

HOW DO R GENES CONFER RESISTANCE?

The mechanisms by which R genes bring about disease resistance to a plant against a specific pathogen are not yet understood. It is believed that the elicitor molecule produced by an *avr* gene of the pathogen is recognized by a specific plant receptor encoded by an R gene. What happens next is mostly speculation. Following recognition of the elicitor by the receptor molecule, one or more kinase enzymes may become activated, which then amplify the signal by phosphorylating, and thereby energizing, other kinases and other enzymes. This leads to a cascade of biochemical reactions that, in ways that are still unclear, result in the hypersensitive response and, thereby, localized host resistance at the point of attack by the pathogen. Of course, in many cases, the hypersensitive response is followed by the development of various levels of systemic acquired resistance (SAR), which is expressed in the vicinity of attack as well as in distant parts of the plant.

DEFENSE THROUGH LACK OF ESSENTIAL FACTORS**Lack of Recognition between Host and Pathogen**

A plant species either is a host for a particular pathogen, e.g., wheat for the wheat stem rust fungus, or it is not a host for that pathogen, e.g., tomato for wheat stem rust fungus.

How does a pathogen recognize that the plant with which it comes in contact is a host or non-host? Plants of a species or variety may not become infected by a pathogen if their surface cells lack specific **recognition factors** (specific molecules or structures) that can be recognized by the pathogen. If the pathogen does not recognize the plant as one of its host plants, it may not become attached to the plant or may not produce infection substances, such as **enzymes, or structures, such as appressoria, penetration pegs, and haustoria, necessary for the establishment of infection**. It is not known what types of molecules or structures are involved in the recognition of plants and pathogens, but it is thought that they probably include various types of **oligosaccharides and polysaccharides, and proteins or glycoproteins**. Also, it is not known to what extent these recognition phenomena are responsible for the success or failure of initiation of infection in any particular host–pathogen combination.

Lack of Host Receptors and Sensitive Sites for Toxins

In host–pathogen combinations in which the pathogen (usually a fungus) produces a host-specific toxin, the toxin, which is responsible for the symptoms, is thought to attach to and react with specific receptors or sensitive sites in the cell. Only plants that have such sensitive receptors or sites become diseased. Plants of other varieties or

species that lack such receptors or sites remain resistant to the toxin and develop no symptoms.

Lack of Essential Substances for the Pathogen

Species or varieties of plants that for some reason do not produce one of the substances essential for the survival of an obligate parasite, or for development of infection by any parasite, would be resistant to the pathogen that requires it. Thus, for *Rhizoctonia* to infect a plant it needs to obtain from the plant a substance necessary for formation of a **hyphal cushion** from which the fungus sends into the plant its penetration hyphae. In plants in which this substance is apparently lacking, cushions do not form, infection does not occur, and the plants are resistant. The fungus does not normally form hyphal cushions in pure cultures but forms them when extracts from a susceptible are added to the culture. Also, certain mutants of *Venturia inaequalis*, the cause of apple scab, which had lost the ability to synthesize a certain **growth factor**, also lost the ability to cause infection. When the particular growth factor is sprayed on the apple leaves during inoculation with the mutant, the mutant not only survives but it also causes infection. The advance of the infection continues only as long as the growth factor is supplied externally to the mutant. In some host–pathogen combinations, disease develops but the amount of disease may be reduced by the fact that certain host substances are present in lower concentrations. For example, bacterial soft rot of potatoes, caused by *Erwinia carotovora* var. *atroseptica*, is less severe on potatoes with low-reducing sugar content than on potatoes high in reducing sugars.