

INDUCED STRUCTURAL AND BIOCHEMICAL DEFENSES**Recognition of the Pathogen by the Host Plant**

Early recognition of the pathogen by the plant is very important if the plant is to mobilize the available biochemical and structural defenses to protect itself from the pathogen. The plant apparently begins to receive signal molecules, i.e., molecules that indicate the presence of a pathogen, as soon as the pathogen establishes physical contact with the plant (Fig).

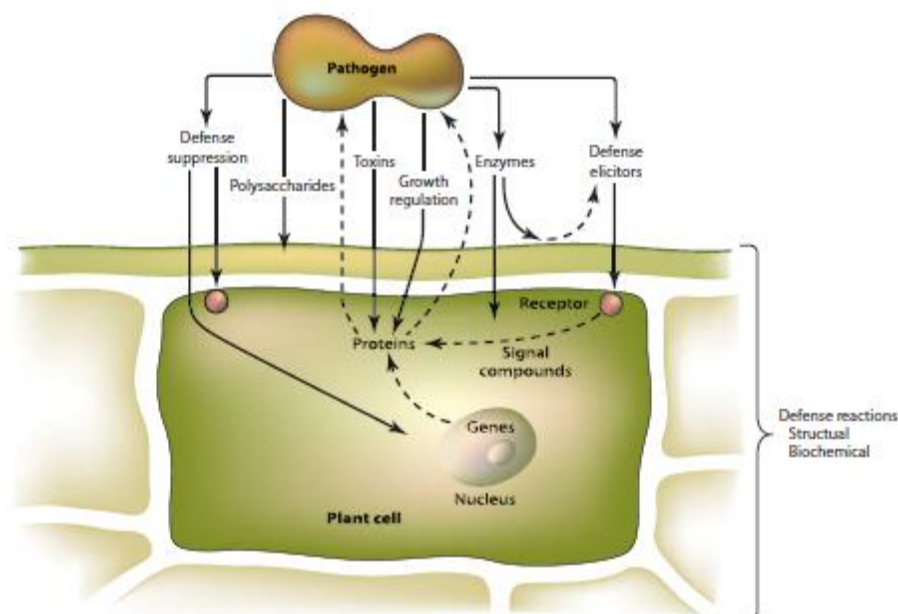


FIGURE 6-3 Schematic representation of pathogen interactions with host plant cells. Depending on its genetic makeup, the plant cell may react with numerous defenses, which may include cell wall structural defenses (waxes, cutin, suberin, lignin, phenolics, cellulose, callose, cell wall proteins) or biochemical wall, membrane, cytoplasm, and nucleus defense reactions. The latter may involve bursts of oxidative reactions, production of elicitors, hypersensitive cell death, ethylene, phytoalexins, pathogenesis-related proteins (hydrolytic enzymes, β -1,3-glucanases, chitinases), inhibitors (thionins, proteinase inhibitors, thaumatin-like proteins), and so on.

Pathogen Elicitors

Various pathogens, especially fungi and bacteria, release a variety of substances in their immediate environment that act as non-specific elicitors of pathogen recognition by the host. Such non-specific elicitors include **toxins, glycoproteins, carbohydrates, fatty acids, peptides, and extracellular microbial enzymes such as proteases and pectic enzymes**. In various host–pathogen combinations, certain substances secreted by the pathogen, such as *avr* gene products, *hrp* gene products (*hrp* genes control the ability of phytopathogenic bacteria to cause disease and to elicit hypersensitive reactions on resistant plants.), and suppressor molecules, act as specific pathogen elicitors of recognition by the specific host plant. In many cases, in which host enzymes break down a portion of the polysaccharides making up the pathogen surface or pathogen

enzymes break down a portion of the plant surface polysaccharides, the released oligomers or monomers of the polysaccharides act as recognition elicitors for the plant.

Host Plant Receptors

The **location** of host receptors that recognize pathogen elicitors is **not generally known**, but several of those studied appear to **exist outside or on the cell membrane**, whereas others apparently occur **intracellularly**. In the powdery mildew of cereals, a soluble carbohydrate that acts as an elicitor from the wheat powdery mildew fungus *Blumeria graminis* f.sp. *tritici* is recognized by a broad range of cereals (barley, oat, rye, rice, and maize) in which it induces the **expression of all defense related genes** tested and also **induced resistance to subsequent attacks with the fungus**. **The elicitor alone, in absence of the powdery mildew fungus, did not induce a hypersensitive response but it did induce an accumulation of thaumatin-like proteins** in the various cereals. Thaumatin-like proteins (TLPs) are the products of a large, highly complex gene family involved in host defence and a wide range of developmental processes in fungi, plants, and animals. Despite their dramatic diversification in organisms, TLPs appear to have originated in early eukaryotes and share a well-defined TLP domain. Nonetheless, determination of the roles of individual members of the TLP superfamily remains largely undone.

Mobilization of Defenses

Once a particular plant molecule recognizes and reacts with a molecule (elicitor) derived from a pathogen, it is assumed that the **plant “recognizes” the pathogen**. Following such recognition, a **series of biochemical reactions and structural changes** are set in motion in the plant cell(s) in an effort to **fend off the pathogen and its enzymes**, toxins, etc. How quickly the plant recognizes the (presence of a) pathogen and how quickly it can send out its alarm message(s) and mobilize its defenses determine whether hardly any infection will take place at all (as in the hypersensitive response) or how much the pathogen will develop, i.e., how severe the symptoms (leaf spots, stem, fruit, or root lesions, etc.) will be, before the host defenses finally stop further development of the pathogen.

Transmission of the Alarm Signal to Host Defense Providers: Signal Transduction

Once the pathogen-derived elicitors are recognized by the host, a series of **alarm signals** are sent out to **host cell proteins** and **to nuclear genes**, causing them to become activated, to produce substances inhibitory to the pathogen, and to mobilize themselves or their products toward the point of cell attack by the pathogen. **Some of the alarm**

substances and signal transductions are only intracellular, but in many cases the signal is also transmitted to several adjacent cells and, apparently, the alarm signal is often transmitted systemically to most or all of the plant. The chemical nature of the transmitted signal molecules is not known with certainty in any host–pathogen combination. Several types of molecules have been implicated in intracellular signal transduction. **The most common such signal transducers appear to be various protein kinases, calcium ions, phosphorylases and phospholipases, ATPases, hydrogen peroxide (H₂O₂), ethylene, and others.** Systemic signal transduction, which leads to systemic acquired resistance, is thought to be carried out by **salicylic acid, oligogalacturonides released from plant cell walls, jasmonic acid, systemin, fatty acids, ethylene, and others.** Some natural or synthetic chemicals, such as **salicylic acid and the synthetic dichloroisonicotinic acid**, also activate the signalling pathway that leads to systemic acquired resistance against several diverse types of plant pathogenic viruses, bacteria, and fungi.

INDUCED STRUCTURAL DEFENSES

Despite the preformed superficial or internal defense structures of host plants, most pathogens manage to **penetrate** their hosts through wounds and natural openings and to produce various degrees of infection. Even after the pathogen has penetrated the preformed defense structures, however, plants usually respond by **forming one or more types of structures that are more or less successful in defending** the plant from further pathogen invasion. Some of the defense structures formed involve the cytoplasm of the cells under attack, and the process is called **cytoplasmic defense reaction**; others involve the walls of invaded cells and are called **cell wall defense structures**; and still others involve tissues ahead of the pathogen (deeper into the plant) and are called **histological defense structures**. Finally, the death of the invaded cell may protect the plant from further invasion. This is called the **necrotic or hypersensitive defense reaction**.

Cytoplasmic Defense Reaction

In a few cases of slowly growing, weakly pathogenic fungi, such as weakly pathogenic *Armillaria* strains and the mycorrhizal fungi, that induce chronic diseases or nearly symbiotic conditions, the plant cell cytoplasm surrounds the clump of hyphae and the plant cell nucleus is stretched to the point where it breaks in two. In some cells, the cytoplasmic reaction is overcome and the protoplast disappears while fungal growth increases. In some of the invaded cells, however, the cytoplasm and nucleus enlarge.

The cytoplasm becomes granular and dense, and various particles or structures appear in it. Finally, the mycelium of the pathogen disintegrates and the invasion stops.

Cell Wall Defense Structures

Cell wall defense structures involve morphological changes in the cell wall or changes derived from the cell wall of the cell being invaded by the pathogen. The effectiveness of these structures as defense mechanisms seems to be rather limited, however. Three main types of such structures have been observed in plant diseases.

(1) The outer layer of the cell wall of parenchyma cells coming in contact with incompatible bacteria swells and produces an amorphous, fibrillar material that surrounds and traps the bacteria and prevents them from multiplying.

(2) Cell walls thicken in response to several pathogens by producing what appears to be a cellulosic material. This material, however, is often infused with phenolic substances that are cross-linked and further increase its resistance to penetration.

(3) Callose **papillae** are deposited on the inner side of cell walls in response to invasion by fungal pathogens. Papillae seem to be produced by cells within minutes after wounding and within 2 to 3 hours after inoculation with microorganisms. Although the main function of papillae seems to be repair of cellular damage, sometimes, especially if papillae are present before inoculation, they also seem to prevent the pathogen from subsequently penetrating the cell. In some cases, hyphal tips of fungi penetrating a cell wall and growing into the cell lumen are enveloped by cellulosic (callose) materials that later become infused with phenolic substances and form a sheath or lignituber around the hypha (Fig).

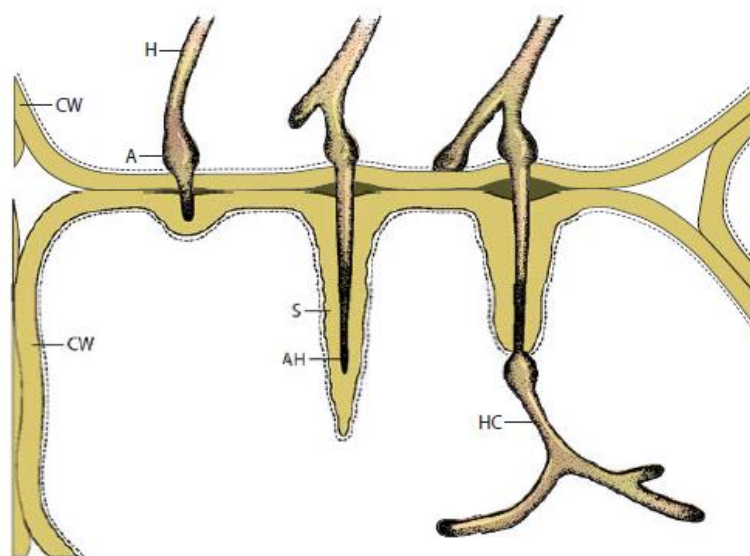


FIGURE 6-4 Formation of a sheath around a hypha (H) penetrating a cell wall (CW). A, appressorium; AH, advancing hypha still enclosed in sheath; HC, hypha in cytoplasm; S, sheath.