Parasite:Host Specificity and Resistance/Susceptibility, Two Concepts, Two Perspectives

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Parasite:host specificity and resistance/susceptibility are two of the broadest concepts within the science of plant pathology. These two concepts are related to every facet of plant pathology, even to efforts that seek to control disease by cultural practices, chemical applications, quarantines, or other means that do not directly use genetic manipulation of crop genotype. There are differing views of these concepts within the literature of plant pathology. Because of their importance and because of these differences in viewpoint, we think that these concepts need to be discussed in a more general forum than is available in a paper that presents research results from a given experiment. To that purpose, we direct this letter. We will present the material as a discussion of our own views and the published views of others, particularly those of Vanderplank (11–13). Within most of the letter we will use terms from the literature we address, although we recognize the terms are not adequate.

Two concepts. We see no absolute relationship between the concepts of parasite:host specificity and resistance/susceptibility to disease. The concept of specificity deals with the biological relationship between two organisms while the concept of resistance/susceptibility deals with practical considerations of the relative value of two or more hosts in control of disease within agriculture. A specific relationship within a parasite:host system may result in resistance, but another specific relationship may result in susceptibility. Specificity is a characteristic of parasite:host associations rather than of single organisms within the associations. Parasite:host specificity embodies, in our view, the concept that a given genotype of a parasite and a given genotype of its host function together to bring about a specific relationship between them.

Plant pathologists often view specificity either from the host side or from the parasite side, but usually work with both. Specificity is most often related from the host side with the idea of "specific resistance". This indicates a host that is resistant only to those parasites that are avirulent. The presence of avirulence in the parasite is implied and the interaction of host genotype and parasite genotype bringing about the specific resistance is implied. Thus, as regards specific resistance, the result of parasite:host specificity can be interpreted both as host resistance and parasite avirulence. In other cases, "host-specific toxin" is used to indicate a toxin, produced by a parasite, that is toxic to some hosts and not others. This implies that some host genotypes are sensitive to the toxin while others are not sensitive. It further implies that the association of the host genotype for sensitivity and the toxin, or toxin-producing parasite, results in pathogenesis. The host cultivar having the genotype for sensitivity is regarded as susceptible; from an agricultural viewpoint this is certainly correct. Toxicity is closely related to the concepts of pathogenesis and susceptibility. In such cases, specificity results in susceptibility. Again, resistance results from specificity in some cases but susceptibility results from specificity in other cases.

Specificity and resistance. Vanderplank (13, pages 5–12) has expressed another view that there is an absolute relationship of specificity to susceptibility within plant disease systems. This view indicates that susceptibility results from specificity within gene-for-gene systems as shown by Flor (3,4), as well as in higher-order variation at the parasite species:host species level. Table 2.1 of Vanderplank's presentation (13, page 6), shows five of the possible 32 homozygous genotypes of pathogen and host within a system having five sets of corresponding gene pairs (8,9). From this table, he has concluded that the diagonal of "Susceptible" shows that susceptibility is specific to "*RR*" host alleles interacting with "*vv*" pathogen alleles. Only "*RR*" host- and "*vv*" pathogen-alleles are accounted in Vanderplank's table, but within that context we can reasonably assume that at loci where pathogen alleles are not specified to be "*vv*," "*VV*" alleles for avirulence occur. Similarly, at loci where host alleles are not specified as "*RR*," "*rr*" alleles occur. Table 1 is a subset of Vanderplank's Table 2.1 (13, page 6). We use the same data configuration, but have shown complete genotypes at each locus. Within this data configuration, the possibility that interaction of "*RR*" host genotypes and corresponding "*VV*" parasite genotypes resulting in "Resistance" is equally as probable as the possibility that "Susceptible" is the result of interactions of "*RR*" and "*vv*." This is true for any-sized set of data that show this configuration. One cannot unequivocally determine whether specificity results in resistance or in susceptibility by examining empirical data configurations.

The empirical data configuration shown in our Table 1 is commonly observed when two hosts are inoculated with two parasites. Flor (3,4) began his investigation of the flax rust system with observations of this data configuration. He conducted experiments in which two hosts were crossed, two parasites were crossed, and members of segregating populations of the host were inoculated with members of segregating populations of the parasite. Results of Flor's experiments indicated that resistance occurred only when certain parasite genotypes and certain host genotypes came together, and susceptibility occurred otherwise. Resistance was the result of specific host genotypes and specific parasite genotypes functioning together. Susceptibility was the result of not having the gene for resistance, not having the corresponding gene for avirulence, or both. When Flor (4) conducted experiments with parental materials that gave results similar to those in our Table 1 he found that there were always at least two host genes and at least two parasite genes segregating in the experimental populations. Flor (4, page 42) clearly indicates that, in his work, resistance was the result of specificity with the statement: "All host-parasite combinations other than N-A_N and L-A_L resulted in susceptibility." We interpret this to indicate that resistance is the result of specific interaction of certain parasite genotypes and certain host genotypes within the flax rust system. The gene-for-gene concept has often been stated as: for every gene for resistance in the host, there is a corresponding gene for pathogenicity in the parasite. We suggest that another important aspect of the relationship is that parasite genotypes and

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host genotypes function together to bring about an outcome, the phenotype of the association.

When studying some parasite:host systems, for example *Phytophthora infestans*:*Solanum*, only empirical data configurations are readily available because one or both organisms cannot be crossed readily. In these cases, we can only draw conclusions about the result of specificity by establishing a rule that cannot be refuted by examining the available data. Vanderplank (13, Table 2.2, page 8) shows a 16 × 16 matrix representing all possible potato genotypes at four loci and all possible races of *Phytophthora infestans*. The races are named according to the host lines to which they are virulent. We have reproduced a subset of that table in our Table 2. We have used the subset for simplicity in presentation; the principle is the same as in Vanderplank's table (13, Table 2.2, page 8). Vanderplank (13, page 9) concludes that, in his table, "At each level susceptibility is on the diagonal and therefore specific, and resistance is off the diagonal and therefore unspecific." In relation to the data matrix used, there are two major problems with this conclusion. First, "S" occurs in row 1, column 1, as a result of potato genotype "*r1r1r2r2*" with race O. The potato genotype "*r1r1r2r2*" has no genes for resistance and race O has no genes for virulence; "S" could not be the result of an interaction between genes for resistance and genes for virulence. Secondly, there is susceptibility off the diagonal as well as on the diagonal; susceptibility occurs in all cells in the first row, all cells in the last column, and in other cells in the upper right portion of the matrix. Thus, the diagonal check for susceptibility does not hold when a data matrix showing all possible genotypes of each organism is considered. Both Person (8) and Robinson (9) also accounted for genes for virulence and relate susceptibility to interaction of genes for resistance and genes for virulence in their models. Thus, they err in much the same way as Vanderplank.

When we examine Vanderplank's Table 2.2 (13, page 8), Person's model (8), Robinson's model (9), or our Table 2, we see in every case that resistance occurs where there is a gene for resistance and no corresponding gene for virulence. From these data, we can only interpret that the absence of a gene for virulence at a locus indicates the presence of a gene for avirulence at that locus. Resistance is the result of interaction of genes for resistance and genes for avirulence within the framework of the data presented for *Phytophthora infestans*:*Solanum*. Resistance is, in this case, specific to genotype of both organisms, *this is not true for susceptibility*. Our Table 2 and Vanderplank's Table 2.2 (13) are essentially theoretical models of a gene-for-gene relationship with the premise that resistance is specific to genotype of both organisms and considering two sets and four sets of corresponding gene pairs, respectively. When a gene for resistance and a corresponding gene for avirulence occur in at least one set of corresponding gene pairs, a phenotype resulting in resistance occurs. This theoretical model holds with reality in the case of *Phytophthora infestans*:*Solanum* and in many other systems. Webster (14) has correctly indicated that a gene-for-gene relationship has not been proven in *Phytophthora infestans*: *Solanum*. The association of resistance with specific parasite:host genotypes, however, holds within the framework of available data.

TABLE 1. Theoretical data showing the interaction of some genotypes of a parasite:host system in which two sets of corresponding gene pairs occur; this table uses the same configuration as a table from Vanderplank[a], but shows complete homozygous gentoypes

	Host genotype:[b]	
Pathogen genotype[b]	*R1R1r2r2*	*r1r1R2R2*
v1v1V2V2	Susceptible	Resistant
V1V1v2v2	Resistant	Susceptible

[a] This table shows a 2 × 2 data configuration and shows complete homozygous genotypes for two loci in each organism. Vanderplank's table (reference 13, page 6) shows a 5 × 5 data configuration, but does not show complete genotypes.

[b] *R* and *r* symbolize alternate allelic conditions at loci 1 and 2 in the host and *V* and *v* symbolize alternate allelic conditions at corresponding pathogen loci 1 and 2.

Specificity and susceptibility. In contrast with cases in which resistance is clearly the result of specificity, we see that susceptibility results from parasite:host specificity in other cases. Vanderplank (13, page 11) briefly discusses the case of Victoria blight in oats where susceptibility is the result of host sensitivity in some cultivars of *Avena* and the presence of a toxin produced by a specific genotype of *Helminthosporium victoriae*. Sensitivity is controlled by genotype at one locus in *Avena*. Toxin production is under genetic control; thus, susceptibility, in this case, is indeed the result of genetic specificity. This is apparently true for several other parasite:host associations in which toxins are involved in pathogenesis. We can only conclude that there is no absolute relationship between parasite:host specificity and resistance/susceptibility.

Effect of temperature and the protein-for-protein hypothesis. Vanderplank (11, pages 20–82; 12, pages 96-112; 13, pages 92–96) has proposed a protein-for-protein hypothesis in which host protein and pathogen protein polymerize specifically in associations that result in susceptibility. Vanderplank (13, page 93) indicates that the hypothesis is built on three pillars: "specificity in gene-for-gene systems is in susceptibility; the relevant molecules store variation massively; and susceptibility is endothermic in gene-for-gene systems." We will not discuss the merits of Vanderplank's protein-for-protein hypothesis on a biochemical basis. We do, however, question two of the three stated pillars of the hypothesis. We have shown above that parasite:host specificity is not always manifested in susceptibility. We also question the hypothesis of "endothermic susceptibility." The hypothesis of endothermic susceptibility indicates that susceptibility is greater with increased temperature in all cases. This does not hold with all available evidence. This concept (11, pages 45–57; 12, pages 97–102; 13, pages 94–96) is based largely on data from the cereal rust systems. In the first discussion (11), negative evidence is cited, but rejected. Gassner and Straib (5) found that the wheat cultivar Malakof inoculated with a culture of *Puccinia recondita* and grown at 6 C produced an infection type "4," while the same biological materials grown at 19 C produced an infection type ";". Infection type "4" is commonly interpreted as susceptibility and infection type ";" is commonly interpreted as resistance. They are, in fact, phenotypes of the parasite:host association (1,7). Vanderplank concluded (11) that the effect Gassner and Straib attributed to low temperature was due to "senescence" of the leaf tissue. He rejected Gassner and Straib's data because it was obtained under "highly artificial" conditions and because time from infection to full development of susceptibility differed between the cultivars. Further evidence (2,6) has indicated that Gassner and Straib's data (5) and conclusions

TABLE 2. A subset of the international system of designating interrelationships of *R* genes of potatoes and races of *Phytophthora infestans*[a]

	Race and putative genotype:			
Host genotype:	0 *V1V2*	1 *v1V2*	2 *V1v2*	1,2 *v1v2*
r1r1r2r2	Susceptible	Susceptible	Susceptible	Susceptible
R1R1r2r2	Resistant	Susceptible	Resistant	Susceptible
r1r1R2R2	Resistant	Resistant	Susceptible	Susceptible
R1R1R2R2	Resistant	Resistant	Resistant	Susceptible

[a] A subset of a table from Vanderplank (13, page 8) with complete genotypes at two host loci shown and putative genotypes of each race added. *R* and *r* symbolize alternate alleles at loci 1 and 2 in the host; *V* and *v* symbolize the alternate alleles at the two corresponding loci in *Phytophthora infestans*.

were correct. In some interactions, susceptibility is greater with decreased temperature. We believe that a better explanation of Gassner and Straib's data is that specific parasite:host genotypes function together to bring about specific phenotypes (1). In this case the phenotype results in resistance. In two subsequent discussions, Vanderplank (12,13) cites data that support his hypothesis of endothermic susceptibility but ignores data to the contrary.

Conclusion. We think Vanderplank's generalizations that specificity always resides in susceptibility and that susceptibility always increases with increasing temperature are not warranted from available evidence. We do see the importance of generalization when the generalization is not refuted by available evidence. Any sound generalization that we can derive concerning parasite:host specificity must treat resistance/susceptibility as a separate concept. Thus, any statement that will apply to parasite:host systems generally cannot include host resistance/susceptibility or the related concept of parasite avirulence/virulence. The concepts and terms of resistance/susceptibility and avirulence/virulence can only be related to specificity within stated parasite:host systems. Loegering (7) has proposed the generalization that parasite genotype and host genotype come together to form an aegricorpus and the phenotype is aegricorpus phenotype rather than parasite phenotype or host phenotype. He (7) has suggested the modifying term "definitive" to indicate genotypes and phenotypes that can be specifically related one to another. We would extend his generalization to the statement: A definitive parasite genotype and a definitive host genotype come together to form a definitive aegricorpous genotype; the definitive aegricorpus genotype functions only in definitive environment to result in a definitive aegricorpus phenotype (1). The definitive aegricorpus phenotype results in resistance in some cases, but susceptibility in others. This generalization can be applied to any parasite:host system. Resistance/susceptibility is man's perception of hosts which are useful or not useful in agriculture. Thus, resistance/susceptibility follows no relation with genetic specificity; specificity is a characteristic of parasite:host systems.

Any attempt to force a relationship between the two concepts may quickly lead to very faulty experimental designs in efforts to understand the biochemical basis of resistance/susceptibility within a given system that results in plant disease, and in other important experiments in plant pathology.

A practical application of parasite:host specificity is the development of resistant cultivars. Regarding specificity, resistant cultivars can be developed by two means: the isolation and cultivation of definitive host genotypes that, together with extant definitive parasite genotypes, lessen disease development; and elimination of definitive host genotypes that together with extant parasite definitive parasite genotypes increase disease development.

The method of using host genotypes that function together with extant parasite genotypes to lessen disease development has been widely used to control cereal rusts and many other diseases. This method, if we are to judge by plant pathology literature, has fallen into disfavor in recent years. Notwithstanding this, the method is currently being used very widely; its usefulness is related to how it is used rather than the fact it depends on parasite:host specificity and the nature of that specificity (10). The method of eliminating host genotypes that function together with extant parasite genotypes to increase disease development has also been very successfully used in disease control. This has been done in the cases of Victoria blight of oats, Milo disease of sorghum, and Southern corn leaf blight. These cases have been cited as examples of long-term control of plant disease by simply inherited resistance (11, pages 136-142). We suggest this control was due to elimination of simply inherited host factors relating to susceptibility. Elimination of host genotypes related to specific susceptibility is not equivalent to using resistance based on nonspecificity. Both methods of disease control depend on differences in parasite:host specificity; the specificity is used in different ways.

Forcing an absolute relationship between specificity and resistance/susceptibility precludes considering the possibility that there may be both genetic specificities that result in increased susceptibility and other genetic specificities that result in increased resistance within the same parasite:host system (7). Concentrating on only one view of specificity within one system could lead to neglect of specificity important in disease control.

Our prospects of advances in disease control through both conventional plant breeding methods and methods of molecular genetics are great. Realization of these prospects must not be hampered either by forming faulty generalizations or by restricting our viewpoints to one or a few parasite:host systems.

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