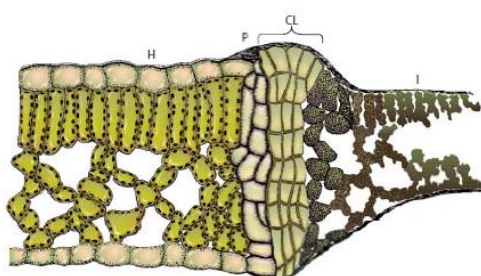


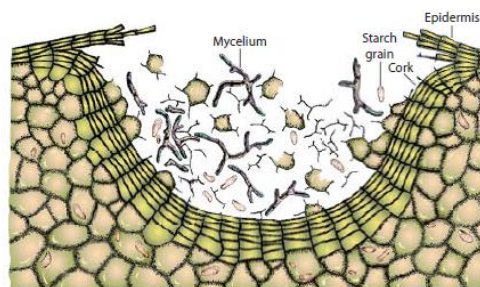
## Histological Defense Structures

### Formation of Cork Layers

Infection by fungi or bacteria, and even by some viruses and nematodes, frequently induces plants to form several layers of cork cells beyond the point of infection (Figs.), apparently as a result of stimulation of the host cells by substances secreted by the pathogen. The cork layers inhibit further invasion by the pathogen beyond the initial lesion and also block the spread of any toxic substances that the pathogen may secrete. Furthermore, cork layers stop the flow of nutrients and water from the healthy to the infected area and deprive the pathogen of nourishment. The dead tissues, including the pathogen, are thus delimited by the cork layers and may remain in place, forming necrotic lesions (spots) that are remarkably uniform in size and shape for a particular host–pathogen combination. In some host–pathogen combinations the necrotic tissues are pushed outward by the underlying healthy tissues and form scabs that may be sloughed off, thus removing the pathogen from the host completely. In tree cankers, such as those caused by the fungus *Seiridium cardinale* on cypress trees (Sanobur tree), resistant plant clones restrict growth of the fungus by forming ligno-suberized boundary zones, which included four to six layers of cells with suberized cell walls. In contrast, susceptible clones have only two to four layers of suberized cells and these are discontinuous, allowing repeated penetration by the fungus past the incomplete barrier.



**FIGURE 6-5** Formation of a cork layer (CL) between infected (I) and healthy (H) areas of leaf. P, phellogen. [After Cunningham (1928). *Phytopathology* 18, 717–751.]

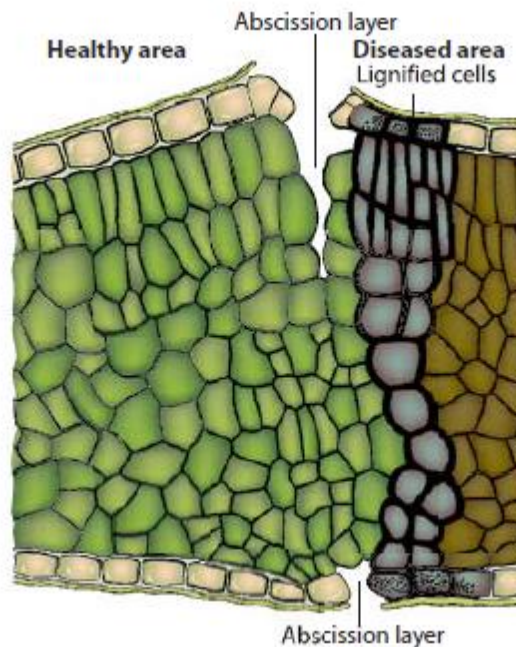


**FIGURE 6-6** Formation of a cork layer on a potato tuber following infection with *Rhizoctonia*. [After Ramsey (1917). *J. Agric. Res.* 9, 421–426.]

### Formation of Abscission Layers

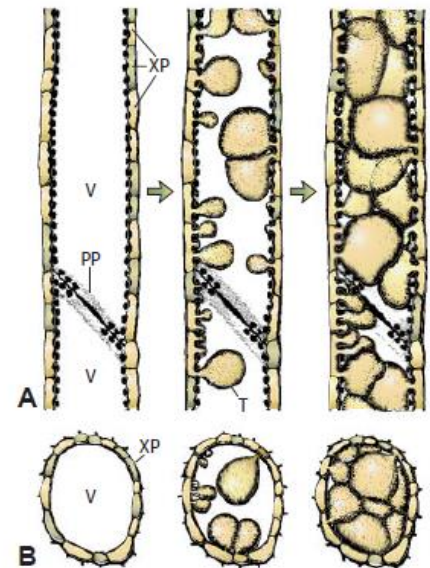
Abscission layers are formed on young, active leaves of stone fruit trees after infection by any of several fungi, bacteria, or viruses. An abscission layer consists of a gap formed between two circular layers of leaf cells surrounding the locus of infection. Upon infection, the middle lamella between these two layers of cells is dissolved throughout the thickness of the leaf, completely cutting off the central area of the infection from the rest of the leaf (Fig.). Gradually, this area shrivels, dies, and sloughs

off, carrying with it the pathogen. Thus, the plant, by discarding the infected area along with a few yet uninfected cells, protects the rest of the leaf tissue from being invaded by the pathogen and from becoming affected by the toxic secretions of the pathogen.



### Formation of Tyloses

Tyloses form in xylem vessels of most plants under various conditions of stress and during invasion by most of the xylem-invading pathogens. Tyloses are overgrowths of the protoplast of adjacent living parenchymatous cells, which protrude into xylem vessels through pits (Fig.). Tyloses have cellulosic walls and may, by their size and numbers, clog the vessel completely. In some varieties of plants, tyloses form abundantly and quickly ahead of the pathogen, while the pathogen is still in the young roots, and block further advance of the pathogen. The plants of these varieties remain free of and therefore resistant to this pathogen.



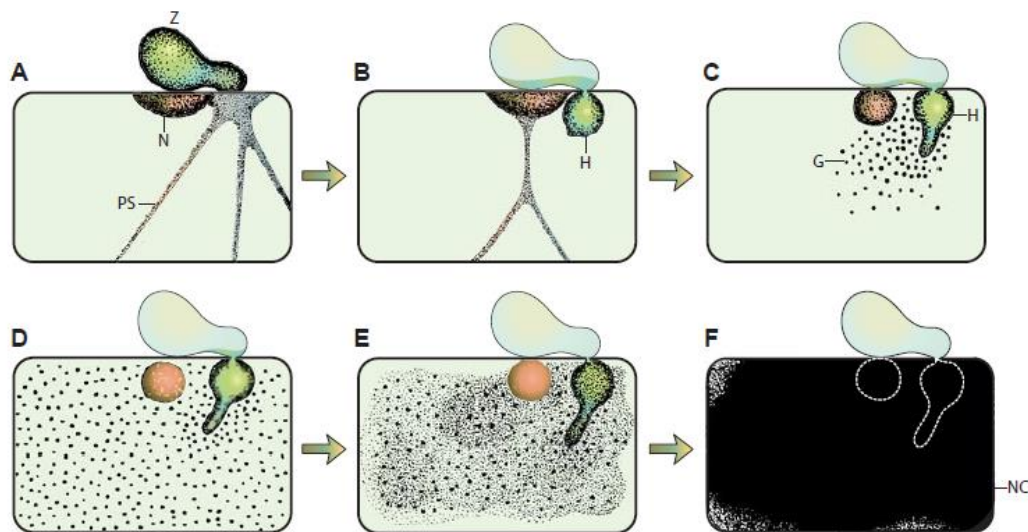
### Deposition of Gums

Various types of gums are produced by many plants around lesions after infection by pathogens or injury. **Gum secretion is most common in stone fruit trees** but occurs in most plants. The defensive role of gums stems from the fact that they are deposited

quickly in the intercellular spaces and within the cells surrounding the locus of infection, thus forming an impenetrable barrier that completely encloses the pathogen. The pathogen then becomes isolated, starves, and sooner or later dies.

### **Necrotic Structural Defense Reaction: Defense through the Hypersensitive Response**

The hypersensitive response is considered a biochemical rather than a structural defense mechanism but is described here briefly because some of the cellular responses that accompany it can be seen with the naked eye or with the microscope. In many host–pathogen combinations, as soon as the pathogen establishes contact with the cell, the nucleus moves toward the invading pathogen and soon disintegrates. At the same time, brown, resin-like granules form in the cytoplasm, first around the point of penetration of the pathogen and then throughout the cytoplasm. As the browning discoloration of the plant cell cytoplasm continues and death sets in, the invading hypha begins to degenerate (Fig.).



**FIGURE 6-9** Stages in the development of the necrotic defense reaction in a cell of a very resistant potato variety infected by *Phytophthora infestans*. N, nucleus; PS, protoplasmic strands; Z, zoospore; H, hypha; G, granular material; NC, necrotic cell. [After Tomiyama (1956). *Ann. Phytopathol. Soc. Jpn.* 21, 54–62.]

In most cases the hypha does not grow out of such cells, and further invasion is stopped. In bacterial infections of leaves, the hypersensitive response results in the destruction of all cellular membranes of cells in contact with bacteria, which is followed by desiccation and necrosis of the leaf tissues invaded by the bacteria.

Although it is not quite clear whether the HR is the cause or the consequence of resistance, this type of necrotic defense is quite common, particularly in diseases caused by obligate fungal parasites and by viruses, bacteria, and nematodes. Apparently, the

## Plant Resistance to Diseases

necrotic tissue not only isolates the parasite from the living substance on which it depends for its nutrition and, thereby, results in its starvation and death, but, more importantly, it signifies the concentration of numerous biochemical cell responses and antimicrobial substances that neutralize the pathogen. The faster the host cell dies after invasion, the more resistant to infection the plant seems to be. Moreover, through the signalling compounds and pathways developed during the hypersensitive response, the latter serves as the springboard for localized and systemic acquired resistance.

**INDUCED BIOCHEMICAL DEFENSES****Induced Biochemical Non-host Resistance**

As mentioned earlier, non-host resistance is the resistance that keeps a plant protected from pathogens that are, through evolution, incompatible with that host. Although the nature of non-host resistance is unknown, for a pathogen it can be as big a gap to bridge as the difference between the features of a potato plant and an oak tree, or as close as the difference between the features of potato and tomato, or barley and wheat. It appears, however, that in some plant/pathogen interactions of taxonomically unrelated plants (e.g., potato and oak or oak and wheat), non-host resistance is controlled by constitutive defenses and/or defenses induced by non-specific stimuli in a nonspecific manner. Such defenses include **physical topography and the structures present on the plant**, the **presence of toxic or the absence of essential compounds**, and so on. In other plant/pathogen combinations, in which the plants are taxonomically related (e.g., potato and tomato, barley and wheat), **non-host resistance involves primarily inducible defenses elicited by the recognition of pathogen-specific molecules**. Some cases of non-host resistance, however, seem to be controlled by a single gene. Some examples of questionable non-host resistance include the resistance of the non-host pea to the *Pseudomonas syringae* pv. *syringae* bacterium, which infects bean but not pea. The reaction occurs when that bacterium carries a gene that is responsible for elicitation of a potentially defensive response in the normally non-host pea, that is expressed as a visible hypersensitive response. In another example, the potato late blight fungus *Phytophthora infestans*, normally does not infect the tobacco species *Nicotiana benthamiana*. The non-host resistance of the tobacco species, however, is lost if the pathogen does not carry an “avirulence-like gene,” which produces a protein that elicits cell death in the tobacco. This is unique in that in other plant/pathogen combinations, the absence of a single “non-host avirulence gene” does not make the non-host plant susceptible. It would appear, therefore, that if the cell death response to the elicitor



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controlled by the avirulence gene really contributes to resistance, and then the non-host resistance in such situations is controlled by more than one component. In still another case, non-host resistance in some cereals [wheat to powdery mildew strains from another cereal (barley), or in barley to *Puccinia* rust races from wheat], involves similar gene-for gene interactions and non-host resistance occurs through defense mechanisms involving recognition of an elicitor and development of a hypersensitive response. Disease resistance does not always involve pathogen recognition events, but, especially in polygenic or quantitative resistance, it may involve directly various structural or chemical defense mechanisms. This also happens in some cases of non-host resistance, e.g., in oat roots to the wheat fungus *Gaeumannomyces graminis* f. sp. *tritici*, while they are susceptible to the oat fungus *G. graminis* f. sp. *avenae*. The non-host resistance of oat roots to the wheat fungus is caused by the presence of the saponin compound avenacin in the oat roots, which is toxic to the fungus. This compound is also toxic to the oat fungus, but the latter produces an enzyme that detoxifies the saponin in oat roots and can infect them. The non-host resistance to the wheat fungus, however, is compromised in saponin-deficient mutants in which the wheat fungus causes a successful infection. **This shows that non-host resistance in some plant/microbe interactions is caused by a direct defense mechanism rather than by recognition events.**

In all these examples, the pathogen or the host is already closely related and nearly fully adopted to the characteristics of non-host resistance presented to it. In less related plants or pathogens, however, in which true non-host resistance is found routinely, it is more likely to be the result of **effective nonspecific defenses** such as physical characteristics and non-specific responses to wounding and damage done by the pathogen during attempted invasion than to defenses elicited by specific recognition events. There is also, however, the case of pathogens that have alternate hosts, such as wheat stem rust and barberry and cedar apple rust on apple and cedar. These are, perhaps, interesting from an evolutionary point of view because, presumably, before the second of the alternate hosts that became a host, it was surely a non-host. How the rust fungus bridged the two taxonomically extremely different hosts is not known. The change in ploidy (from haploid to diploid and back to haploid) was probably involved, but how the fungus broke the non-host resistance of the other host and how it used the non-resistant host as a completely cooperative host is still a mystery. The present consensus is that plants that exhibit non-host resistance against pathogens of other plants do not need to

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carry resistance genes that recognize these pathogens because they carry genes that provide the plants with nonspecific defenses that are fully effective in protecting the plant from these pathogens. However, it may be possible that non-host resistance, along with polygenic and monogenic host resistance, forms a continuum of resistance that begins to overlap as the taxonomic (evolutionary) distance between host and non-host plants becomes closer and results in a complex and continuous network of plant/pathogen interactions.

**Function of Gene Products in Quantitative Resistance**

Unlike most major (or R) genes involved in monogenic resistance, which appear to code for components that help the host recognize the pathogen and to subsequently express the hypersensitive response, genes for quantitative resistance seem to be involved directly in the expression or production of some sort of structural or biochemical defense. Quantitative resistance defenses are basically the same ones that follow the hypersensitive response in monogenic resistance; in qualitative resistance, however, defenses generally do not follow a hypersensitive response and cell death because the latter do not usually occur in quantitative resistance. Genes involved in quantitative resistance are present in the same areas of plant chromosomes that contain the genes involved in defense responses, such as the production of phenylalanine ammonia lyase, hydroxyproline-rich glycoproteins, and pathogenesis-related proteins. The defenses in quantitative resistance, however, develop slower and perhaps reach a lower level than those in the race-specific (R gene) resistance. Quantitative resistance is also affected much more by changes in the environment, mostly of changes in temperature during the various stages of development of resistance.