Letter to the Editor

Suppressors of Defense Reactions: A Model for Roles in Specificity

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Heath (10,11) has summarized the significance of basic compatibility in relation to host-parasite specificity. In her view, the parasite that can cause disease in a given higher plant species has the ability to avoid or to negate the consequences of general defenses, both preformed and induced. Such a parasite is basically compatible with its host and thus has established compatibility at the species level of specificity. Only after basic compatibility is established can specificity at the race-cultivar level develop. Heath justifiably concludes that the events controlling specificity at the two levels probably cannot be understood separately.

One of the devices that could determine specificity at one or both levels is the suppression of general defense reactions. Initially postulated to suppress phytoalexin production (15,18) suppressors have recently been implicated in specificity at the species level in rust fungus-higher plant combinations (8,9) and at the race-cultivar level in Phytophthora-higher plant combinations (2–4,7). Although emphasizing that suppressors are only one of many possible means for controlling specificity, Heath (10) presented a model which showed how suppressors might operate at either the species or race-cultivar level. The model followed the principles expressed briefly by Ward and Stoessl (19).

We independently developed a model which was similar to Heath’s in several respects. Our purpose was to explore the possibility that suppressors might be involved in the specificity of rusts and powdery mildews. Like Heath, our view of specificity was influenced by the immunity of nonhost species to parasitic fungi (14,16) so that our model was based on the assumption that specificity is controlled by suppression of general defense responses. Our initial model was refined in consultation with Heath, builds upon her earlier version, and helps to conceive the nature of specificity (Figs. 1–3).

With respect to race-cultivar specificity, the model is based on the generally accepted gene-for-gene theory in which single corresponding genes in host and parasite condition incompatibility (5). With respect to species specificity, the model is based on Hogenboom’s theory of incongruity in intimate partner relationships (12,13) as adapted for host-parasite systems (1). This theory predicts that compatibility is conditioned by corresponding dominant genes in host and parasite, and grew out of consideration of specificity at the species level in pistil-pollen systems. Although some genetic evidence exists for this theory in pistil-pollen systems, very little evidence exists for corresponding genes that condition basic compatibility in host-parasite systems (5). Although conditional mutants (sensitive to high temperatures) indicate that the parasite Colletotrichum lindemuthianum has genes that
condition basic compatibility (6), there is no evidence yet that corresponding genes exist in the host. Nevertheless, Hogenboom's theory provides a simple cognitive scheme for coevolution of host and parasite and the consequent development of species-level specificity.

The model assumes that many species of fungal pathogens have in common one or more secreted or wall-bound substances that elicit defense responses in higher plants (Fig. 1). The responses most commonly elicited are production of phytoalexin, hypersensitive cell death, walling-out phenomena, and combinations thereof. Such defenses are assumed to be elicited nonspecifically in combinations of parasites with nonhost species by binding of the elicitor to a receptor in the nonhost. The model further assumes that parasites produce species-specific suppressors which prevent the nonspecific elicitors from acting. Specificity is conferred by a series of corresponding sites on suppressor and receptor which must all match if the suppressor is to fit the receptor and so be effective in suppressing elicitor action (Fig. 2A). Coevolution of the matching sites (Fig. 2A-C) is assumed to follow Hogenboom's theory and results in the acquisition of corresponding dominant genes which condition compatibility. These genes condition the matching sites on suppressor and receptor.

A distinctive feature of Hogenboom's theory, as we have applied it to host-parasite systems, is that the coevolutionary process is driven by environmental factors other than the parasite; ie, external factors which lead to changes in host structures or activities. For example, a change in habitat might lead to a change in a part of the host which serves as a receptor in a basically compatible host-parasite combination (Fig. 2B). The parasite must modify its suppressor to successfully parasitize the modified host (Fig. 2C). A series of such changes leads to a new species of host which is basically compatible with a new *forma specialis* (or species) of parasite. Several such suppressors could exist in a given host-parasite combination.

Structural and chemical factors other than suppressor-receptor sites are postulated to evolve in accordance with Hogenboom's theory. Thus, environmental change might lead to a change in leaf surface structure or composition which is unsuitable for differentiation of an existing compatible parasite. The parasite would have to develop a corresponding capacity to differentiate on the new surface. Many such accommodations allow the parasite to coevolve with the host; the matching of suppressors to receptors is among these accommodations.

To invoke suppressors in race-cultivar specificity, we postulate that individual structural sites conditioned by single genes exist on the suppressor, but that these sites are initially unrelated to receptor fit (Fig. 3A). However, a change can occur in the host receptor such that it no longer fits the suppressor in the region of one of the preexisting gene-specified sites (such as site R in Fig. 3B). The gene which conditions the new receptor site in the host is a gene for resistance. The gene which specified the suppressor site in the parasite would then be considered to be a gene for avirulence. Finally, the parasite stops producing the specific suppressor site through a change to virulence, restoring compatibility between host and parasite (Fig. 3C).

We envision that the host with no genes for resistance (the universal suscep) would have receptor configurations as in Fig. 1. Relation between elicitor and receptor of parasite and nonhost. Incompatibility is elicited by one or more fungal products, secreted or wall bound, common to many species of fungi. The elicitor binds to a receptor common to many higher plant species. The incompatibility reaction that is triggered is one of a battery of defenses used to nonspecifically repel most potential invaders.

![Fig. 1](image1)

![Fig. 2](image2)

**Fig. 2.** Relations between elicitor, suppressor, and receptor in the coevolution of basic compatibility between host and parasite. Each designated site on suppressor or receptor is conditioned by a single gene in host or parasite. A, Parasite X' is basically compatible with host species X. The parasite produces a species-specific suppressor which prevents elicitor from binding to receptor so that nonhost defenses are not triggered. Species specificity is conditioned by a series of dominant genes that condition corresponding sites on suppressor (A', B', etc) and receptor (A, B, etc.). Suppressor and receptor fit only if all sites are matched. (P1 and P2 are structural sites involved in race-cultivar specificity.) B, A change occurs in the receptor at site C in the course of evolution of host species Y from host species X. The change is caused by factors other than the parasite. Site C interferes with suppressor attachment to receptor. In the absence of suppression, the elicitor triggers a nonhost defense. C, The parasite adapts to host species Y by producing site C' on the suppressor which matches site C on the receptor. Parasite Y' is then an appropriate *forma specialis* or species for host species Y. A series of such changes produces several corresponding dominant genes in host and parasite, which condition the corresponding sites on suppressor and receptor (following Hogenboom's theory of incongruity in intimate partner relations).

![Fig. 3](image3)

**Fig. 3.** Relations between elicitor, suppressor, and receptor in the coevolution of race-cultivar specificity. A, Initially, host and parasite are basically compatible as in Fig. 2A. The parasite has genes which condition sites P1, P2, etc. on the suppressor which are initially unrelated to the receptor and do not interfere with suppressor–receptor fit. B, Host acquires a gene which conditions site R, which interferes with suppressor–receptor fit at P1. Elicitor then triggers defense reaction. C, Parasite counters by removing P1 so that P1 no longer interferes with fit. Suppression is restored; host and parasite are compatible. A similar sequence can occur at P2 and at additional sites.
and that the parasite with no genes for avirulence (universally virulent) would have a suppressor which lacks all the structural sites such as P1 and P2 of Fig. 3 that have the potential of interfering with receptor-suppressor fit. With multiple alleles for resistance at a locus in the host, we envision that each allele conditions small portions of a complex receptor site such that each portion can interfere with suppressor sites conditioned by different genes for avirulence in the parasite.

What is the origin of the suppressor sites assumed to prevent suppressor-receptor fit and to be conditioned by genes for avirulence? If the suppressor has no function other than suppression of a defense reaction, extraneous features unrelated to suppressor activity would be unlikely. We postulate, instead, that suppressors originate through modification of preexisting fungal molecules (probably macromolecules) and that the sites which are conditioned by genes for avirulence have functional, but not essential, roles in the parasite, as others have speculated (10,17).

That general defenses are suppressed when host and parasite are basically compatible is a way of saying that the susceptibility is induced (11). When compatibility is restored in race-cultivar specificity by appearance of a gene for virulence (Fig. 3C), the ability to induce susceptibility is restored. Thus, the corresponding genes that condition avirulence and resistance lead to the formation of substances which negate the production of susceptibility. Although the result is negative, it is determined by products of gene action, in line with the fact that the genes for avirulence and resistance are usually dominant.

The model as presented shows suppressor binding to host receptor, in line with indirect evidence that suppressor binds to host membranes (2). A more general model would allow for the alternative that suppressor binds to elicitor. The model as presented also suggests that the suppressor and receptor molecules must be large enough to contain several structural sites conditioned by single genes. These sites could originate, for example, from point mutations which condition localized parts of proteins, or from mutations which condition glycosyl transferases which, in turn, determine terminal glycosyl configurations of glycoproteins. However, the model is not intended to set limits on the types of molecules that might be involved or to specify the relative sizes of elicitor, suppressor, and receptor.

As illustrated in Fig. 3, the sites controlling race-cultivar specificity are independent of the sites controlling basic compatibility. However, the changes that interfere with suppressor-receptor fit in race-cultivar incompatibility (Fig. 3B) could be modifications of one or more of the specific sites required for basic compatibility. The possibilities would depend on the structural nature of the sites.

The suppressor is certainly not the only device that enables a parasite to avoid nonhost defenses, or through which race-cultivar specificity is determined. Indeed, the available evidence is not convincing that suppressors have a role at any level of specificity. Nevertheless, we join Heath in the idea that the mechanisms controlling species level and race-cultivar specificity occur concomitantly. By showing that suppressors could be operating at either or both levels of specificity in a manner consistent with the known genetics of host-parasite specificity, the model lends support to experimental efforts to find such suppressors.

LITERATURE CITED


