Letter to the Editor

A Generalized Concept of Host-Parasite Specificity

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Although the basis of host-parasite specificity has been the subject of much investigation, discussion, and controversy among plant pathologists, there are few plant disease interactions in which this phenomenon is unequivocally understood (8). Over the last 10 yr, my work with the rust fungi has gradually led me to believe that there are two types of pathogen specificity and that these must be conceptually distinguished before physiological investigations are begun. The first is plant species specificity (determining host species range), and the second is cultivar specificity (determining cultivar range within a given host species). To me, the principles involved in establishing these two types of specificity are quite different, and it is possible that host-pathogen interactions controlling each type may coexist in the same tissue. Much of this concept is not original or new (most of it has been published in fragmented form elsewhere [5,7,9,10,11,15]) but it is presented here in a more coherent form in the hope that it will be more widely understood and discussed. This letter is primarily concerned with fungus-plant interactions, but it may prove that the concept is more widely applicable. A diagram of the postulated events leading to species and cultivar specificity is shown in Fig. 1.

Nonhost resistance. In some plant species, resistance to certain fungal plant pathogens may be explained by the presence of preformed physical or chemical factors (16). However, other examples of nonhost resistance (ie, resistance shown by plant species not considered to be hosts for the pathogen in question) seem to depend on active plant responses (9), presumably triggered by some fungal factor or activity. In such cases, it seems unlikely that each plant has a specific and different gene to govern the recognition of each of the thousands of potential fungal pathogens (18,19); more probably, each plant species has evolved, not only a range of preformed potential "deterrents" to infection, but also a small battery of nonspecific defense reactions of which one or more are almost certain to be triggered if invasion is attempted (9). One might predict that the triggers (elicitors) of these reactions are similarly nonspecific and are likely to be surface-bound or secreted products common to many pathogens. Such a hypothesis is supported, for example, by the fact that fungal cell-wall components often seem to be relatively indiscriminate elicitors of antimicrobial phytoalexin accumulation (eg, 1 and references therein).

Basic compatibility. Regardless of the mechanisms of nonhost resistance, the ability of a fungus to successfully parasitize a plant must be due to the *specific* "accommodation" of the pathogen to its host that renders these defense factors or reactions ineffective (10,18). The theoretical means for achieving such "basic compatibility" (5) between pathogen and host species are legion (10). "Passive" accommodation by the pathogen could involve the development of tolerance to preformed or induced antimicrobial compounds; more "active" accommodation could rely on the production of an enzyme to modify these compounds to nontoxic derivatives (10,16). Other forms of active interference with resistance for which there is some experimental evidence include the production of host-selective toxins, which can be regarded as killing the cells before resistance can be expressed (3), and the secretion of fungal blockers (suppressors) which prevent specific defense reactions from taking place (12,14). By such active interference with resistance, the pathogen is effectively "inducing susceptibility" in its host species, and it is important to realize, as discussed later, that there is no theoretical or experimental argument against such a phenomenon being involved in establishing *basic compatibility*, as long as it is distinguished from cultivar specificity (10). Moreover, there is no reason why the pathogen, in addition to negating the nonspecific defense reactions of its host, cannot also promote certain metabolic changes which are to its advantage (3,4).

Whatever mechanisms are involved, the significant feature of the concept of basic compatibility as presented here is that the highly specific interactions between host plant and parasite occur in the compatible (host), not the incompatible (nonhost), situation. This explains why a given fungal pathogen rarely shows any marked

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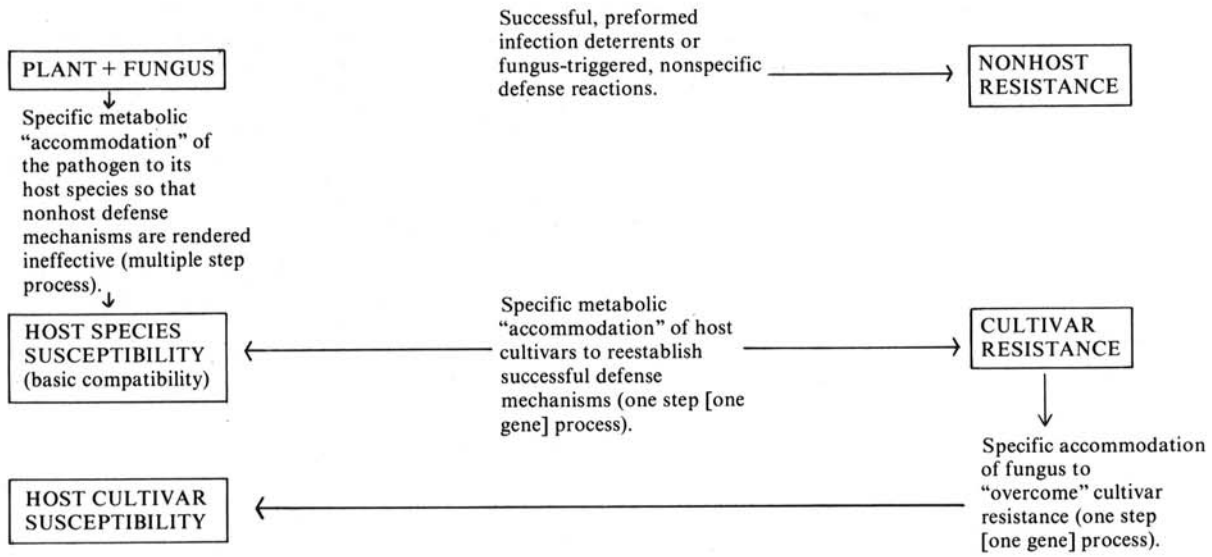

Fig. 1. Postulated events leading to species and cultivar specificity of a fungal pathogen.

change in host species range; presumably random mutation cannot easily generate the necessary specific "accomodation" of the pathogen, particularly if more than one defense reaction or feature of the plant has to be contended with.

Cultivar resistance. Once a basic compatibility has been established between a pathogen and its host species, the host is now susceptible to successful infection. Consequently, there must be a strong selection pressure on the host to evolve some subsequent form of "resistance" to reduce the adverse effects of pathogenesis. An individual plant that achieves this by random mutation or the assistance of a plant breeder, becomes a "resistant cultivar." Like basic compatibility, cultivar resistance theoretically could be achieved in a multitude of ways, but in contrast, it involves a specific "accommodation" of the *host* rather than the pathogen. For example, in the resistant cultivar, activities of the pathogen that normally induce susceptibility may be negated, perhaps in a manner analogous to the degradation in resistant bean cultivars of the extracellular polysaccharide of *Pseudomonas phaseolicola* which would otherwise cause water-soaking of the tissue (4). In other cases, the receptor site for a host-selective toxin could be eliminated or modified, rendering the toxin ineffective and allowing nonspecific "nonhost-type" interactions to be triggered nonspecifically. Yet another possibility is that active defense reactions could be *specifically* elicited by some fungal product or activity, but the onus is on the host to "find" some fungal feature that it can "recognize" for this purpose. In such a situation, it is easy to see how the characteristic gene-for-gene relationship between plant and pathogen becomes established; the gene controlling fungal recognition automatically becomes the gene for resistance, while the fungal gene controlling the production of the recognized feature is now the gene for avirulence. The fungal product is now also a "specific elicitor" of resistance since it is only active in individuals bearing the relevant gene controlling recognition. However, there is no reason why this elicitor should have been initially designed to condition avirulence (15) or have an important role in pathogenesis (10). Nevertheless, one could imagine a situation in which the host "recognizes" a blocker involved in determining basic compatibility, thus allowing nonhost-type defense reactions to be triggered unhindered (2,10). It is equally possible, however, that some other fungal feature could be recognized; thus, recognition could result in events differing from those involved in "nonhost-type" resistance (11).

The prediction from this "one-step," gene-for-gene type of development of cultivar resistance is that it may be rather easily "overcome" by the pathogen through random one-step mutations which could, for example, render the elicitor no longer recognizable by the product of the host gene for resistance; this prediction is supported by the common development in the field of new, virulent races of the pathogen, and the relative ease with which pathogens can mutate from avirulence to virulence in the laboratory (16). In contrast, random mutation might be expected to derive host genes for resistance with a relatively low frequency if the gene product has to *specifically* interfere with some fungal activity, but higher frequencies might be predicted if cultivar resistance is not based on specific recognition of the fungal product (eg, the nonspecific modification of a receptor site for a fungal toxin). Significantly, mutagenic treatments may (17) or may not (eg, 6) easily generate new forms of cultivar resistance.

Conclusions. The idea that cultivar resistance is superimposed on a basic compatibility between the parasite and its host species was first suggested from genetical considerations of host-pathogen specificity (5,15), but the biochemical and physiological implications of such a concept seem to have been generally ignored. Most importantly, the concept suggests that the genetic constraints placed upon the proposed physiological basis of cultivar specificity do not apply to basic compatibility for which there is as yet no genetic information. Thus, the establishment of basic compatibility may involve active or passive features of the pathogen and there is no need to suggest (13) that because "secondary" cultivar susceptibility is related to the absence of a "definitive allele" (and therefore by implication, to the absence of an active process) in the pathogen, basic compatibility must be determined in a similar manner. Furthermore, if host species compatibility is determined by the specific metabolic accommodation of the pathogen, and cultivar resistance involves the subsequent, equally specific, metabolic accommodation of the host, the mechanisms controlling both the species and the cultivar range of each pathogen have to be unique in detail. Thus, generalizations from one system to another are unlikely to be valid, except in the broadest terms.

Another important implication of this concept is that if basic compatibility involves the active participation of the pathogen, such as the secretion of blockers of defense reactions or the detoxification of otherwise toxic plant metabolites, then these processes may continue in the resistant cultivar in the presence of other processes specifically determining this resistance. For example, it would be theoretically possible for a defense reaction such as phytoalexin accumulation to be specifically inhibited during the establishment of basic compatibility, and specifically triggered during cultivar resistance, resulting in specific inhibitors and elicitors being present in the same tissue (10). Obviously, in this and other easily conceived situations, the physiologist would find it extremely difficult to determine the basis of cultivar resistance without some knowledge of the processes involved in establishing

basic compatibility; nevertheless, cultivar resistance has been studied to the relative exclusion of species specificity. If the concepts described here prove to be correct, this lack of emphasis on host species specificity may, at least in part, explain why the basis of cultivar resistance, and host-pathogen specificity in general, are still so poorly understood.

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Editor's Note: This Letter to the Editor should be read and considered in conjunction with the Letter to the Editor entitled "Suppressors of defense reactions: A model for roles in specificity" by W. R. Bushnell and J. B. Rowell (Phytopathology 71:1012-1014, 1981).