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4. Georg, L. K., W. Kaplan, and L. B. Camp. 1957. American Journal of Veterinary Research 18:798-810.
5. Lomas, H. 1939. British Veterinary Journal. 95:290-292.
6. Ainsworth, G. C. and P.K.C. Austwick. 1955. Veterinary Record 67: 88-97.
7. ———— and ————. c1959. Fungal Diseases of Animals. Commonwealth Agricultural Bureau of Animal Health Review Series No. 6. Great Britain.
8. Kral, F. c1960. Compendium of veterinary dermatology. Chas. Pfizer and Co., Inc. New York.
9. Hindle, E. c1914. Flies in relation to disease. Cambridge University Press. Cambridge, Eng.
10. Scott, J. W. 1922. Wyoming Agriculture Experiment Station Bulletin No. 133.
11. Herms, W. B., and M. T. James. c1961. Medical entomology. 5th ed. The Macmillan Co., New York.
12. Doty, A. C. 1937. Journal of Economic Entomology. 30:367-369.
13. Richard, J. L. 1963. Unpublished M.S. Thesis. Iowa State University.
14. Louria, D. B. and H. G. Brown. 1960. Annals of the New York Academy of Sciences 89 (1):39-46.
15. Wilson, J. W. c1957. Clinical and immunologic aspects of fungus diseases. Charles C. Thomas. Springfield, Ill.

Teaching and Applying the Gene-for-Gene Hypothesis for Interactions in Host:Parasite Systems[1]

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Abstract. A "lock-for-key" method of teaching the gene-for-gene hypothesis for interactions in host:parasite systems, and an example of the application of the hypothesis to an "unknown" disease, stem rust of oats, are presented.

Many plant pathogenic fungi are specific, not only for a given host genus or species, but also for a given agronomic or horticultural variety. In some cases, a disease resistant variety may differ from a susceptible variety by a single gene which conditions disease expression. Teachers of plant pathology are challenged frequently to explain the specificity displayed by such interacting organisms. The gene-for-gene hypothesis provides a basis for understanding the genetic interactions of host and pathogen.

Flor (1) stated: "A simple explanation for the high degree of physiologic specialization of the rust fungi is the hypothesis that during their parallel evolution host and parasite developed complementary genic systems. For each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite. Pustule type, the criterion both of re-

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acion in the host and of pathogenicity in the parasite, is conditioned by complementary genes in the two plants." This hypothesis, developed for the *Linum:Melampsora* system, has since been shown applicable also to the *Solanum:Phytophthora* (2), *Hordeum:Erysiphe* (3), *Triticum:Erysiphe* (4), *Malus:Venturia* (5), and apparently *Avena:Ustilago* (6,7) host:parasite systems. Since these systems involve pathogens representing all 3 major classes of fungi, it appears that the gene-for-gene hypothesis for interactions in highly specific host:parasite systems may be universal. In fact, Person (8) showed that "gene-for-gene relationships are to be expected as a general rule and not as an isolated event."

Obviously, students must understand the rudiments of the gene-for-gene hypothesis if they are to understand the genetic basis of the relationship of host and pathogen. The hypothesis was first advanced for flax and flax rust, and data from this system are well suited to explaining the gene-for-gene relationship. However, I have had difficulty in teaching the flax:flax rust work to even advanced students in plant pathology who have a limited background in genetics. Students first must get past varietal names, race numbers, and genetic symbols to reach the material that so succinctly illustrates the hypothesis. While working with such students there has evolved a system of teaching this hypothesis, which presented briefly before Flor's data makes comprehension of his concepts easier and more permanent. This paper discusses this teaching method and illustrates a simple application of the gene-for-gene hypothesis.

The presentation builds on familiar concepts. Each person uses locks on the doors of his residence, business, and automobile to prevent the entry of an unwanted person, especially one who might rob or plunder. Only a person with a key specific for the lock can enter. Similarly, we consider that the flax plant has "locks" (resistance genes) which prevent unwanted visitors (pathogens) from entering. If, however, the pathogen has the "keys" (pathogenicity genes) to the specific "locks" of the flax plant, then and only then is the pathogen able to open the "lock" enter the plant, and to rob and plunder (exert its pathogenicity).

The interaction of "locks" and "keys" is illustrated in Figure 1. Resistance "locks" (genes) are shown as being dominant, which is the usual situation in crop species, including flax. Pathogenicity "keys" are shown as being recessive, following the mode of inheritance of pathogenicity in the dicaryotic flax rust (1,9) and oat loose smut (7) fungi. However, whether resistance and pathogenicity are conditioned by dominant or recessive genes is not germane to the operation of the gene-for-gene hypothesis.

LINE	RESISTANCE "LOCKS" 1/	PATHOGENICITY "KEYS" 2/	DISEASE EXPRESSION 3/
1		ANY, FOR NONE NEEDED	S
2	A_	NONE	R
3	A_	aa	S
4	A_ B_	aa	R
5	A_ B_	bb	R
6	A_ B_	aa bb	S
7	A_ B_ C_	aa bb	R
8	A_ B_ C_	aa bb cc dd ee	S
9	A_ B_ C_ D_ E_	bb cc dd ee	R
10	A_ B_ C_ D_ E_	MASTER KEY	NO EVIDENCE FOR

Figure 1. The use of resistance "locks" and pathogenicity "keys" to illustrate the gene-for-gene hypothesis for interactions in host-parsite systems: 1—the "hasp" represents a locus, at which any one of an allelic series of "locks" can occur. The absence of a "lock" on a given "hasp" indicates the recessive allel so that no "effective lock" is present. 2—The absence of a given "key" indicates the dominant allel so that no "effective key" is present. 3—S indicates susceptible, R resistant.

More important is the understanding that disease expression (whether resistant or susceptible in the host; virulent or avirulent in the pathogen) is conditioned by a given number of genes.

If the host has no resistance "locks" (i.e., assuming resistance is dominant, it has recessive genes at each locus which conditions disease reaction), then no particular "key" is needed to gain entrance, and the reaction is one of susceptibility (Figure 1, Line 1). If however, the host has "lock" A_, and the pathogen has no "key", then "lock" A_ effectively limits the advance of the pathogen, and the reaction is one of resistance (Line 2). On the other hand, if the pathogen possesses "key" aa specific for "lock" A_, the reaction is susceptible (Line 3). The addition of "lock" B_ renders the host resistant to the pathogen with only gene aa (Line 4) or gene bb (Line 5), but the pathogen with "keys" aa and bb is able to attack successfully a host with "locks" A_ and B_ (Line 6). The addition of a third "lock" C_ excludes the pathogen with only "keys" aa and bb, however, as C_ becomes the limiting factor in the host: parasite interaction (Line 7). The addition of "key" cc (Line 8) enables the pathogen to attack host A_B_C_. Additional "keys" dd and ee neither help nor hinder the attack on A_B_C_ but may be carried in reserve until needed on a host with some combination of "locks" A_,B_, C_,D_, and E_. A host plant with these five "locks" is resistant to any race of the pathogen which is deficient in one or more

of the five necessary pathogenicity "keys." Thus the absence of "key" aa for "lock" A_ becomes the limiting factor in the development of pathogen A_ bbccddee on host A_B_C_D_E_ (Line 9).

The capable student may ask whether a single master "key" can open several resistance "locks" (Line 10). There is no evidence for such a relationship; all gentic evidence points to a single specific "key-for-lock" system, not a "master-key-several lock" system.

Flor (1) has shown that flax has at least 25 different rust resistance genes but that they occur as multiple alleles of only 5 different loci. Thus, 5 appears to be the maximum number of different rust resistance genes a flax plant can possess unless additional loci are found. This is quickly, if somewhat crudely, illustrated (Figure 1) to students by assuming there is room on the "door" to the flax plant for only 5 "hasps" (loci). Any of several possible "locks" can be used in a given "hasp" but, because of the limited number of "hasps," only 5 "locks" can be utilized at one time. By contrast, there appears to be no limit (no allelic groups) to the number of different "keys" (pathogenicity genes) a given race of the flax rust can have (1).

Among the possible applications (1,9) of the gene-for-gene hypothesis is its use to determine the disease-expression genotypes of host or pathogen when the genotype of only one of the interacting organisms is known. Since the disease expression is the phenotype for both the host and pathogen, knowing the phenotype and one genotype gives the other genotype. Flor (1,9) has shown that, where the genotypes of flax and flax rust are determined experimentally, they corroborate each other.

It is stimulating to students to apply the hypothesis to an "unknown" disease. Stem rust of oats serves well as an "unknown" disease because the inheritance of resistance of commercial varieties to common stem rust races has been reasonably well determined.

Table 1 gives the rust-resistance genotypes for the oat varieties Richland, Rodney and White Tartar, and the rust reactions of

Table 1. Results of applying the gene-for-gene hypothesis to interactions of 3 oat varieties and 7 races of the oat stem rust fungus, under the assumption of dominance of resistance in the host and avirulence in the pathogen

		Race genotypes conditinoing indicated disease expression													
Oat Variety	Oat Genotype	Race 2		Race 6		Race 7		Race 7A		Race 8		Race 8A		Race 13A	
		a/	b/												
Richland	AAbbdd	R	A_	S	aa	R	A_	R	A_	S	aa	S	aa	S	aa
Rodney	aaBBdd	R	B_	R	B_	R	B_	S	bb	R	B_	S	bb	S	bb
White Tartar	aabbDD	R	D_	S	dd	S	dd	S	dd	R	D_	R	D_	S	dd

a/Disease expression (or "host reaction"). R indicates resistant, S susceptible.
b/ Genotype, read vertically for each race.

these varieties to certain rust races (10). The genotypes of these oat varieties and their reactions to these races were determined experimentally. Applying the gene-for-gene hypothesis (and assuming avirulence to be dominant), one can assign genotypes to the different rust races as shown in Table 1.

Note that the oat genotype is read horizontally; the race genotype, vertically. Thus, the genotype of Richland is AAbbdd and that of race 7 is A_B_dd. It is apparent from Table 1 that any race, even race 2, can attack successfully a variety such as Markton with genotype aabbdd (Table 2). On the other hand, only race 13A has the minimum genotype necessary to parasitize

Table 2. Disease expression caused by interactions of 5 oat varieties and 7 races of the oat stem rust fungus

		Disease Expression						
Oat Variety	Oat Genotype	Race 2	Race 6	Race 7	Race 7A	Race 8	Race 8A	Race 13A
Markton	aabbdd	S a/	S	S	S	S	S	S
Garry	AABBdd	R a/	R	R	R	R	S	S
C.I. 7144	AAbbDD	R	S	R	R	R	R	S
Burnett	aaBBDD	R	R	R	S	R	R	S
C.I. 6909	AABBDD	R	R	R	R	R	R	S

a/ S Indicates susceptible, R resistant

an oat variety with the genotype AABBDD. Similarities and differences among the races (which are morphologically indistinguishable) soon become apparent. These relationships are more pronounced and meaningful when expressed at the genotypic level than they are at the level of disease expression (the phenotype).

Unfortunately, from the pedagogical standpoint, the gene-for-gene hypothesis does not explain adequately all facets of interactions in host:parasite systems. It appears adequate for systems with clear cut host:parasite interactions, especially those involving a hypersensitive disease expression; however, typical of biological phenomena, many expressions of disease are frequently not black and white but many shades of gray. In the cereal rusts, several infection types between complete resistance and complete susceptibility occur. Some cereal varieties may possess only adult plant or field resistance. Frequently such resistance is inherited quantitatively. Sometimes disease reactions that are monogenically inherited are not easily explained by the gene-for-gene hypothesis. For instance, the E gene in oats conditions a highly resistant reaction to some stem rust races, a completely susceptible reaction to others, and an indeterminant reaction (resistant- and susceptible-type pustules on the same leaf) to still others. Table 2 would not appear so simple had the E gene been included in the host genotypes.

The difficulty in applying the gene-for-gene hypothesis to more complex host:parasite systems does not preclude its application to such systems. This hypothesis contributed greatly to understanding the genetics of interactions in the less complex systems (such as flax and flax rust) and, as additional data accumulate on the genetics and physiology of complex host:parasite systems, it will undoubtedly facilitate understanding of these systems also.

Literature Cited

1. Flor, H. H. 1956. Advances in Genetics 8:29-54.
2. Toxopeus, H. J. 1956. Euphytica 5:221-237.
3. Moseman, J. G. and C. W. Schaller. 1960. Phytopathology 50:736-741.
4. Powers, H. R., Jr., and W. J. Sando. 1960. Phytopathology 50:454-457.
5. Boone, D. M. and G. W. Keitt. 1957. Phytopathology 47:403-409.
6. Holton, C. S. 1959. C. S. Holton *et al.* ed., Plant Pathology: Problems and Progress, 1908-1958. Univ. of Wisconsin Press, Madison, pp. 145-156.
7. Holton, C. S. and P. M. Halisky. 1960. Phytopathology 50:766-770.
8. Person, C. 1959. Can. J. Botany 37:1101-1130.
9. Flor, H. H. 1955. Phytopathology 45:680-685.
10. Murphy, H. C. and F. A. Coffman. 1961. F. A. Coffman, Ed., Oats and Oat Improvement. The American Society of Agronomy, Madison, Wisconsin, pp. 207-226.

Media Sterilization With Propylene Oxide

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Abstract: Sterilization of potato dextrose agar against bacteria air contaminants occurs during the 6th hour of exposure and against fungi in the 4th hour. The fungicidal activity was broader than the bactericidal activity of propylene oxide. Direct application of propylene oxide to Petri plates containing PDA was ineffective. With plastic plates a chemical reaction took place between the chemical and the plastic. In a closed system sterilization of the plates and medium was accomplished at approximately 1.25 ml of propylene oxide per liter of volume. The addition of propylene oxide directly to nutrient broth effected 90% sterility under certain conditions.

The use of propylene oxide as a sterilizing agent for various types of biological products (1, 2, 3) suggests that it could be used for field sterilization of microbiological media or be useful in high-school and college laboratories where sterilizing equipment is absent or inadequate. According to Hansen (4) there is very little physical-chemical alteration of organic substances which have been exposed to propylene oxide. A disadvantage is that the vapors are highly flammable in low concentrations. This experiment was designed to determine if an inexpensive, effective method of sterilization with propylene oxide could be developed.

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