

## Genetical Aspects of Interaction Between Plant Hosts and Their Soilborne Pathogens

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The assumption inferred by the title is that the genetics of interactions between plants and soilborne pathogens differs somewhat from the genetics of interactions with pathogens that attack aerial plant parts. I tend to think this is not true. It seems reasonable that the basic genetics of interactions would be similar, but that the manifestations of these interactions might be quite different. The reason for believing that the basic genetics would be similar is based on the observations of the universality of interorganismal genetics (6). Since the same basic genetic pattern seems to emerge for interactions between plants and fungi (basidiomycetes, ascomycetes, and phycocomycetes), bacteria, nematodes, insects, etc., it seems only logical that the same genetic pattern would extend to fungi that attack the plant below the soil line.

There are a number of apparent parallels in the development of concepts of host plant resistance, pathogen variability, methods for characterizing the phenotypic differences, and methods for analyzing the differences according to formal genetics. Similar observations have been made for both aerial and soilborne pathogens, but the intensity with which the research has been pursued in each area has been quite different.

The existence of differences within a host species in reaction to a pathogen has been known for a long time. The first analysis via Mendelian genetics occurred very shortly after the rediscovery of Mendel's work. Biffen (2) showed that the difference between the reactions of two wheat cultivars to *Puccinia striiformis* (the cause of stripe rust) was controlled by one gene. He depended on inoculation by natural infection in the field, and recorded symptom development both on the parent cultivars and F<sub>1</sub> and F<sub>2</sub> plants during the development of the epidemic and shortly before harvest. The results were different for the two sets of data.

In the four decades following Biffen's (2) publication, considerable effort was made to screen wheat germ plasm for resistance to the rust diseases. Numerous wheat cultivars were bred, released, and grown as rust resistant. Resistant cultivars were found to become susceptible as the result of the occurrence and increase in frequency of new races of the pathogens, and the concept of physiological races of pathogens gained prominence. Resistance to the rusts was easy to classify, because individual cultivars inoculated with different strains of a pathogen had unique phenotypes. High levels of resistance were usually preferred for breeding programs.

Flax was an important crop in the Dakotas and Minnesota at the turn of the century. It was considered to be a "new lands" crop, primarily because of the wilt disease. Research had shown that the causal agent is *Fusarium lini*; that the fungus spreads in plant debris, seed, and soil; and that, once established in the soil, it may persist for many years. Resistance was found by selecting plants that could survive in "wilt sick" plots, established first in North Dakota and later in Minnesota. Cultivars that survived best in plots in North Dakota were not necessarily those that survived best in plots in Minnesota. The proof of the existence of races was established by inoculations with individual isolates. The demonstration of the existence of races was more difficult because discreet phenotypes were not observed, but reproducible

differences were established to prove the differences among isolates of the pathogen. The wilt-resistant cultivars of flax retained their wilt resistance. The segregation of the resistance in crosses suggested that resistance was controlled by many genes, but this conclusion may have its relevance in the difficulty of distinguishing phenotypes of resistant and susceptible plants in segregating populations. Resistance in flax to *Fusarium* wilt seems to be lasting in spite of the existence of genetic variability in the pathogen. Resistance to *Fusarium* wilt in tomatoes seems to be quite simply inherited, but also seems to have lasted a long time.

**Breeding for resistance.** Is there anything that distinguishes the genetic interactions of leaf pathogens and soilborne pathogens with their hosts? Are the interactions with their hosts controlled by genes that follow different basic patterns? Are there obvious differences in how the experiments were performed that led to the conclusion that there were different kinds of host and parasite genes? If the inheritance of interactions between plants and leaf or root pathogens follows similar patterns, are the manifestations sufficiently different to suggest differences that lead to misinterpretations of the data? Do soilborne pathogens have unique biological characteristics that make the genetics of their interactions with the host plant appear unique? These are but a few of the questions that should be asked.

The inheritance of both host and parasite variability has been analyzed for many of the leaf diseases. The concepts of resistance and susceptibility of a host plant and virulence and avirulence in a pathogen changed greatly with the research of Flor (4,5). His research on flax rust showed the importance of considering the genes in both host and parasite; a host plant is not resistant unless the pathogen has the corresponding gene for avirulence. Screening of host lines for resistance is, in fact, a screening to determine what genes for avirulence are present in the pathogen population. The manifestations are dependent on the frequency of the gene for avirulence, the selection of the alternate alleles of that gene, and the expression of that gene in a given environment (physical and biological).

Screening for resistance to stem rust in wheat illustrates the approach used with many leaf pathogens prior to about 1950. The host lines were challenged with large doses of inoculum of the pathogen. The plants that survived, preferably the immune plants, were selected for further breeding. The technique selected for genes that gave high levels of resistance. Flor had strains of the pathogen that gave different infection types on host lines with single *R* genes. For the studies on the inheritance of resistance, he selected strains to which the host lines had high levels of resistance. He wanted to magnify the differences between alternate alleles but, in doing so, he imparted the suggestion that the gene-for-gene relationship held only for host genes that gave high levels of resistance. Beginning in the 1950s, a great interest developed in using resistance that did not give immunity, but only slowed the rate of development of epidemics. The inference was that these genes did not follow the gene-for-gene relationship.

Selection for resistance to root pathogens seems to have evolved quite differently. Available levels of resistance have usually been low, the methods of testing have usually involved selecting at the population rather than the individual plant level, the use of recurrent selection to increase levels of resistance, and screening with resident and mass isolates of the pathogen. In many respects, the selection for the resistance to soilborne pathogens has been for more subtle differences between host lines.

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In the past 10–20 yr the trend in research on leaf and root pathogens seems to have begun to be reversed. Much emphasis has been placed on developing protocols to detect genes that give low levels of resistance to leaf pathogens. The emphasis with soilborne pathogens has been to develop protocols to detect resistance controlled by genes that give high levels of resistance and where each gene can be identified individually. Regardless of the approach used to detect genes for resistance to leaf pathogens, the same basic genetic pattern of interactions seems to emerge. Though the detailed genetic studies have rarely been made for root pathogens, the pattern that emerges is consistent with the gene-for-gene hypothesis.

**Genetics of resistance.** Reasonably detailed genetic analyses have been made for resistance of potato plants to root knot nematodes and of soybeans to *Phytophthora megasperma* var. *sojae*. Cultivars of soybeans were evaluated by a technique whereby the pathogen was introduced into the stem of the plant. This procedure selects for high levels of resistance to the pathogen once it was introduced into the plant. It is doubtful whether the procedure would select genotypes that give resistance to infection, or low levels of resistance of any type. The selective scheme to identify and genetically characterize resistance of soybean to *Phytophthora* root rot is very similar to the scheme used in the early studies of rust resistance in wheat. It detects genes that give sufficiently high levels of resistance that the plant will survive when inoculated with large doses of the pathogen. The genes identified in soybean follow a pattern with strains of *P. megasperma* var. *sojae* that is consistent with the gene-for-gene hypothesis.

A genetic analysis in both host and pathogen has been made with a number of leaf diseases. The basic pattern of the gene-for-gene relationship seems to hold for almost all naturally occurring genetic variability whether the resistance is either for high or low levels (3). It has been possible to do the formal genetic analysis of one organism and predict the genetics of the other with excellent accuracy. Can a similar analysis be made for resistance to a soilborne pathogen (eg, resistance of soybean to *Phytophthora* root rot)? Athow (1) has described five (+1) genes for resistance to *P. megasperma* var. *sojae* in soybeans. The reactions of seven host lines to nine races of *P. megasperma* var. *sojae* are presented in Table 1. The genes in the host have been analyzed in great detail by Athow and co-workers (1). There are three *Rps* alleles at the *Rps* 1 locus, and one *Rps* allele at three other loci (I have given the tentative designation of *Rpsx* to the gene in Altona). If it is assumed that the gene-for-gene relationship holds (there is no reason to believe it doesn't), then one can write the genotype of each race of the pathogen. Once this is done, it is easy to see what other races of the pathogen can emerge, given this set of host differential lines.

**Patterns of genetic variation.** The following brief discussion on the comparative genetics of interactions will begin with three postulates: one, that there is genetic variability within a host species and/or pathogen species for the genes controlling the interactions; two, that the basic pattern of genetic interactions is the same for

plants and both aerial and soilborne pathogens; and three, that there is a fundamental difference in the physical and biological environments in which a plant and a parasite interact on the leaves or on the roots of the plant.

There are very large differences in the magnitude of the genetic variability among combinations of single plant species with single pathogen species. There are differences both in the numbers of genes involved and in the expression of alternate alleles at loci involved in interactions. Wheat has more than 30 loci for reaction to stem rust, and each locus can be studied independently because the differences between alternate alleles can be made to be large. There seems to be very little genetic variability in corn to the *Fusarium* spp. that cause stalk and root rots. There are differences, but they are small, and it has not been possible to determine the contribution made by individual genes. Where it has been possible to identify individual loci that control the interactions in both host and parasite, the basic pattern is almost always consistent with gene-for-gene interactions. The same basic pattern seems to emerge from detailed studies of the genetics of interaction between plants and fungi, bacteria, insects, nematodes, viruses, etc.

There are several possible reasons why there should appear to be such great differences in the amount of genetic variability in interactions. A common explanation is that the most evolved interactions would show the greatest variability. By this criterion, wheat has evolved with *P. graminis* for a long time. There may also be a difference in the kinds of interactions between plants and parasites depending on whether they are obligate or facultative parasites. In some cases, there may be much genetic variability, but the protocols used may not allow the expression of the genes involved. In other cases, they may be restrictions on the extent of genetic variability that is tolerated in some host-parasite combinations. Genetic variability may be tolerated for only certain types of loci.

It is possible to generate genetic variability. Mutations to increased virulence in pathogens and to susceptibility in host plants have been generated with relative ease. These mutations are of genes that follow the gene-for-gene relationship. Such mutations would be expected because they are from alleles that possess the specificity of interactions of the gene products to alleles whose products do not interact. Pathogen genes whose functions are necessary for growth in the host have been identified by the induction of temperature-sensitive mutants. The characterization of the *ts* mutants has not been accomplished, nor has the induction of comparable mutations in host plants.

**Interactions among pathogens.** Is there a fundamental difference in the physical and biological environments in which plants interact with aerial or soilborne pathogens that affects the apparent genetics of the interactions? There are several reasons for believing this to be true, and a few will be dealt with here. The number of fungi on the leaves of plants seems to be lower than the numbers on roots. Though there are large numbers of spores of fungi in the air, there seem to be few that are found on healthy leaves. I have looked at

TABLE 1. The reaction<sup>a</sup> of seven soybean lines, and the designated genes for each, to nine races of *Phytophthora megasperma* var. *sojae*. The postulated genotype of each race is also given

Race	Pathogen strains						Soybean lines						
	Postulated genotype						Harosoy	Mudken <i>Rps</i> 1	PI84637 <i>Rps</i> 1b	PI54615-1 <i>Rps</i> 1c	Altona <i>Rpsx</i>	PI86972-1 <i>Rps</i> 3	PI86050 <i>Rps</i> 4 <sup>b</sup>
1	<i>P</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>P</i> x	<i>P</i> 3	<i>P</i> 4	S <sup>c</sup>	R	R	R	R	R	R
2	<i>p</i> 1	<i>p</i> 1b	<i>p</i> 1c	<i>p</i> x	<i>p</i> 3	<i>p</i> 4	S	R	S	R	R	R	R
3	<i>p</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>P</i> x	<i>P</i> 3	<i>P</i> 4	S	S	R	R	R	R	R
4	<i>p</i> 1	<i>P</i> 1b	<i>p</i> 1c	<i>P</i> x	<i>P</i> 3	<i>P</i> 4	S	S	R	S	R	R	R
5	<i>p</i> 1	<i>P</i> 1b	<i>p</i> 1c	<i>p</i> x	<i>P</i> 3	<i>p</i> 4	S	S	R	S	R	R	S
6	<i>p</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>p</i> x	<i>p</i> 3		S	S	R	S	S	...	...
7	<i>p</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>p</i> x	<i>p</i> 3	<i>p</i> 4	S	S	R	S	S	S	S
8	<i>p</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>p</i> x	<i>P</i> 3		S	S	R	R	R	...	...
9	<i>p</i> 1	<i>P</i> 1b	<i>P</i> 1c	<i>p</i> x	<i>P</i> 3		S	S	R	S	R	...	...

<sup>a</sup>Data are from Athow et al (1).

<sup>b</sup>*Rps*4 in F<sub>2</sub> progeny from PI86972-1 × PI86050.

<sup>c</sup>R = resistant and S = susceptible.

thousands of leaves and have rarely found spores of fungi on those leaves in sufficient numbers to affect the germination of spores of the fungus artificially placed on the leaves, and we know very precisely the influence of density of *Erysiphe graminis* spores on spore germination and the infection process. Plantings from leaves haven't given nearly the populations of organisms that were obtained from roots of the same plants. The data suggest that the roots are in close association with large numbers of many different organisms, and that the numbers and closeness of the association are sufficient to affect the infection by a given pathogen. If the surfaces of the roots really do contain a large number of each of many different organisms, then the interaction of one pathogen with the host could be affected by the interactions of the other microorganisms that are present and the host. Sidhu and Webster (7,8) have demonstrated that the interaction of one pathogen with a plant may have no effect on the plant's reaction to a second pathogen, may increase resistance, or may increase susceptibility. Sidhu and Webster (7,8) examined the genetics of resistance in tomatoes to three pathogens. No genetic analyses were made with the pathogens. From a knowledge of the genetics of the host plant, the genotypes of the pathogens could be postulated. But what are the genotypes of the pathogens that govern their interactions with each other? Do these interactions follow the pattern of interorganismal genetics? Interactions between plant pathogens have not been studied with respect to the genetic control of those interactions, at least not to my knowledge. Are there genes that specifically control the interactions between two fungi, or a fungus and a nematode, etc?

The data for tomatoes and tomato pathogens show that the phenotype of interactions of a plant with one pathogen can affect the phenotype of the interaction of the plant with a second pathogen. Whether the interaction is between the two pathogens or between two separate plant-pathogen interactions is not known. Can one plant-pathogen interaction induce either resistance or susceptibility in the plant to a second pathogen? This is a scenario involving only two pathogens on the plant roots. The complexity would grow quickly with the presence of more species of pathogens. If consideration is given to the possibility that organisms in the rhizosphere near, but not on or in, the roots of the plant also could participate in interactions with the plants, the complexity becomes still more staggering.

Resistance to leaf pathogens has frequently appeared to have a very complex inheritance. As the analyses have become more detailed and the sources of variance were sorted out, certain basic genetic patterns of interaction have emerged (3). To accomplish the more detailed analyses, it has frequently been necessary to use special environmental conditions to magnify the genetic differences between host lines. The results of research with soybean and *Phytophthora megasperma* var. *sojae* illustrate how the development of a particular procedure has enabled the identification of a number of *Rps* genes in soybean. The procedure enables the classification of individual plants and the identification of genes that give high levels of resistance. It is doubtful that it would identify genes that condition low levels of resistance. The identification of individual host *Rps* genes and races of *P. megasperma* var. *sojae* has shown that the genetic pattern of interactions is consistent with the gene-for-gene hypothesis. Many of the analyses would probably not have been possible if a third organism were affecting the interactions of the first two. It becomes easy to see why so little is known about the genetics of interactions between plants and root pathogens.

**Durability of resistance.** There seems to be a general feeling that resistance to root pathogens is more durable in commercial production than is resistance to leaf pathogens. I don't know whether this is an acceptable generalization, or a consequence of the kinds of research efforts, an artifact of procedures, total nonsense, etc. It may be worth considering what affects the length of time a gene for resistance, an *R* gene, remains effective against a leaf pathogen.

If a gene, *px*, is present, but in very low frequency, the corresponding *R* gene will usually appear to be very effective. There are many such examples in the literature describing isolates of a

virulent pathogen that is present in low frequency at the time of release of a new cultivar. The cultivar will remain resistant only until the frequency of *px* in the pathogen population increases in frequency. Usually resistance does not last long. In some examples, the cultivar was susceptible by the time the seed had been increased for commercial production.

The rust diseases provide numerous examples in which the *px* gene was present, but not expressed. In these examples, the *R* gene was effective against all the tested isolates of the pathogen. The *R* gene was effective because the isolates of the pathogen were functional diploids (dikaryons) and heterozygous for the recessive *p* gene. In this situation, the host line appears to have an *R* gene for which the pathogen has only the dominant *P* allele. Resistance will last until the pathogen can express the recessive allele, followed by the subsequent selection of the strains of the pathogen that express the recessive *p* allele.

Nearly every plant breeder has encountered a situation in which resistant progeny were obtained from crossing two susceptible parents. This is frequently encountered in field ratings with natural infection. In the simplest cases, these observations were due to combining two *R* genes, *R1* and *R2* (one from each parent) and to the presence of both *p1* and *p2* in the pathogen population, but not both in a single strain of the pathogen. Resistance based on a combination of *R* genes for which the corresponding *p* genes are present in the pathogen population, but not in the proper combinations, will last until the pathogen can recombine and express its *p* genes and selection can increase the frequency of the pathogen strains that have the proper combination of *p* genes.

A fourth type of situation occurs when an *R* gene is introduced into a cultivar and the corresponding *p* gene is not present in the pathogen population. That *R* gene will remain effective until the *p* allele is introduced into the population by mutation  $P1 \rightarrow p1$  (or, if they exist, in isolates from other geographic areas), the *p* allele is expressed, and selected so that it increases in frequency.

Examples of all of the above are extensive for leaf pathogens. Are there reasons to believe that differences between leaf and root pathogens would produce different results? It is easy to conceive scenarios that suggest selection pressures that would make rapid changes in gene frequencies in both leaf and root pathogens or that would prevent rapid changes. The two aspects that seem especially pertinent are the methods of dispersal and the competition among pathogen units for infection sites. Wind can be important in long-distance dispersal of both leaf and root pathogens. Irrigation water can disseminate root pathogens over medium distances (throughout a field). Epidemics of leaf pathogens usually spread distances measured in centimeters and meters, whereas root pathogens may spread only millimeters and centimeters. The ability of a pathogen unit to compete for infection sites with other pathogen units once it arrives at the host plant is poorly understood. It appears that the role of other organisms in infection is far more important for root pathogens than leaf pathogens.

The relative importance of the sexual cycle for genetic recombination for leaf and root pathogens seems equal. Both groups of pathogens probably have the equal proportions of members with a prevalent sexual cycle. The intimacy of association in the rhizosphere may make the role of heterokaryosis and other somatic mechanisms of genetic exchange more important for sources of genetic variability in soil pathogens than in leaf pathogens. The ability to express a new phenotype may be more limited with soil pathogens because of the intimacy of interactions between pathogens and saprophytes on the root surface.

There seems to be no obvious reason to suspect that the frequency of mutations is significantly higher in either leaf or root pathogens, but there may be some significant differences in the type of selection for individual genotypes. The importance of the genotype of the individual seems to be greater for leaf pathogens. With root pathogens, the expression of mutants may be more important at a population level. If this is true, a more rapid change in gene frequencies would be expected in leaf pathogens than in root pathogens. The rate with which gene frequencies change in leaf pathogens has been experimentally investigated. There is evidence that the pathogen may have to pay a price for the  $P \rightarrow p$  change in

the presence of either *R* or *r*. What price, if any, is associated with the  $P \rightarrow p$  transition with root pathogens is not known.

I am always surprised to find that a large number of plant pathologists and breeders have never taken the time to understand the genetics of interaction between plant hosts and parasites. The perceived complexities become quite simple once the rules governing the genetic interactions are understood. If so few people understand the genetics of interactions between two organisms, the chances of sorting the components when three or more organisms are interacting becomes essentially impossible. Thus, it is not surprising that very little is known about the genetics of interaction between plants and soilborne pathogens.

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