen genotype pp
H_	0	4
hh	4	4

Fig. 1. The idealized gene-for-gene model for a single set of corresponding gene pairs for the cereal rusts.

hand corner. Thus the four-way model deals with nine genotype combinations and, in addition, represents a genetic system more complex than it appears. For clarification, the model requires considerable explanation, definition of several new terms, and introduction of a set of symbols.

IV. Categories of Genetic Interaction that Control Disease Development

A. DISEASE VERSUS AEGRICORPUS

Traditionally, "disease" is defined as a process and/or a condition of the host and may include the idea of cause. In actual usage the disease and pathogen are often considered synonymous. The result is that "pests" include weeds, insects, and diseases instead of weeds, insects, and pathogens. A third concept of disease is found in models such as Fig. 2A where disease results from the interaction of pathogen–host–environment. These variable concepts of disease make the word too imprecise to be useful in interorganismal genetics.

The "aegricorpus" results from the interactions among genes in

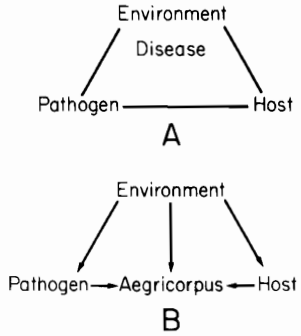

Fig. 2. Comparison of the disease (A) and aegricorpus (B) models.

pathogen and host. Environment, which includes the genetic background of each symbiont as well as external factors, may affect the interaction by acting on the pathogen, host, and aegricorpus independently or in combination (Fig. 2B). We know, for example, that temperature has a large effect on the phenotype of the *Sr6* CGP for low IT, resulting in variation from IT 0;1 at 20°C to IT 4 at 24°C. We do not know, however, if the effect is on the pathogen, host, aegricorpus, or combinations of them. Because environment does have an effect on the final phenotypic expression of CGPs, it will be considered a constant in the following discussion.

B. CATEGORIES OF GENETIC INTERACTIONS

Four categories (Fig. 3) of genetic interactions may occur in symbioses. Two of these occur at the intraorganismal level: category I involves the interaction between alleles at a single locus in a single organism, which results in dominance and recessiveness; category II involves the interaction among the genotypes at two or more loci in a single organism, which results in epistasis in all its forms. It will be assumed that the reader is familiar with these kinds of interactions. In interorganismal genetics, two parallel categories of genetic interaction have been shown but at a different level of biological activity. Category III is the interaction within one set of CGP as seen in the idealized

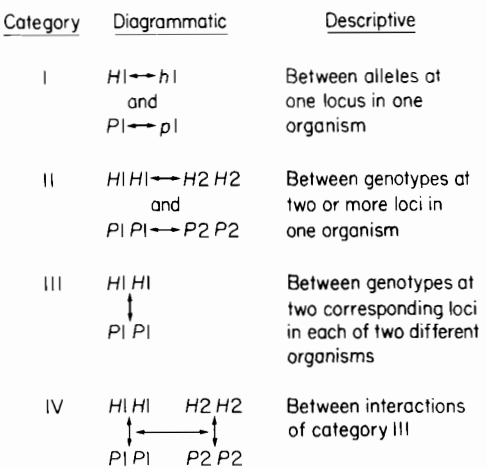

Fig. 3. Categories of genetic interaction that may be found in a host–pathogen association. *H*, *h*, *P*, and *p* indicate alleles for reaction of the host and pathogenicity of the pathogen, respectively. Numbers indicate loci. Adapted from Loegering and Powers (1962).

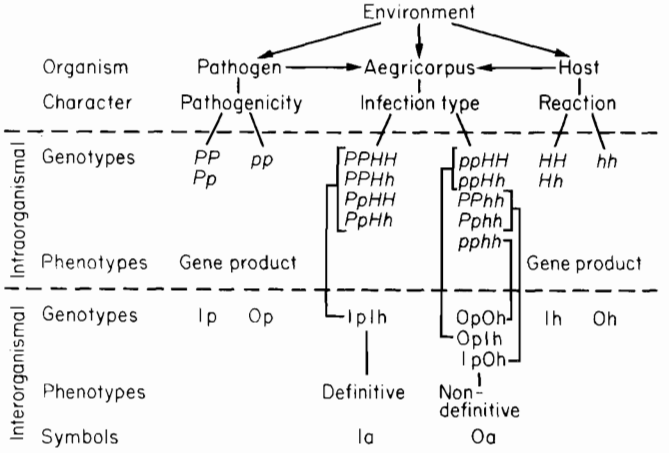

Fig. 4. The category III interaction of interorganismal genetics representing a set of corresponding gene pairs typical of the cereal rusts.

gene-for-gene model (Fig. 1). The pathogen and host genotypes are parallel to the dominant and recessive alleles of Category I. The category IV interaction is among two or more sets of CGP. Each set of CGP is parallel to the loci in category II interactions.

1. The Category III Genetic Interaction

Figure 4 is an expanded version of the gene-for-gene model (Fig. 1) and shows the complexity of the category III interaction. As presented, it represents what has been found for most, but not all, known CGP in the cereal rusts, but it can be adapted to all symbiotic associations.

a. The Organisms. The pathogen and host together produce the aegricorpus, and all three are organisms (Fig. 4). Certainly, the aegricorpus is not an organism in the same sense as the pathogen and host but exists at a different level of biological activity. We recognize the various cereal rusts macroscopically not only by signs (color of the massed urediospores) and symptoms (chlorosis and/or necrosis), but also by the shape and location of the pustules, which are neither signs or symptoms. Thus the aegricorpus has an identity of its own, is organized, and is living (sometimes the life span is very short). It is in this sense that the aegricorpus is considered an organism.

b. The Characters. A genetic character deals with the variations in phenotype resulting from the presence of more than one allele at a locus. For example, seed color in wheat is a character that is variable in

its expression as a result of various combinations of alleles at three loci. In the cereal rusts the characters include pathogenicity of the pathogen, reaction of the host, and the IT of the aegricorpus (Fig. 4). None of these indicates what the phenotype is. Pathogenicity is used in the sense that Nelson *et al.* (1970) defined it, "as the ability of an intity to incite disease on given members of a host species." Reaction is a poor term, because it suggests that the host reacts and the pathogen attacks. This is a decidedly anthropocentric point of view and is not what the word is meant to convey, but is used with respect to the host in the same way as pathogenicity is considered a character of the pathogen. Infection type is used in its traditional sense. In the cereal rusts ITs are generally identified by codes for descriptions of their appearance. In many other diseases such codes are not available; only codes for the amount and/or incidence of disease are available. It is important that we do not use the latter codes in the same way we use infection type in studies of interorganismal genetics.

c. Intraorganismal Genotypes. The intraorganismal genotypes of the pathogen and host are taken from Fig. 1. The genotypes of the aegricorpus are the nine possible combinations of the homo- and heterozygous genotypes of pathogen and host (Fig. 4).

d. Intraorganismal Phenotypes. As pointed out previously, only the aegricorpus has a phenotype as measured by infection type. The pathogen and host have phenotypes (Fig. 4) measured only by specific gene products (or elaborations of them) or absence of such products (Loegering and Sears, 1981). It is important to distinguish these two concepts, because biochemical research is often based on IT as a measure of one of the organisms in the symbiosis. Furthermore, biochemical characteristics are ascribed to that organism, when in actuality the IT is the result of the biochemistry of both pathogen and host and the biochemical interactions taking place in the aegricorpus. The aegricorpus itself has no genetic control of the latter, even though substances might be isolated from the association that do not occur in either host or pathogen. The host and pathogen, however, do not have phenotypes as measured by IT.

e. Interorganismal Genotypes. As the shift is made from intra- to interorganismal genetics in Fig. 4, a new set of symbols and terminology is presented. The need for these will become apparent in discussion of the category IV interaction, of postulating pathogen and host genotypes, and of general resistance. Although there is a similarity

between the concepts of category I and III genetic interactions, they are not the same. In Fig. 1 we see that the four combinations of *P*__/*H*__ give the same result; thus a single symbol can be used for *P*__ and another for *H*__, and two additional symbols are needed for *pp* and *hh*, a total of four symbols that can be combined in four ways. This suggests the use of the "truth" tables of Boolean algebra. Two of the possible 16 truth tables of Boolean algebra fit the category III interaction:

1 + 1 = 1	0 + 0 = 1
1 + 0 = 0	0 + 1 = 0
0 + 1 = 0	1 + 0 = 0
0 + 0 = 0	1 + 1 = 0
A	B

These "truth" tables with their plus and equals signs are used in computer programming and should not be confused with mathematical functions, where 1 + 1 = 2. In Boolean algebra, where 1 + 1 = 1, we mean that item 1 combined with a second item 1 results in a 1 for something else. I chose tabulation A partly at random and partly because it avoids confusion with ideas of "resistance," "virulence," and so on.

In Fig. 4 this "truth" table is put to use. We can have a 1p or 0p genotype in the pathogen culture for a particular CGP and corresponding 1h or 0h genotype in the host cultivar. Note that the 1p represents both *PP* and *Pp*, and the 0p represents *pp*. Likewise, 1h and 0h are used to represent the host genotypes. It is very important to recognize that the 1–0 symbolization represents the genotypes of the two symbionts. These symbols are *not* the intraorganismal genotypes. Thus in using the 1–0 symbolization we do not need to know in a diploid whether the symbols represent the homozygous dominant, the heterozygote, or the homozygous recessive.

f. Interorganismal Phenotypes. At the interorganismal level the pathogen and host do not have phenotypes. The phenotype of the aegricorpus is either *definitive* or *nondefinitive* (Fig. 4). The use of "definitive" was suggested by Wheeler (1975). The definitive phenotype results when both symbionts have the 1 genotype and the nondefinitive when either or both symbionts have the 0 genotype. These phenotypes are symbolized as 1a (definitive) or 0a (nondefinitive), where a is aegricorpus. With these symbols we can change Fig. 1 to represent any category III genetic interaction for any symbiotic association (Fig. 5). (The p and h symbols can be changed to fit the particular association being studied.)

This symbolization adequately symbolizes the cereal rusts because, in so far as we know, 1a is always a "low" IT. It also avoids considera-

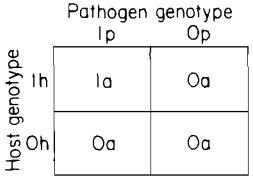

Fig. 5. A generalized model for a single set of corresponding gene pairs in any symbiotic association.

tion of dominance and recessiveness in the associated organisms. If a cultivar is inoculated with a culture and the result is "low" IT, we know that the genotype for at least one CGP is 1p/1h, although we have no information as to dominance and recessiveness in the individual symbionts. If the result is "high" IT, all we know is that at least one of the symbionts does not have the definitive genotype for each of the CGP. If information on the dominance or recessiveness of the 1p or 1h genotype is desired, then it becomes necessary to do an intraorganismal genetic study. One organism must be held constant while the other is crossed to a 0 genotype and the segregating population studied by inoculation of or with the constant member of the association.

g. The 1–0 *Symbolization Can Be Used for All Symbiosis.* The 1–0 symbols can be utilized for representation of the genetics of all symbiotic associations and is used to represent alleles and genotypes at the intraorganismal level as well as genotypes and phenotypes at the interorganismal level. At the intraorganismal level *P* and *H* are 1p and 1h alleles, whereas *p* and *h* are 0p and 0h alleles. They are shown in Fig. 4 as dominant and recessive by conventional symbolization; that is, capital letters indicate dominant alleles. At the interorganismal level, the 1p genotype represents the dominant genotypes of the pathogen, *PP* and *Pp*, whereas the 0p genotype represents the recessive *pp* genotype. Likewise, the 1h genotype represents the dominant genotypes of the host, *HH* and *Hh*, whereas the 0h genotype represents the recessive *hh* genotype. This representation is typical for nearly all CGP in the cereal rusts. However, there are exceptions, as for example the *Sr17* host gene, where the 1h allele is recessive; thus 1h*sr17* represents the recessive host genotype, and the dominant genotypes are represented by 0h*sr17*. Likewise, the 1p allele at the *Vwb* locus in *M. lini* is recessive, and the homozygous recessive is the 1p genotype and the dominant genotypes are represented by 0p. The 1–0 representation therefore is not dependent on the dominance or recessiveness of the category I interaction of Mendelian genetics.

In Fig. 4 both symbionts are shown as diploid. In the cereal rusts this is usual, although haploid basidiospores produce haploid pycnia on the alternate host. In the powdery mildews of cereals, the fungus growing in association with the cereal host is haploid. Here the 1p genotype represents one allele and 0p the other allele. The idea that 1 and 0 represent genotypes but are not the genotypes can be extended to include heterokaryotic genotypes and even cytoplasmic factors.

In Fig. 4 there is no indication what the definitive and nondefinitive phenotypes are. Because this treatise deals with the cereal rust, it perhaps is assumed that the nondefinitive phenotype (0a) is IT 4 ("susceptibility"), while the definitive phenotype (1a) is less than IT 4 ("resistance"). At present we know of no exceptions to this in the cereal rusts; however, in diseases such as Victoria blight of oats the reverse is true, thus definitive and nondefinitive should not and cannot be used as synonyms for "resistance" and "susceptibility." The 1a phenotype results only when the 1p and 1h genotypes occur in the two associated symbionts and in no way indicate what the phenotype is. In summary, the 1–0 symbolization of interorganismal genetics is a representation of alleles, genotypes, and phenotypes without indicating dominance at the intraorganismal level or the appearance of the resulting phenotype of the aegricorpus. The representation is useful for postulating genotypes from IT data (see Section V,B), to design experiments in biochemistry and genetics, and in modifying our philosophical approach to the study of plant pathology.

2. *The Category IV Genetic Interaction*

The preceding conceptual discussion of the category III genetic interactions and the symbolization introduced permit discussion of the category IV interactions, which are what the geneticist and plant breeder must always deal with. Each set of CGP occur at multiple loci in the genomes of the symbionts. These genomes have two kinds of genes: (1) intraorganismal, which control the characters of each symbiont such as awns, type of panicle, color of spores, and cell wall constituents, and (2) interorganismal, which control the symbiosis. It is highly probable that some genes in one or both symbionts have pleotropic effects and are involved at both the intra- and interorganismal levels.

Category IV involves the interactions between and among CGP. For example, in stem rust of wheat, at least 33 loci for reaction have been clearly identified in the host with seven additional 1h alleles at two of these loci. Because allelism rarely occurs in *Puccinia graminis tritici*, there are as many as 40 loci in the pathogen corresponding to the loci

and alleles in the host. It has been suggested on the basis of hypothetical genetic studies that there are two to three times this number of loci in host and pathogen—a highly polygenic system that can be illustrated using four sets of CGP. Each set of CGP is identified by a number following the interorganismal symbol to represent the respective loci (e.g., 1p1/0h1, 0p2/1h2). Because each set of CGP can occur in four combinations, four sets can occur as 16 different formulas. Four of these are illustrated and discussed next. In these illustrations it is assumed that IT 4 is the nondefinitive (0a) phenotype, whereas any lower IT is a definitive (1a) phenotype.

Pathogen genotype	$\frac{1p1\ 0p2\ 1p3\ 0p4}{0h1\ 1h2\ 0h3\ 0h4}$	= IT 4 = 0a (1)
Host genotype		
Category III phenotypes	IT4 IT4 IT4 IT4	
Category III phenotype symbol	0a1 0a2 0a3 0a4	

In Eq. (1) each of the CGP has a genotype that gives the 0a phenotype even though the pathogen has two definitive genotypes (1p1 and 1p3), whereas their corresponding host genotypes are nondefinitive (0h1 and 0h3). Likewise the host has one definitive genotype (1h2), but the pathogen has the corresponding nondefinitive genotype (0p2). The total result of the genotypes of the four CGP is IT 4 = 0a. Note that the presence of definitive genotypes in one or the other organism does not affect the Category IV phenotype.

Pathogen genotype	$\frac{1p1\ 0p2\ 1p3\ 0p4}{1h1\ 1h2\ 0h3\ 0h4}$	= IT 2 = 1a1 (2)
Host genotype		
Category III phenotype	IT2 IT4 IT4 IT4	
Category III phenotype symbol	1a1 0a2 0a3 0a4	

In Eq. (2) one change has been made from Eq. (1). The genotype for locus 1 in the host has been changed from 0h1 to 1h1. The definitive 1pl/1hl gives IT 2, whereas the other three CGP again result in IT 4. Obviously, both phenotypes cannot be expressed. In all work with cereal rusts done to date the definitive phenotype—IT 2 in Eq. (2)—is expressed and thus is "epistatic" to the nondefinitive category III phenotypes.

Pathogen genotype	$\frac{1p1\ 1p2\ 1p3\ 0p4}{1h1\ 1h2\ 0h3\ 0h4}$	= IT 1 = 1a2 (3)
Host genotype		
Category III phenotype	IT2 IT1 IT4 IT4	
Category III phenotype symbol	1a1 1a2 0a3 0a4	

In Eq. (3) one change has been made from Eq. (2). The genotype for locus 2 in the pathogen has been changed from 0p2 to 1p2. the defini-

tive 1p2/1h2 gives IT 1, whereas the other three CGP have the same phenotypes as in Eq. (2). It is obvious that all three phenotypes cannot be expressed. What has been found is that the lowest definitive phenotype (IT 1) in Eq. (3) is usually expressed.

Pathogen genotype	$\frac{1p1\ 1p2\ 1p3\ 0p4}{1h1\ 1h2\ 1h3\ 0h4}$	$= \text{IT } 0_j = 1a2+3$ (4)
Host genotype		
Category III phenotype	IT2 IT1 IT3 IT4	
Category III phenotype symbol	1a1 1a2 1a3 0a4	

In Eq. (4) one change has been made from Eq. (3). The genotype for locus 3 in the host has been changed from 0h3 to 1h3. This illustrates complementary interaction between two sets of CGP. In Eq. (4) both the host and pathogen have definitive genotypes at loci 2 and 3, and there is an interaction that results in IT 0_j instead of IT 1 as would be expected on the basis of Eq. (3). Because 0_j is the lowest phenotype, it is expressed. If we change Eq. (4) to

$$\frac{0p1\ 1p2\ 1p3\ 0p4}{0p1\ 1h2\ 1h3\ 0h4} = \text{IT } 0_j = 1a2+3$$

we may assume the interaction involves only CGP *2* and *3*. If additional studies are made and it is found that

$$\frac{1p2\ 1p3}{0h2\ 1h3} = \text{IT } 3 \text{ and } \frac{1p2\ 1p3}{1h2\ 0p3} = \text{IT } 1$$

then the interaction is not in the pathogen, and if

$$\frac{0p2\ 1p3}{1h2\ 1h3} = \text{IT } 3 \text{ and } \frac{1p2\ 0p3}{1h2\ 1h3} = \text{IT } 1$$

then the interaction is not in the host.

Although this does not prove that the interaction is occurring in the aegricorpus, this hypothesis should be considered seriously. There are numerous reports of interaction between genes for low reaction and a few for low pathogenicity, but none of these has been adequately demonstrated because the necessary complex studies have not been made. More likely, the reported interactions are not category II (intraorganismal) but category IV interactions (interorganismal).

The concept that there are interactions occurring between and among CGP is indispensable when considering the cereal rust diseases, because "gene-for-gene" is a polygenic system, not an oligogenic one as commonly stated in the literature. The latter idea has led to much of the misunderstanding concerning the biology of the pathogen–host

system. In experimental designs and in discussion of the gene-for-gene concept, the assumption that there is only one gene for "resistance" or "virulence" in the association being studied is nearly always false. Person (1959) demonstrated this in his model using five CGP. It is time that biologists recognize that in the cereal rusts (and many other diseases) we do not deal with an oligogenic system, even though it is easy to follow one gene at a time. It is true that a cultivar may not be damaged because it has a 1h genotype at one locus and the pathogen population is homogeneous for a corresponding 1p genotype. This is a pragmatic and useful point of view, but it fails miserably when used to develop biological hypotheses to be tested.

C. INTERORGANISMAL GENETIC INTERACTIONS ARE COMPLEX

The complexities of interorganismal genetics are illustrated in Fig. 6 using two CGP. In the category I interaction there may be dominance, incomplete dominance, or recessiveness. Although there are no documented instances of category II interactions, this does not mean they do not occur, and possibly reports of inhibitors are of this category. The category III interaction is relatively simple, because genotypes are represented without specifying what is dominant or if there is category II epistasis. The category IV interaction, which is characteristic of the cereal rusts, is extremely complex, involving "epistasis" of several kinds and possibly at two levels. Yet it is the category IV interaction that we see expressed in the cereal rusts.

D. INCOMPLETE DOMINANCE

The occurrence of incomplete dominance in category I causes additional complexities and may lead to misinterpretations of genetic data. The data of Samborski (1963) illustrate this. He worked with CGP *lr9*.

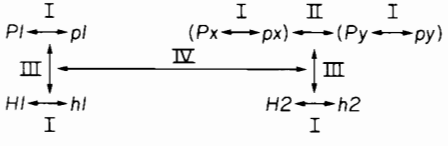

Fig. 6. The genetic interactions of symbiosis. *P*, *p*, and *H*, *h* are generalized gene symbols for pathogen and host, respectively. *1*, *2*, *x*, and *y* are loci, and I–IV are the categories of genetic interaction.

The 1h*lr9* allele was transferred from *Aegilops umbellulata* Zhuk. to hexaploid wheat by Sears (1956), and the breeding line was named "Transfer." Over a period of several years this cultivar was essentially immune to all cultures of *Puccinia recondita* Rob. ex. Desm.; however, Samborski (1963) observed IT 1+ on "Transfer" in plots at Winnipeg. A culture was established on the cultivar "Thatcher," which lacks the 1h*lr9* genotype, and telia were produced. The S_1 gave three kinds of cultures, which on "Transfer" gave ITs 0;, 1+, and 4, respectively, indicating that the original culture was heterozygous for p*lr9*, and incomplete dominance occurred. Samborski retained cultures that appeared to be homozygous for the partially dominant 1p*lr9* allele and the recessive 0p*lr9* allele. He next crossed "Transfer" and "Thatcher" and produced an adequate number of F_1 (heterozygous) seeds. He then inoculated the two cultivars and their F_1 with the three cultures of *P. recondita* and obtained the following results (Samborski, 1963):

Host genotype	Pathogen genotype					
	PP		*Pp*		*pp*	
RR (Transfer)	0	(R)	1+	(R)	4	(S)
Rr F_1	0	(R)	3	(S)	4	(S)
rr (Thatcher)	4	(S)	4	(S)	4	(S)

With the homozygous 1p*lr9* culture he found that the 1h*lr9* allele was clearly dominant and resulted in IT 0 in both the homozygous and heterozygous condition. With the heterozygous culture he obtained IT 1+ on "Transfer," IT 3 on the F_1, and IT 4 on "Thatcher." This indicated that the 1h*lr9* allele was incompletely dominant or, if we use the concepts of race identification in which IT 3 and 4 = susceptibility, then the 1h*lr9* allele was recessive. This latter interpretation is shown in parentheses above using R and S as phenotypic symbols. These data suggest a "reversal of dominance," and several studies with other rusts have been interpreted in this manner. Very likely, the true interpretation is that two cultures, one homozygous and the other heterozygous, were used in those studies.

E. GENE SYMBOLS

The intraorganismal gene symbols for the cereal rusts have been assigned by three methods. In the wheat rusts the common name of the disease has been the basis; for example, *sr*, stem rust; *lr*, leaf rust;

and *yr*, yellow rust (stripe rust). In the oat rusts the Latin name of the pathogen has been the basis, for example, *pc* = *Puccinia coronata*. Flor assigned letters to the loci in the host and then used the allele designation to indicate the corresponding gene in the pathogen. Uniformity would be desirable but is not necessary; however, the use of a symbol based on the disease name is more logical, because both the pathogen and host produce the disease and the same designation can be used for both species.

McIntosh (1973) has recommended that for the wheat rusts the convention of upper and lower case initial letters should not be used to indicate dominance and recessiveness. This will avoid confusion that can arise from several sources. Not all 1h (or 1p) genotypes represent dominant genotypes; for example, h*sr11* is dominant for the 1h genotype, whereas h*sr17* is dominant for the 0h genotype. Perhaps more important is that dominance and recessiveness become meaningless in cases where allelism occurs. If a cultivar homozygous for 1h*sr9a* is crossed with one homozygous for 1h*sr9b* and the segregating population inoculated with a culture of the genotype 1p*sr9a*0p*sr9b*, the *sr9a* allele is dominant and the *sr9b* allele recessive. If, however, the segregating population is inoculated with a culture of the genotype 0p*sr9a*1p*sr9b* the reverse is true. Thus the apparent dominance and recessiveness exhibited in the host is due not to the host alleles, but to the genotype of the pathogen. Furthermore, there are no *proven* cases of alleles for the 0h genotype. This first became apparent from the work of Kerr (1960), who showed that the recessive 0h allele in Bison flax at the *l* locus was in reality a dominant 1h allele (*L9*) when certain Australian *M. lini* cultures were used to inoculate Bison. Loegering and Sears (1981) clearly demonstrated that absence of a locus resulted in the 0h genotype for the *sr6*, *sr8*, *sr9a*, and *sr11* loci in wheat. In addition, they showed that even when the 1h alleles were present, the 1a phenotype could be changed to 0a by changing the pathogen genotype or in the case of *sr6* by changing the temperature.

When conducting studies at the intraorganismal level, the conventional symbolization may be used by specifying what the definitive and nondefinitive phenotypes are and then holding one member of the symbiosis contant.

V. Applications of Interorganismal Genetics

Over the past 40 years concepts of interorganismal genetics have evolved as a result of the discovery of the gene-for-gene relationship.

As a consequence, there has been a change in how we think of the pathogen–host association. We can view the aegricorpus as a natural and normal symbiotic association instead of an unnatural and abnormal disease. Such a viewpoint should and can change our view of plant pathology from its present pragmatism to a more fundamental understanding of the biology of the pathogen–host symbiosis.

There are many possible applications of interorganismal genetics with respect to cereal rusts. Only two will be discussed: (1) deriving hypothetical genotypes of pathogen and host from IT data and (2) developing a theoretical genetic basis for general resistance.

A. HYPOTHETICAL GENOTYPES BASED ON IT DATA

Computers are useful tools for development of hypotheses regarding the pathogenicity genotypes of pathogen cultures and reaction genotypes of host cultivars without making crosses. Such hypotheses are useful in developing experimental designs for basic studies in genetics and biochemistry, and as a basis for breeding programs. In the past breeders and pathologists have intuitively used the principles when they have transferred a "new" gene for "resistance" to new commercial cultivars. Unfortunately, their viewpoint that the resulting resistance was determined by the host gene alone has been adopted by geneticists, pathologists, physiologists, and biochemists. As a result a basic understanding of the pathogen–host association has eluded us.

To develop hypothetical genotypes of pathogen and host from IT data, we use the principles of interorganismal genetics. In the cereal rusts we know that 1p/1h = 1a. This category III formula can be reversed, and we find that 1a = 1p/1h. Thus if we inoculate a cultivar with a culture and obtain a 1a phenotype, we know that in at least one set of CGP the pathogen has a 1p genotype and the host the corresponding 1h genotype. On the other hand, if we find a 0a phenotype we know that if the pathogen has any 1p genotypes, the host has the corresponding 0h genotypes, or if the host has 1h genotypes, then the pathogen has the corresponding 0p genotypes. Thus we would not know the genetic reason for the 0a phenotype. In day-to-day laboratory investigation we actually work in this manner. For example, when we inoculate two cultivars with two cultures the results are obtained as infection types:

		Culture	
		X	Y
Cultivar	A	0;	4
	B	4	4

Initially, we knew nothing about the genotypes of the cultures or cultivars, but the IT 0; tells us that for one set of CGP, culture X has the 1p genotype and cultivar A the corresponding 1h genotype. Thus we can insert this information into the box.

			Culture X 1p	Culture Y
Cultivar	A	1h	0;	4
	B		4	4

We now know the genotype for culture X and cultivar A with regard to one set of CGP. If cultivar A has the *1h* genotype but when inoculated with culture Y we obtain the 0a phenotype, we know the culture must have the corresponding *0p* genotype. The same reasoning can be used with culture X and cultivar B. Thus we can fill in the genotypes as follows:

			Culture X 1p	Culture Y 0p
Cultivar	A	1h	0;	4
	B	0h	4	4

To use this in computerized studies we need to put the diagram in its final form as follows:

			Culture X 1p	Culture Y 0p
Cultivar	A	1h	1a	0a
	B	0h	0a	0a

In the explanation of Flor's work we started with known genotypes and derived phenotypes. Here we start with known phenotypes and derived genotypes. To understand that this shift has been made is fundamental to deriving pathogen and host genotypes from IT data.

There are seven basic patterns (Fig. 7) of results from inoculating two cultivars with two cultures, although there may be as many as four variations in each pattern (Loegering and Burton, 1974). For example, in Fig. 7A, the 1a could be found in any one of the four corners of the diagram. This results in a change in the hypothetical genotypes for the

cultures and cultivars but does not change the principle. The three additional variations of Fig. 7A are as follows:

	1p	0p
0h	0a	0a
1h	1a	0a

	0p	1p
0h	0a	0a
1h	0a	1a

	0p	1p
1h	0a	1a
0h	0a	0a

In Fig. 7B using the same logic we can identify two sets of CGP. Of the seven basic patterns only Fig. 7A and B permit assigning genotypes to both cultures and both cultivars. The other five patterns leave at least one of the genotypes as an unknown. Figure 7C and D leave the genotype for one culture and cultivar, respectively, as unknown. Because there is at least one set of CGP, we arbitrarily assign 1p/1h to one of the 1a phenotypes. The second 1a phenotype could be due to the

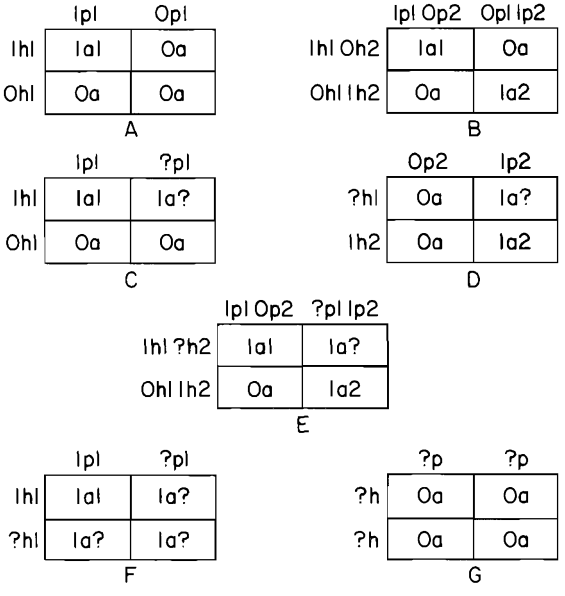

Fig. 7. The seven basic "box arrangements" resulting from inoculating two host cultivars (sides) with two pathogen cultures (tops). 1p, 0p, 1h, and 0h represent definitive (*1*) and nondefinitive (*0*) genotypes of pathogen (p) and host (h); 1a and 0a represent the definitive (1) and nondefinitive (0) phenotypes of the aegricorpus (a); 1 and 2 following 1a or 0a represent corresponding gene pairs and the definitive phenotype. The question mark has two meanings: 1a? means that a 1a phenotype was observed, but it is uncertain what the 1p/1h genotype is; ?p1 means that it is not certain whether the genotype is 1p1 or 0p1. Adapted from Loegering and Burton (1974).

same CGP or due to a second set. Thus one 1a phenotype is of unknown genotype, and one culture in Fig. 7C and one cultivar in Fig. 7D are of unknown genotype. Figure 7E is made up of Fig. 7B, C, and D. It is like Fig. 7B because there are two 1a phenotypes on a diagonal opposite a 0a phenotype, demonstrating that two CGP are present. This diagonal is extremely useful in examining large data sets visually. The 1a phenotype in the corner opposite the 0a phenotype makes Fig. 7E a combination of Fig. 7C and D, and the same unknown genotypes of these two configurations are present. Figure 7F and G are the last two configurations and give little or no useful information regarding genotypes but are important for this reason.

These seven basic boxes are used in computer analysis of a data set made up of the infection types from inoculation of a group of cultivars with a group of cultures. Three kinds of information can be obtained: (1) grouping cultivars and/or cultures that in a data set have the same patterns, (2) comparing cultures and/or cultivars of unknown genotype with ones of known genotype, and (3) postulating genotypes of the cultivars and/or cultures in the data set. The methods (Loegering *et al.*, 1971; Loegering and Burton, 1974) of doing these operations will not be detailed here. There is some variation in the procedures as developed by these authors and that developed by Browder and Eversmeyer (1980) and as applied by Roelfs *et al.* (1982). This variation is based on the degree of confidence the respective workers have in their ability to distinguish variations in the 1a phenotypes; however, the basic principles are the same.

In the method of Loegering *et al.* (1971; Loegering and Burton, 1974), all data are classified as 1a or 0a (L and H in their original publications); thus there are only two classes, whereas Browder and Eversmeyer (1980) and Roelfs *et al.* (1982) use each different IT, and whenever a diagonal of two ITs occurs opposite a higher IT, those on the diagnoal indicate two different CGP. The valid criticism made of the Browder and Eversmeyer method by Knott and Johnson (1981), that the final results of the computer analysis are dependent on the initial arrangement of the data set, is also true for the method of Loegering *et al.* (1971). It must be remembered, however, that the results obtained by either method only develop hypotheses to be tested and not proof.

B. GENERAL RESISTANCE

For centuries farmers observed that some individual plants were less damaged by diseases than other individuals. These less damaged plants

were propagated, and sometimes the farmers were successful in avoiding some of the damage from certain diseases. Evidence for this is found in the winter wheats grown in the south-central United States. Even in severe outbreaks of leaf rust, many of the wheats return an acceptable yield. These wheats originated from collections made in farmers' fields in southwest Asia where they had been grown for centuries.

In 1905, Biffin found that resistance to *Puccinia striiformis* West was controlled by a single gene. The "monogene" concept of resistance in plants to diseases was born, and over half a century of breeding for disease resistance was based on the lodestone of "monogene" resistance. Then, suddenly, what farmers had known for centuries was rediscovered but was now called "horizontal resistance." Attempts to define horizontal resistance have not been successful, and many other names have been applied to the phenomenon. Of these, "general resistance," as defined by Caldwell (1968) as being "durable," has some validity and is used in this chapter. Considering our present understanding of the phenomenon, the definition used by Loegering (1972), "that 'non-specificity' can only be defined as a host–pathogen relationship for which specificity has not been demonstrated," is useful.

At present it seems likely that much of what we call general resistance is due to specificity. The first indication that this might be true was published by Slezinsky and Ellingboe (1969). They worked with powdery mildew of wheat and studied the transfer of ^{35}S from the host to the pathogen using the quadratic-check experimental design. The relative amount of ^{35}S per conidium was low for 1p/1h, high for 0p/0h and 1p/0h, but intermediate for 0p/1h, indicating that the 0p/1h genotype was physiologically different from the 1p/0h and 0p/0h genotypes, even though the phenotypes (ITs) did not appear to differ. This raised the question whether or not careful measurements would reveal differences in disease development. That such differences do occur was shown by Martin and Ellingboe (1976) using powdery mildew of wheat. They found that 0p*pm4*/1h*pm4* compared with 1p*pm4*/0h*pm4* and 0p*pm4*/0h*pm4* showed reduced infection efficiency and longer generation time, even though the final phenotype was IT 4.

In stem rust of wheat Rowell (1981) showed for the *Srtt1* CGP that 0p/1h had a strong effect on the 0a phenotype. Skovmand *et al.* (1978), however, could not demonstrate differences between 0p/1h and 0p/0h for the *Sr5*, *Sr6*, *Sr7b*, and *Sr11* CGP. Thus it appears that for some CGP the 0p/1h is not equal to the 1p/0h and 0p/0h genotypes, whereas for other CGP they are.

Applying interorganismal genetics to these observations, it can be

shown how general resistance theoretically could be due to specificity using the following assumed information for three CGP designated as X, Y, and Z. The 1a phenotype for each of the three CGP is IT 0; and for the 0a phenotypes, IT 4. We then measure the latent period, spore production, and infection efficiency of the nine possible 0a phenotypes. By comparing these we find that 0p/1h for *Srx* has a latent period of one extra day, for *Sry* 20% fewer spores, and for *Srz* an infection efficiency of 75%. Let us also assume that each of these reduces the rate of increase by 3%. Such a decrease would be difficult to determine by simple observation, but if all three occur together as

$$\frac{0px\ 0py\ 0pz}{1hx\ 1hy\ 1hz} = 0a$$

the additive effects of a category IV interaction perhaps would result in a reduction in rate of increase greater than 3%. This would still be difficult to measure under field conditions. Very likely, however, the effect would be more than additive, in that it would take longer to produce fewer spores and fewer spores would produce infections. Thus reduction in rate of increase might be great enough to be observed in the field and might be mistaken for "general resistance."

There are two features of such a system that can explain the ideas of durability and polygenic inheritance, which are often used in defining the phenomenon of general resistance. Durability is built into the system, because if the pathogen acquires the 1p genotype at any of the three loci through hybridization, mutation, or parasexualisim, the result would be IT 0:. Because any of the 1p/1h genotypes would result in this phenotype, a culture with a 1p genotype at one of the loci would not survive, thus the 0p genotype is maintained. In nature a host that changed from 1h to 0h at any of the three loci would have the potential of being more heavily damaged and thus would have a reduced survival capability. Thus there would be a tendency for the 0p/1h system to be self-perpetuating in nature. When using IT as the measure of resistance in a breeding program, one would be apt to lose the host 1h genotype combinations associated with general resistance without being aware of doing so.

The origin of the idea that general resistance is polygenic and a character of the host should be apparent. Crosses of the 0px0py0pz culture with a 1px1py1pz culture and using the 1hx1hy1hz cultivar as a tester would result in segregation based on IT. However, if the cultivar was crossed with a 0hx0hy0hz cultivar and inoculated with the 0px0py0pz culture there would be a range of disease development in terms of rate of increase. The distribution of the segregating progeny

would be dependent on the variation of the hypothetical 3% reduction of each pair of CGP as well as on what effect heterozygosity would have on disease increase.

When general resistance results from the 0p/1h genotype, it differs genetically from the 1p/1h genotype, as demonstrated by Johnson and Taylor (1976), where reduced sporulation was the result of a 1p/1h genotype. The definitive 1p*sr13*/1h*sr13* gives IT 3-, which also results in reduced sporulation. There seems to be no reason that some morphological characters of the host could not result in reduced disease. General resistance due to an accumulation of 0p/1h genotypes in the pathogen–host association and/or to morphological characters of the host would likely be durable, whereas that due to 1p/1h genotypes perhaps would not because a change from 1p to 0p would have survival value for the fungus.

References

Biffin, R. H. (1905). Mendel's laws of inheritance and wheat breeding. *J. Agric. Sci.* **1,** 4–48.

Browder, L. E., and Eversmeyer, M. G. (1980). Sorting of *Puccinia recondita:Triticum* infection-type data sets toward the gene-for-gene model. *Phytopathology* **70,** 666–670.

Caldwell, R. M. (1968). Breeding for general and or specific plant disease resistance. *Proc. Wheat Genet. Symp., 3rd. 1968* pp. 227–238.

Flor, H. H. (1946). Genetics of pathogenicity in *Melampsora lini*. *J. Agric. Res. (Washington, D.C.)* **73,** 335–357.

Flor, H. H. (1947). Inheritance of reaction to rust in flax. *J. Agric. Res. (Washington, D.C.)* **74,** 241–262.

Flor, H. H. (1959). Genetic controls of host-parasite interactions in rust diseases. *In* "Plant Pathology: Problems and Progress, 1908–1957" (C. S. Holton *et al.*, eds.), pp. 137–144. Univ. of Wisconsin Press, Madison.

Flor, H. H. (1971). Current status of the gene-for-gene concept. *Annu. Rev. Phytopathol.* **9,** 275–296.

Johnson, R., and Taylor, A. J. (1976). Spore yield of pathogens in investigations of the race-specificity of host resistance. *Annu. Rev. Phytopathol.* **14,** 97–119.

Kerr, H. B. (1960). The inheritance of resistance of *Linum usitatissimum* to the Australian *Melampsora lini* race complex. *Proc. Linn. Soc. N.S.W.* **85,** 273–321.

Knott, D. R., and Johnson, R. (1981). The sorting and analysis of infection types from *Triticum aestivum/Puccinia recondita* interactions. *Phytopathology* **71,** 1010–1012.

Loegering, W. Q. (1966). The relationship between host and pathogen in stem rust of wheat. *Hereditas, Suppl.* **2,** 167–177.

Loegering, W. Q. (1972). Specificity in plant disease. *In* "Biology of Rust Resistance in Forest Trees," Misc. Publ. 1221, pp. 29–37. Forest Service, Washington, D.C.

Loegering, W. Q., and Burton, C. H. (1974). Computer-generated hypothetical genotypes for reaction and pathogenicity of wheat cultivars and cultures of *Puccinia graminis tritici*. *Phytopathology* **64,** 1380–1384.

Loegering, W. Q., and Powers, H. R., Jr. (1962). Inheritance of pathogenicity in a cross of physiologic races 111 and 36 of *Puccinia graminis* f. sp. *tritici*. *Phytopathology* **52,** 547–554.

Loegering, W. Q., and Sears, E. R. (1981). Genetic control of disease expression in stem rust of wheat. *Phytopathology* **71,** 425–428.

Loegering, W. Q., McIntosh, R. A., and Burton, C. H. (1971). Computer analysis of disease data to derive hypothetical genotypes for reaction of host varieties to pathogens. *Can. J. Geneti. Cytol.* **13,** 742–748.

McIntosh, R. A. (1973). A catalogue of gene symbols for wheat. *Proc. Int. Wheat Genet. Symp. 4th, 1973* pp. 893–937.

Martin, T. J., and Ellingboe, A. H. (1976). Differences between compatible parasite/host genotypes involving the *Pm4* locus of wheat and the corresponding genes in *Erysiphe graminis* f. sp. *tritici*. *Phytopathology* **66,** 1435–1438.

Nelson, R. R., McKenzie, D. R., and Scheifele, G. L. (1970). Interaction of genes for pathogenicity and virulence in *Trichometasphaeria turcica* with different numbers of genes for vertical resistance in *Zea mays*. *Phytopathology* **60,** 1250–1254.

Person, C. (1959). Gene-for-gene relationships in host:parasite systems. *Can. J. Bot.* **37,** 1101–1130.

Roelfs, A. P., Baker, F. D., and McVey, D. V. (1982). An interactive computer-based system for comparing cultures of *Puccinia graminis* and postulating *Sr* genotypes in wheat. *Phytopathology* **72,** 597–600.

Rowell, J. B. (1981). The relationship between slow rusting and a specific resistance gene for wheat stem rust. *Phytopathology* **71,** 1184–1186.

Rowell, J. B., Loegering, W. Q., and Powers, H. R., Jr. (1963). Genetic model for physiologic studies of mechanisms governing development of infection type in wheat stem rust. *Phytopathology* **53,** 932–937.

Samborski, D. J. (1963). A mutation in *Puccinia recondita* Rob. ex. Desm. f. sp. *tricici* to virulence on Transfer, Chinese Spring × *Aegilops umbellulata* Zhuk. Can. J. Bot. **41,** 475–479.

Sears, E. R. (1956). Genetics in plant breeding. *Brookhaven Symp. Biol.* **9,** 1–22.

Skovmand, B., Roelfs, A. P., and Wilcoxon, R. D. (1978). The relationship between slow-rusting and genes specific for stem rust resistance in wheat. *Phytopathology* **68,** 491–499.

Slezinski, R. S., and Ellinboe, A. H. (1969). The effects of various parasite/host genotypes on ^{35}S transfer from wheat to *Erysiphe graminis* f. sp. *tritici*. *Phytopathology* **59,** 1050 (abstr.).

Wheeler, H. (1975). "Plant Pathogenesis." Springer-Verlag, Berlin and New York.