Letter to the Editor

Hypersensitivity, the Cause or the Consequence of Rust Resistance?

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Since its conception as a descriptive term for the rapid, but limited, cell death associated with some incompatible rust infections (11), the "hypersensitive response" has been widely applied to describe the rapid necrosis observed in many other types of incompatible host-parasite interaction. Whether such a response is the cause or the consequence of resistance has been the source of a controversy which, if anything, has increased in fervor over the last few years (4, 6). For rust infections, some of the most recently published research on this topic suggests that necrosis is not a primary determinant of certain types of varietal resistance (7, 8). The stated conclusion from this work, however, is that "necrosis or cell collapse is not a determinant of incompatibility with *rust disease*" (7, my italics). What I find disturbing about such a statement, and about the whole controversy, is the implication that what may be true for one or two types of interaction, must automatically be true for all. Since my work (3), among that of others, has been quoted as centering on the hypersensitive response as a cause of rust resistance (8), I should like to add my thoughts, and warnings, on this thorny topic.

If one looks at the published descriptions of host and nonhost resistant responses to various rust infections, a conspicuous feature (to me at least) is that each one differs from the others in one or more ways, particularly at the ultrastructural level. Until we know the significance of these differences, it seems to me to be extremely unwise to regard each host-parasite interaction as anything other than unique. Indeed, the fact that host specificity exists, confirms the presence of unique features in each host to which the pathogen has had to adapt. Thus, it is not only quite conceivable, but even quite likely, that hypersensitivity may play a different role in different resistant responses; it also is equally probable that some plants may be resistant without evoking the hypersensitive response, as has been demonstrated in several instances (e.g., 2), but this in no way belies the importance of such a response in other situations.

The second point that I should like to emphasize is one already pointed out by Littlefield (5); that hypersensitivity is not necessarily a single phenomenon. There is ample evidence from other pathogenic situations to suggest that there is more than one way in which a cell may die (12) and there are reasonable indications that diverse types of cell death may be found among different types of rust resistance. This can be demonstrated rather superficially by the fact that different resistant responses often show different visible coloration of their necrotic cells. At the ultrastructural level, I have found cases of cultivar rust resistance where the extremely rapid, and apparently simultaneous, disorganization of both the invaded cell and its haustorium suggests that host cell death is associated with the formation of some fungitoxic substance (1); conversely, I also have observed other interactions in which a different type of cell death is seemingly involved since the equally rapid disorganization of the invaded cell does not result in the immediate death of the haustorium (Heath, *unpublished*). In all of these interactions, cytoplasmic disorganization does not seem to be a result of preparative procedures for electron microscopy (as suggested in 7) since browning of invaded cells can be observed in fresh sections cut from living tissue.

In those examples where the rapid death of both the invaded cell and the *first-formed* haustorium is followed by no further fungal growth (1), I find it hard to believe that such necrosis does not play an important role in resistance, even though at least one of the cultivars showing this response possesses another, equally effective, method of restricting fungal growth if necrosis does not take place (3). Admittedly, in those instances in which hypersensitivity does not result in any immediately visible effect on the fungus, its role in resistance is less clear and may be of secondary importance. However, it should be borne in mind that host necrosis may affect fungal growth in rather subtle ways. For example, the haustorium is generally assumed to take up essential materials from the host cell, although there is some evidence that these materials may not be just simple nutrients (10). Presumably, the haustorium begins to function soon after its formation, and a delayed "nontoxic" necrosis of the invaded cell could allow the passage of enough of these materials to support a limited amount of intercellular growth. The prime factor in determining the extent of such growth would be the time interval between haustorium formation and the onset of necrosis, not the number of necrotic cells. In fact, one would expect an increase in fungal growth to result in an increase in necrosis since more cells would be invaded by haustoria. When viewed in this light, the observed lack of an inverse correlation between numbers of necrotic cells and fungal growth does not present as good a case against the importance of hypersensitivity in these interactions as has been suggested (9).

Rust resistance is obviously a complex phenomenon and the hypersensitive response, even if a primary determinant in restricting fungal growth, may only be the visible product of a number of previous interactions between the plant and its potential pathogen. The purpose of this letter, therefore, is not to advocate the importance of the hypersensitive response in rust resistance but to plead for a close, unbiased look at each individual system from as many angles as possible. Even though my remarks are directed specifically toward work with the rusts, this plea, I feel, is equally applicable to all types of resistant responses to plant pathogens.

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