

Defense through increased levels of phenolic compounds**Defense through Production of Secondary Metabolites: Phenolics****Simple Phenolic Compounds**

It has often been observed that certain common phenolic compounds that are toxic to pathogens are produced and accumulate at a faster rate after infection, especially in a resistant variety of plant relative to a susceptible variety. Chlorogenic acid, caffeic acid, and ferulic acid are examples of such phenolic compounds. In peach, chlorogenic acid is present in quite high concentration both in immature fruit and in fruit of varieties resistant to the brown rot disease caused by the fungus *Monilinia fructicola*. The fruit is resistant in both cases, not because of the toxicity of the acid to the causal fungus, but rather because it inhibits the production of fungal enzymes that cause degradation of host tissue. In date palm tree roots, cell wall-bound hydroxybenzoic acid and sinapic acid increased 11–12 times as much in cultivars resistant to *Fusarium* than they did in susceptible cultivars. In plants such as vetch (*Vicia sativa*), resistance to the higher parasitic plant *Orobancha aegyptiaca* appears to result from higher levels of free and bound phenolics, lignin and peroxidase activity produced in the roots of resistant varieties following infection, compared to susceptible ones. In cacao infected with the witches' broom fungus *Crinipellis perniciosa*, infected young stems contain 7–8 times as much caffeine, which inhibits growth of the fungus in culture, than healthy stems. In another polygenic disease, the black sigatoka disease of banana caused by the fungus *Mycosphaerella fijiensis*, plant defenses included an activation of phenylalanine ammonia lyase and a subsequent accumulation of phenolic compounds. It also caused early activation of a banana response to the fungal compound trihydroxytetralone (THT), which, in resistant varieties, caused necrotic microlesions and elicitation of infection-induced defense reactions leading to incompatibility (resistance) between the pathogen and the host plant. In susceptible varieties, however, the fungus produced necrotizing levels of THT only at the later stages of pathogenesis after a compatible interaction had been established and typical symptoms had developed. Although some of the common phenolics may each reach concentrations that could be toxic to the pathogen, it should be noted that several of them appear concurrently in the same diseased tissue, and it is possible that the combined effect of all fungitoxic phenolics present, rather than that of each one separately, is responsible for the inhibition of infection in resistant varieties. It has even been proposed that because of the universal uniform or strategic location of phenolics-storing plant cells, these cells can, by de-

compartmentation and rapid oxidation of their phenolic contents, self-sacrifice, leading to the first line of defense — cell death — or leading to the production of a slower defense line — a peridermal defense layer.

Phytoalexins

Phytoalexins are toxic antimicrobial substances produced in appreciable amounts in plants only after stimulation by various types of phytopathogenic microorganisms or by chemical and mechanical injury. Phytoalexins are produced by healthy cells adjacent to localized damaged and necrotic cells in response to materials diffusing from the damaged cells. Phytoalexins are not produced during compatible biotrophic infections. Phytoalexins accumulate around both resistant and susceptible necrotic tissues. Resistance occurs when one or more phytoalexins reach a concentration sufficient to restrict pathogen development. Most known phytoalexins are toxic to and inhibit the growth of fungi pathogenic to plants, but some are also toxic to bacteria, nematodes, and other organisms. More than 300 chemicals with phytoalexin like properties have been isolated from plants belonging to more than 30 families. The chemical structures of phytoalexins produced by plants of a family are usually quite similar; e.g., in most legumes, phytoalexins are isoflavonoids, and in the Solanaceae they are terpenoids. Most of the phytoalexins are produced in plants in response to infection by fungi, but a few bacteria, viruses, and nematodes have also been shown to induce the production of phytoalexins. Some of the better studied phytoalexins include phaseollin in bean; pisatin in pea; glyceollin in soybean, alfalfa, and clover; rishitin in potato; gossypol in cotton; and capsidiol in pepper. Phytoalexin production and accumulation occur in healthy plant cells surrounding wounded or infected cells and are stimulated by alarm substances produced and released by the damaged cells and diffusing into the adjacent healthy cells. Most phytoalexin elicitors are generally high molecular weight substances that are constituents of the fungal cell wall, such as glucans, chitosan, glycoproteins, and polysaccharides. The elicitor molecules are released from the fungal cell wall by host plant enzymes. Most such elicitors are nonspecific, i.e., they are present in both compatible and incompatible races of the pathogen and induce phytoalexin accumulation irrespective of the plant cultivar. A few phytoalexin elicitors, however, are specific, as the accumulation of phytoalexin they cause on certain compatible and incompatible cultivars parallels the phytoalexin accumulation caused by the pathogen races themselves. Although most phytoalexin elicitors are thought to be of pathogen origin, some elicitors, e.g., oligomers of galacturonic acid, are produced

by plant cells in response to infection or are released from plant cell walls after their partial breakdown by cell wall degrading enzymes of the pathogen. The formation of phytoalexins in a susceptible (compatible) host following infection by a pathogen seems, in some cases, to be prevented by suppressor molecules produced by the pathogen. The suppressors seem to also be glucans or glycoproteins, or one of the toxins produced by the pathogen. The mechanisms by which phytoalexin elicitors, phytoalexin production, phytoalexin suppressors, genes for resistance or susceptibility, and the expression of resistance or susceptibility are connected are still not well understood. Several hypotheses have been proposed to explain the interconnection of these factors, but much more work is needed before a satisfactory explanation can be obtained. Species or races of fungi pathogenic to a particular plant species seem to stimulate the production of generally lower concentrations of phytoalexins than non-pathogens. For example, in the case of pisatin production by pea pods inoculated with the pathogen *Ascochyta pisi*, pea varieties produce concentrations of pisatin that are approximately proportional to the resistance of the variety to the pathogen. When the same pea variety is inoculated with different strains of the fungus, the concentration of pisatin produced is approximately inversely proportional to the virulence of each particular fungal strain inoculated on the pea variety. Also, in soybean plants infected with the fungus *Phytophthora megasperma* f. sp. *glycinea*, inoculations of fungal races on incompatible host cultivars resulted in earlier accumulations and higher concentrations of the phytoalexin glyceollin than inoculations of fungal races on compatible cultivars. It has been suggested that the higher concentrations of glyceollin in incompatible host–pathogen combinations are the result of reduced biodegradation rather than increased biosynthesis of the phytoalexin. In some host-pathogen systems, however, e.g., in the bean/*Colletotrichum lindemuthianum* and the potato/*Phytophthora infestans* systems, the respective phytoalexins, such as phaseollin and rishitin, reach equal or higher concentrations in compatible (susceptible) hosts compared to incompatible (resistant) ones. However, pathogenic races or species of fungi seem to be less sensitive to the toxicity of the phytoalexin(s) produced by their host plant than non-pathogenic fungi. It has been suggested that pathogens may have an adoptive tolerance mechanism that enables them to withstand higher concentrations of the host phytoalexin after earlier exposures to lower concentrations of the phytoalexin. It is known, however, that many pathogenic fungi can metabolize the host phytoalexin into a nontoxic compound, thereby decreasing the toxicity of the phytoalexin to the pathogen. It is also known that

numerous pathogenic fungi are successful in causing disease, although they are sensitive to or unable to metabolize the host phytoalexins. Furthermore, some fungi that can either degrade or tolerate certain phytoalexins are unable to infect the plants that produce them. In general, it appears that phytoalexins may play a decisive or an auxiliary role in the defense of some hosts against certain pathogens, but their significance, if any, as factors of disease resistance in most host–pathogen combinations is still unknown.