Letter to the Editor

Where Is the Specificity in Gene-for-Gene Systems?

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The recent letters to the editor (1,16) concerning resistance/susceptibility and specificity of host-parasite interactions especially in gene-for-gene systems have helped clarify thinking on these subjects. The basic disagreement is whether or not there is specific resistance in gene-for-gene systems. The statements of Vanderplank (15,16) that resistance is never specific are not supported by the literature. Ellingboe has used the term recognition for incompatibility as opposed to recognition for compatibility in gene-for-gene systems and provided compelling arguments in support of the former hypothesis (3–5). I am equating specific resistance and recognition for incompatibility in this discussion.

The genetic basis of the specific resistance and specific susceptibility question can be most easily understood by referring to the quadratic check (Fig. 1A) that occurs in gene-for-gene systems (10). One interpretation of the quadratic check is that the dominant genes, *R* in the host and *A* in the parasite, interact to condition specific resistance in the upper left quadrant. This is termed recognition for incompatibility. The remaining three quadrants are nonspecific because one or both of the necessary gene products or sites for recognition in the interaction are presumed absent (3–5). The interpretation of Vanderplank differs in that he hypothesizes the protein gene products polymerize to condition specific susceptibility in the three compatible quadrants (15, pages 93 and 99). The explanation for the upper left quadrant for incompatibility or vertical resistance is "that the host protein and pathogen protein fail to associate, or if they associate, fail to complete the development of the appropriate quaternary structure" (15, page 95). Vanderplank's explanation for the upper right quadrant involves the polymerization of "a more hydrophobic host protein and a less hydrophobic pathogen protein" (15, page 100) than those in the lower left quadrant. The lower right quadrant, which Vanderplank did not discuss explicitly, would have an interaction of the most hydrophobic host (*rr*) and pathogen (*aa*) proteins. Based on Vanderplank's interpretation, one might predict the lower right quadrant to exhibit a third class of increased specific susceptibility compared with the other two compatible quadrants. This situation does not appear to be supported by available evidence. In fact, reduced compatibility in some lower left quadrant interactions when compared with the upper and lower right quadrants is reported (12), but this has been attributed to residual or ghost effects of the *R* genes.

Literature in support of specific susceptibility exists from many host-specific toxin systems as depicted (Fig. 1B) for *Helminthosporium victoriae* in Victoria blight of oats (3). The dominant *Vb* gene in the host confers sensitivity to the HV-toxin and susceptibility to the disease. The ability to produce or not produce the toxin is under apparent single gene control (13). Interactions in the other three quadrants result in resistance because either the toxin or the *Vb*-encoded sensitive site or both are not present. This is a classic case of recognition for compatibility or specific susceptibility. I do not consider these systems comparable to the gene-for-gene systems involving biotrophic fungal pathogens.

The cultivar-race examples given or alluded to by Vanderplank (stem rust of wheat, flax rust, powdery mildews of wheat or barley, and potato late blight) clearly involve gene-for-gene systems in which there are many loci, some with allelic series in the host for resistance to various races of the pathogen. These systems are not recognized to involve pathogens that produce host-specific toxins. It is these kinds of gene-for-gene systems, not host-specific toxin systems, to which the arguments supporting specific resistance and refuting specific susceptibility will be addressed.

One argument in support of specific resistance comes from Flor's study of inheritance of X-ray-induced mutations in race 1 of *Melampsora lini* (6). He gives convincing evidence that mutation of the avirulence gene controlling reaction to the flax (*Linum usitatissimum* L.) cultivar Koto results from deletion. In the absence of the avirulence gene, a compatible or susceptible host-parasite interaction occurred.

The implication is that the absence of the locus in the parasite that controls the interaction results in virulence, that is, absence of a functional gene product is equated with virulence. This is the basis of the concept of mutation to wider virulence. Flor's concluding paragraph to the paper needs no interpretation: "In host-parasite relationships of the rusts, resistance usually results when the reciprocal or complementary genes in both host and parasite are dominant. Susceptibility usually results when either or both are recessive. These facts suggest that the dominant members of a pair govern the production of substances that are mutually antagonistic or act as antimetabolites. *The evidence that virulence is the result of the deletion of the dominant avirulence gene supports this hypothesis and indicates that it is some inhibitive function of the dominant resistance-avirulence genes that characterizes physiologic specialization in the flax rust fungus.*" (Emphasis mine). This statement directly refutes Vanderplank's contention (16) that Flor was "consistent and unequivocal" about specificity for pathogenicity rather than avirulence.

Additional arguments involving the epistasis of gene pairs specifying incompatibility over gene pairs specifying compatibility (4), the *Sr6*/*P6* temperature-sensitive interaction in stem rust of wheat, and the failure of L2-L10 recombinants in flax to recognize appropriate rust isolates are discussed by Ellingboe (3) and support the concept of recognition for incompatibility.

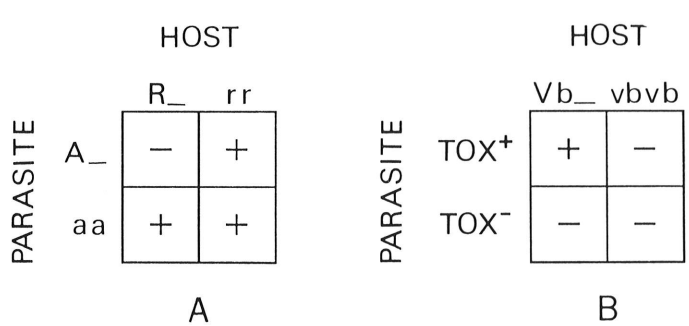

Fig. 1. The quadratic check comparing the interaction of near-isogenic hosts and parasites characteristic of many gene-for-gene systems (**A**) and characteristic of host-specific toxin systems as indicated by Victoria blight of oats (**B**). (−) = incompatible, (+) = compatible.

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Further evidence in support of recognition for incompatibility or specific resistance comes from the work of Staskawicz et al (14) with the putative gene-for-gene system (2); *Pseudomonas syringae* pv. *glycinea*/ *Glycine max*. When cosmid clones of race 6 genomic DNA were transconjugated into three other races, the host range incompatibilities, not compatibilities of the wild type race 6, were mimicked. Race specificity on differential hosts by transconjugants always changed from virulent to avirulent in line with race 6's pattern. The change from avirulent to virulent, as one would expect with specific susceptibility, was not observed, although opportunities for that expression were present in the experimental design. The failure to produce the race 6 virulence pattern in the transconjugants argues strongly against Vanderplank's specific susceptibility hypothesis.

Similar results have been obtained by Gabriel using the *Xanthomonas campestris* pv. *malvacearum*/ *Gossypium hirsutum* system (9). Five different cloned *A* genes, each interacting with a specific *R* gene in cotton, has provided a clear demonstration of Flor's gene-for-gene hypothesis.

The previous examples have dealt with genes in the parasite that interact with or recognize genes in the host and result in specific resistance. Loegering and Sears (11) have presented evidence that aneuploids of wheat that have lost the locus-conferring resistance to the appropriate races of *Puccinia graminis* f. sp. *tritici* behave as if they had an "allele" for susceptibility. They conclude that alleles for susceptibility may, in some cases, be the result of a nonfunctional or a noninteractive DNA sequence in euploids. Again, resistance requires the physical presence and apparent expression of the dominant allele in the host. The recessive allele need not be physically present to result in susceptibility. The infection type was the same whether the recessive allele was present or deleted. How could the recessive allele then actively specify susceptibility? From where is the hydrophobic host protein gene product coming? Vanderplank (15, page 101) has interpreted this as "in the susceptible aneuploid lines the pathogen was living on the products of the remaining 39 or more loci." He goes on to state "A 1/40 or 2.5% loss of food would have been undetectable in the experiments of Loegering and Sears." The obvious question then is why does the pathogen fail to develop in the euploid if the *Sr* genes for resistance are not actively recognized? After all, the other 39 loci are still present to feed the pathogen as Vanderplank suggested they did in the aneuploid.

There appear to be genes in the host and parasite that condition basic compatibility (3) or the ability to be a parasite or a pathogen (7,8), but these are not the genes specified in gene-for-gene systems upon which Vanderplank has built his case. In conclusion, I am not aware of any convincing evidence in support of Vanderplank's hypothesis of specific susceptibility caused by gene-for-gene interactions (15,16) comparable to the evidence that exists for specific recognition for incompatibility or specific resistance.

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