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Resistance of Plants to Rust Infection

Michèle C. Heath

Associate professor, Botany Department, University of Toronto, Toronto, Ont. M5S 1A1 Canada

For 40 yr or more, resistant cultivars have been used as the primary means of controlling rust diseases in crop plants (eg, 5). Yet, in spite of two decades of physiological and biochemical investigations, we still have no clear understanding of the nature of host resistance or the mechanisms that protect nonhost plants (ie, species considered not to be a host for the rust fungus in question) from successful rust infection. The reasons for our current ignorance are legion, and include the fact that physiological and biochemical investigations of resistance require an "educated guess" as to what phenomenon should be investigated and, by hindsight, it seems that the choice of research approach often was influenced more by available techniques and the vogue of the day than by direct evidence that the phenomenon selected for measurement was important in plant resistance.

Only recently has it begun to be widely appreciated that cytological studies can provide valuable information about what phenomena may be the most promising to investigate physiologically. Therefore, in this paper, I should like to discuss my own interpretation of what morphological investigations currently tell us about the nature of resistance to rust fungi, and of the determinants of host specificity. Since space is limited, most examples will be drawn from my own work; more comprehensive reviews of the cytological literature can be found in Littlefield and Heath (21) and Heath (15).

Prehaustorial resistance. The value of cytological data for extending our understanding of plant resistance depends on how easily, and accurately, these data can be interpreted. In my opinion, some of the best data in this respect come from interactions in which resistance is expressed before the first haustorium is formed. Typically, such resistance is exhibited by nonhost plants (15) and in several instances it seems to result in reduced penetration of the fungus into the tissue. For example, urediospores of the cowpea rust fungus (*Uromyces phaseoli* var. *vignae*) germinate very poorly on leaves of cabbage, pea, and tomato, and although this probably reflects the unwettability of the waxy cabbage and pea leaves, it seems possible that tomato leaves possess an inhibitor of germination (8). However, even on nonhost plants, reports of reduced germination are rare and, more commonly, urediospores germinate well but the germ tubes appear to have difficulty in locating and recognizing stomata (8,10,34). For many rust fungi, the ability of the germ tube to find a stoma is governed by its response to the surface topography of the leaf (34); however, the molecular events that allow the germ tube to recognize and respond to physical features of the plant surface are essentially unknown. Nevertheless, "incorrect" prepenetration behavior and poor germination must, by reducing penetration frequency, significantly reduce the energy required for the more active responses elicited once the fungus enters the nonhost tissue.

Another common situation, observed both in host and nonhost plants, is the cessation of fungal growth during penetration into the plant through epidermal cells (eg, 25) or through stomata (eg, 8,10). While physical barriers have been implicated in some instances (eg, 25), physiological factors such as the presence, or the fungus-induced accumulation, of toxic materials have by no means been ruled out, and this type of resistance should be investigated further.

Many cytological studies reveal that after a fungus has entered the intercellular spaces of an incompatible leaf, adverse effects on the growth or appearance of the substomatal vesicle or infection hypha are noted. Even though this is more common in nonhost plants (8,10,20,33) it also has been reported in certain types of cultivar resistance (eg, 31). When dealing with biotrophic organisms such as the rust fungi, it is difficult to tell whether poor growth inside the tissue is due to growth inhibition or lack of stimulation (24); however, since infection structures of the cowpea rust fungus develop further on artificial membranes devoid of exogenous nutrients than they do in certain nonhost plants, it seems that in these latter situations true inhibition of growth is taking place (10). In fact, such inhibition may be a common

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phenomenon in nonhost plants since increased growth of infection hyphae comparable to that in susceptible tissue often has been observed after the plants have been heat shocked or exposed to inhibitors of RNA or protein synthesis (12,33); these results also imply that the inhibitors of fungal growth are either induced by infection or subject to metabolic turnover. The inhibiting factor involved in the resistance of Shokan 1 oats to the crown rust fungus (*Puccinia coronata* f. sp. *avenae*) is probably induced, since adverse effects on infection hyphae coincide with increased synthesis of messenger RNA and protein (32) as well as increased Golgi body activity in the plant (29).

Irrespective of whether infection hypha development is adversely affected at some infection sites, most infection hyphae in resistant host cultivars produce at least one haustorium. This is not the case for most nonhost interactions (8,10,20,33) and although in some situations the absense of haustoria may merely be a consequence of the poor growth of the infection hypha, in others, infection hyphae seem to grow normally until the time that a haustorium should be initiated (8,10). For such interactions involving the cowpea rust fungus, at least two phenomena have been identified by electron microscopy, which would account for the absence of haustoria. In broad bean, the layers of the fungal wall separate, apparently allowing the haustorial mother cell to become detached from the plant cell wall. This observation, plus indications of secretory activity of the adjacent mesophyll cell, point to the release of wall-degrading enzymes by the nonhost plant (8). In contrast, the same fungus in French bean leaves triggers the deposition of silicon-rich, electron-opaque deposits on and in mesophyll cell walls next to infection hyphae (7,8,11). These deposits seem to prevent the first haustorium from breaching the affected wall (7); further, any treatment that increases haustorium production also appears to decrease the silicon deposition reaction (11,17).

Posthaustorial resistance. The cytological data most difficult to interpret with respect to possible resistance mechanisms come from studies of infection types in which resistance is manifested after the first haustorium is formed. Usually such resistance is shown by cultivars of the host plant, and electron microscopy generally has supported the conclusion from transplant experiments (eg, 1) that the fungus has been inhibited rather than killed. Therefore fungistatic, rather than fungitoxic, phenomena must be involved, but this is not very informative considering the innumerable ways in which growth, particularly of a biotroph, might be slowed or inhibited. Since cultivar resistance is usually accompanied by morphological responses of the host plant, many cytological studies have focused on these in the hope that they might be directly involved in the restriction of pathogen development. What has emerged is that rust resistance may be morphologically expressed in many ways. The most common and obvious of these is host cell death, which probably is not the uniform phenomenon that it appears to be at first sight, and which differs ultrastructurally (and probably biochemically) in different interactions (9,21). Other host responses detected by light and electron microscopy include the deposition of fibrillar material in the extrahaustorial matrix (21,22), reduced incorporation of radioactive compounds from the host into the haustorium (22,26,27), changes in the appearance of the extrahaustorial membrane (18), the development of callose-containing collars around the necks of haustoria (18), and the subsequent encasement of the haustoria by continued synthesis of collar material (18).

In many cases, more than one of these events can be detected in a given host-pathogen interaction and the problem then becomes one of sorting out which, if any, of these responses is directly related to the cessation of fungal growth. What makes this problem especially difficult is that most of the observed responses are associated with the haustorium, and as yet there is no clear understanding of how impaired functioning of the haustorium affects intercellular growth. Indeed, we have no detailed knowledge of the normal role(s) of haustoria. Microautoradiography of compatible interactions supports the widely held notion that haustoria may take up nutrients that are then passed to the intercellular hyphae (27) but it also suggests that infection hyphae, at least, are capable of taking up simple nutrients before the first haustorium is formed (28). Possibly, the complex structural differentiation of the dikaryotic haustorium reflects a more specialized role than that of being a simple organ of nutrition (21). Nevertheless, the first-formed haustorium may die without causing the cessation of intercellular growth (30), although this is not the case in all interactions (eg, 7). Perhaps the role of haustorium death in resistance depends on whether, and for how long, the haustorium can function after it is formed, and also whether the host necrosis (which usually accompanies or precedes haustorium death [21]) elicits the accumulation of substances inhibitory to fungal growth. These substances (phytoalexins) may be operative in the types of resistance in which necrosis seems to be accompanied by a marked reduction in the growth rate of the fungus, as in the case of a necrotic-fleck type of resistance of French bean to the bean rust fungus (*U. phaseoli* var. *typica*). In this interaction, further evidence that host necrosis is related to fungal inhibition comes from the observation that daily injections of extracts from compatible bean rust-infected tissue (13) not only prevent this necrosis but also allow the fungus to continue its growth (S. Kaminskyj and M. C. Heath, *unpublished*). If cell death is responsible for the cessation of fungal growth, the necrotized cell must be exerting its effect through a diffusible inhibitor, rather than through death of encompassed haustoria, since often it is the host cells just beyond the area colonized by the fungus that die (*unpublished*). Combined cytological and biochemical studies of several types of host resistance to the flax rust fungus (*Melampsora lini*) also indicate that potentially inhibitory compounds are present at the right time and place to account for the observed restriction of fungal growth (19).

Whether the necrosis of haustorium-containing cells is the cause or the consequence of the death of the encompassed haustorium is a controversial issue (9), and although most ultrastructural studies show that the host cell becomes disorganized before, or at the same time, as the haustorium (21), in some rare cases the haustorium appears to die first (6). However, without any clear morphological or biochemical definition of when cell death *begins*, the temporal relationship between detectable disorganization of fungus and plant may be meaningless.

A point that must be considered in any discussion of defense mechanisms against rust fungi is that reduced fungal growth can occur in the absence of any observable host response or cytological indication that the intercellular mycelium or haustoria are adversely affected (eg, 23). In situations such as these, one is forced to conclude that the plant has other means besides the more typical responses to inhibit fungal growth. However, in some of these cases, the nearby host cells eventually die; then it becomes pertinent to ask whether this necrosis plays any role in resistance. Significantly, necrosis that develops at some infection sites during adult plant resistance to yellow rust (*P. striiformis*) is accompanied by adverse effects on haustoria and intercellular hyphae and the fungus does not sporulate; in contrast, spores do develop at infection sites lacking necrosis in spite of slow rates of fungal growth (23). Thus, in this particular example, it seems that necrosis is required for full resistance irrespective of early reductions in fungal growth rate.

Multiple resistance mechanisms. The observation of more than one plant response in a single infection site (eg, host necrosis and the increased fibrillar content of the extrahaustorial matrix) could be explained in various ways: each is a different expression of basically the same resistance mechanism; only one is a primary determinant of resistance; resistance is determined by some phenomenon unassociated with any of the observed responses of the plant; or there can be more than one factor determining fungal behavior at a given infection site. As already discussed, it is difficult to distinguish between these possibilities in most examples of cultivar resistance. However, it does seem clear that if the entire population of infection sites is considered, host and nonhost plants apparently may possess more than one type of resistance effective against a single rust fungus. For example, in cowpea cultivar Queen Anne, the cowpea rust fungus normally forms only one haustorium and, in about 60% of infection sites, the invaded cell and the haustorium disorganize simultaneously. In the remaining sites,

phospholipidlike material accumulates on the extrahaustorial membrane, the haustorium shows signs of starvation and eventually becomes encased in callose-containing material (18). Assuming that the first and most obvious feature of each interaction (ie, host cell death and the change in the extrahaustorial membrane) directly or indirectly causes the fungus to stop growing, each seems equally effective. However, the effect of the change in the membrane can only be expressed at infection sites if the invaded cell does not die.

Among nonhost interactions, multiple forms of resistance seem even more common (14). For example, some germ tubes of the cowpea rust fungus make "mistakes" on cabbage leaves and form appressoria away from stomata (8); others locate a stoma and enter, but growth within the leaf is then strongly inhibited (12). Presumably two resistance mechanisms must be involved here since it is unlikely that the control of the surface topography of a cabbage leaf is related to the synthesis of the inhibitor of fungal growth inside the tissue. It also seems probable that, if the silicon-rich deposits elicited by the same fungus in French bean leaves act as physical barriers to haustorium formation, then a different resistance mechanism must account for the cessation of fungal growth in the rare instances when a haustorium is formed (7).

Induced susceptibility and suppression of resistant responses. Daly (3) has suggested that rust fungi "induce susceptibility" in host plants by bringing about a metabolic state essential for fungal growth. If this is the case, perhaps many of the expressions of resistance described above are merely indications that the compatible state has not been achieved, which explains the observation that fungal growth can be reduced in their absence. The existence of such "induced susceptibility" is consistent, not only with the subtle metabolic changes that seem to accompany successful infection of the host (eg, 2), but also with the observation that haustorium-containing cells and their neighbors become incapable of expressing the haustorium-inhibiting responses normally triggered by a variety of incompatible rust fungi (13). However, if metabolic cooperation with the host is essential for fungal survival, one might expect heat shock and inhibitors of protein and RNA synthesis to reduce host susceptibility; yet there are at least two reports that these treatments have no such effect (12,33). Moreover, the reduced resistance of already infected tissue could also be explained in terms of the compatible fungus specifically suppressing defense reactions, and indeed, there are data that suggest such "suppressors" exist in rust infections. The necrosis-inhibiting activity of extracts from rusted bean leaves has already been mentioned, and those crude extracts also suppress the formation of the silicon-rich deposits elicited in French bean by the cowpea rust fungus (13,17). Since the bean rust fungus also elicits these deposits in older leaves in which the "suppressor" is less effective (17), it seems that the suppressing activity may play a role in establishing the fungus in its host; significantly, injection of crude suppressor preparations into these older leaves reduces the frequency of the deposits and increases the incidence of infection sites at which haustoria develop (17).

Extracts that suppress silicon deposition do not suppress the different response(s) that prevents the sunflower rust fungus (*P. helianthi*) from forming haustoria in French bean (12,13). This specificity of extract action contrasts with the nonspecific state of susceptibility of French bean cells near established colonies of the bean rust fungus, which allows both sunflower and cowpea rust fungi to form haustoria (13). Thus, it appears that there are two types of "induced susceptibility" associated with successful bean rust infection. The first involves the specific suppression of silicon-rich deposits, thus allowing the first haustorium to form. The second, which occurs after the fugus is established and may be mediated by the haustorium, is less specific and it remains to be determined whether it involves the general suppression of resistant responses or the induction of a metabolic state which precludes these responses from taking place.

Host species specificity. Induced susceptibility, in any form, implies a specific interaction between the fungus and its host, but it cannot be ignored that the available genetic evidence suggests that it is resistance, not susceptibility, that involves such specific interactions (4). However, this evidence applies only to the resistance shown by cultivars of the host plant, and there is no theoretical reason why the types of induced susceptibility involved in bean rust infections may not be important in establishing a "basic compatibility" (4) with the host *species* upon which cultivar, gene-for-gene, resistance is superimposed (16). This concept raises the question of what determines the host species range of a given rust fungus. Some of the proposed mechanisms involved in nonhost resistance, such as preformed inhibitors or the "wrong" topography of the leaf surface, require no active response by the plant and it could be argued that they adversely affect certain rust fungi by chance; thus, a host species is one that, also by chance, has a favorable topography and no products inhibitory to the rust fungus in question.

One might also argue that the seemingly "active" nonhost defense reactions are of no real significance since, even in their absence, the plant would not supply the correct "environment" (such as nutrients or other plant factors) required by the fungus because of its physiological adaptation to its host species. In my opinion, neither of these concepts of "passive" nonhost resistance is consistent with the observation that, once inside the nonhost tissue, the fungus is usually stopped during prehaustorial stages of its development. Such development does not seem to require a special "environment" since many rust fungi can form apparently normal infection structures away from the living plant or exogenous nutrient sources (34). Moreover, treatments that upset the plant's metabolism often allow the fungus to develop as it would in susceptible tissue and form at least one haustorium (12,33).

These results suggest to me that most forms of nonhost resistance exhibited after tissue penetration are "active" and that their apparent nonspecificity with respect to species of rust fungus (10) and mode of induction (eg, silicon deposition can be induced by water injection, 11) indicates that they may be part of a small battery of general defense reactions possessed by every plant. The important question is why are they not observed after infection by the compatible species? One answer is that compatibility depends on the specific accommodation of the rust fungus to "overcome" the basic defense mechanisms of its host. Thus, the ability to form infection structures and the first haustorium in a plant depends on the fungus being able to suppress, to specifically not trigger, or become insensitive to, the nonspecific responses or products which would hinder this development. As discussed earlier, development after the first haustorium has formed may depend upon whether the haustorium can develop some type of metabolic relationship with the plant, be it to stimulate essential metabolism, suppress defense reactions, or obtain essential growth factors. Interestingly, the extensive growth of certain rust fungi in some nonhosts after haustorium-inhibiting responses are prevented (12) suggests that this relationship can be at least partly established in species other than the normal host. Thus, the host range of a rust fungus may be restricted more by its lack of ability to specifically "overcome" the prehaustorial "nonhost-type" defenses of the plant than by whether it can subsequently establish an essential metabolic relationship with the tissue.

Host-cultivar specificity. Once a given rust fungus can successfully infect a population of the host species, there is strong selection pressure within that population for the development of some sort of resistance to minimize the harmful effects of the pathogen. To do this, a "recognizable" feature of the fungus must act as a trigger of the defense reaction(s); it can be argued that this automatically results in the observed gene-for-gene relationship between resistance and avirulence (16). The fact that cultivar resistance is usually expressed after the first haustorium is formed suggests to me that it is based on various forms of "interference" with the metabolic relationship which must be established at this stage between plant and fungus. Note, however, that if this resistance is superimposed on a "basic compatibility" which is actively induced by the fungus, specific processes leading to the establishment of this compatibility may be taking place at the same time as the specific events leading to cultivar resistance (16).

SUMMARY

Cytological studies provide evidence that higher plants possess a number of types of defense mechanisms active against rust fungi. These mechanisms include: 1—inhibitors of germination; 2—topographical triggers of "incorrect" germ tube behavior; 3—barriers (physical or chemical?) to penetration into the tissue; 4—inhibitors (preformed or induced?) of infection structure growth; 5—responses (eg, degradation of haustorial mother cell walls, deposition of silicon-rich material on plant walls) which inhibit haustorium formation; and 6—factors (eg, impaired functioning of the haustorium, phytoalexin release during plant necrosis, inability of the fungus to establish a necessary metabolic relationship with the plant) which reduce the growth of the intercellular mycelium after the first haustorium is formed. Although more than one of these mechanisms may be active at different infection sites in the same plant-fungus interaction, resistance of host plants (cultivar resistance) is typically expressed after the first haustorium is formed (mechanism 6) while nonhost resistance is exhibited earlier (mechanisms 1–5). It is suggested that the ability to overcome these general nonhost-type defense mechanisms is the most important step in determining the host species range of the rust fungus, and that various forms of "induced susceptibility," including suppression of defense reactions, may be employed. Once a "basic compatibility" has been established between the rust fungus and its host species, specific events leading to cultivar gene-for-gene resistance can be selected for. Such resistance does not seem to rely on "replacing" nonhost-type defense mechanisms, but appears to be based on interfering with the relationship between fungus and host developed after the first haustorium is formed.

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