East African Scholars Journal of Agriculture and Life Sciences
Abbreviated Key Title: East African Scholars J Agri Life Sci
ISSN 2617-4472 (Print) | ISSN 2617-7277 (Online)
Published By East African Scholars Publisher, Kenya
Volume-7 | Issue-10 | Oct-2024 | DOI: https://doi.org/10.36349/easjals.2024.v07i10.001

OPEN ACCESS

Review Article

Host Recognition for Survival of Plant Pathogens

Senthilmurugan Palanisamy[1*], Atul Singh[2], Potunuri Hema Prashanthi Lakshmi[2], Bhuvana C.R[2], Glory, K.B[2]

[1]Associate Professor & HOD, Department of Plant Pathology [1]College of Agriculture, Koneru Lakshmaiah Education Foundation, KL University, Green Fields, Guntur, Andra Pradesh
[2]Assistant Professor, Department of Plant Pathology [1]College of Agriculture, Koneru Lakshmaiah Education Foundation, KL University, Green Fields, Guntur, Andra Pradesh

Article History
Received: 15.03.2024
Accepted: 22.04.2024
Published: 31.10.2024

Journal homepage:
http://www.easpublisher.com

Quick Response Code

Abstract: The intact, healthy plant is a community of cells built in a fortress-like fashion. Plant cells consist of cell wall contains the nucleus and various organelles and all the substances for which the pathogens attack them. The cytoplasm and the organelles it contains are separated from each other by membranes that carry various types of proteins embedded in them (Fig. 5-2). The plant surfaces that come in contact with the environment either consist of cellulose, as in the epidermal cells of roots and in the intercellular spaces of leaf parenchyma cells, or consist of a cuticle that covers the epidermal cell walls, as is the case in the aerial parts of plants. Often an additional layer, consisting of waxes, is deposited outside the cuticle, especially on younger parts of plants Pathogens attack plants because during their evolutionary development they have acquired the ability to live off the substances manufactured by the host plants, and some of the pathogens depend on these substances for survival. Many substances are contained in the protoplast of the plant cells, however, and if pathogens are to gain access to them they must first penetrate the outer barriers formed by the cuticle and/or cell walls. Even after the outer cell wall has been penetrated, further invasion of the plant by the pathogen necessitates the penetration of more cell walls. Furthermore, the plant cell contents are not always found in forms immediately utilizable by the pathogen and must be broken down to units that the pathogen can absorb and assimilate. Moreover, the plant, reacting to the presence and activities of the pathogen, produces structures and chemical substances that interfere with the advance or the existence of the pathogen; if the pathogen is to survive and to continue living off the plant, it must be able to overcome such obstacles. Therefore, for a pathogen to infect a plant it must be able to make its way into and through the plant, obtain nutrients from the plant, and neutralize the defense reactions of the plant. Pathogens accomplish these activities mostly through secretions of chemical substances that affect certain components or metabolic mechanisms of their hosts. Penetration and invasion, however, seem to be aided by, or in some cases be entirely the result of, the mechanical force exerted by certain pathogens on the cell walls of the plant.

Keywords: healthy plant, Plant cells, Host Recognition.

INTRODUCTION

Frequently, a single plant is attacked by fungi, bacteria, protozoa and nematodes. Each plant species is affected by approximately 100 different kinds of fungi, bacteria, mollicutes, viruses, by hundreds, thousands, and, in leafspot diseases of large trees, probably hundreds of thousands of individuals of a single kind of pathogen. Although such plants may suffer damage to a lesser or greater extent, many survive all these attacks and, not uncommonly, manage to grow well and to produce appreciable yields.

In general, plants defend themselves against pathogens by a combination of weapons from two arsenals: (1) structural characteristics that act as physical

***Corresponding Author:** Dr. Senthilmurugan Palanisamy
Associate Professor & HOD, Department of Plant Pathology [1]College of Agriculture, Koneru Lakshmaiah Education Foundation, KL University, Green Fields, Guntur, Andra Pradesh

barriers and inhibit the pathogen from gaining entrance and spreading through the plant and (2) biochemical reactions that take place in the cells and tissues of the plant and produce substances that are either toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant. The combinations of structural characteristics and biochemical reactions employed in the defense of plants are different in different host–pathogen systems. In addition, even within the same host and pathogen, the combinations vary with the age of the plant, the kind of plant organ and tissue attacked, the nutritional condition of the plant, and the weather conditions.

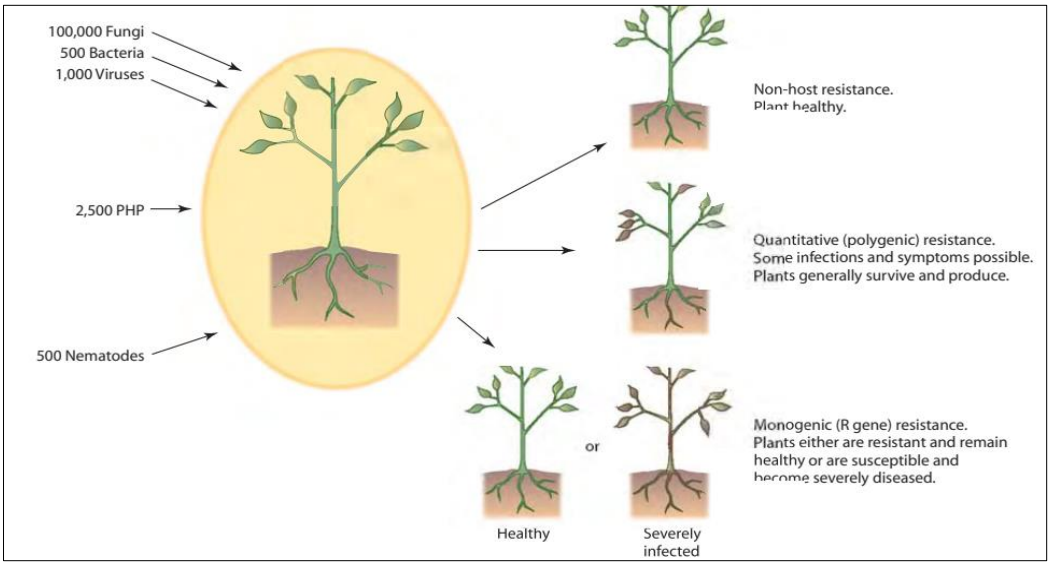
Recognition between Host and Pathogen

It is still unclear how pathogens recognize their hosts and vice versa. It is assumed that when a pathogen comes in contact with a host cell, an early event takes place that triggers a fairly rapid response in each organism that either allows or impedes further growth of the pathogen and development of disease. The nature of the "early event" is not known with certainty in any host–parasite combination, but it may be one of many biochemical substances, structures, and pathways. These may include specific host signal compounds or structures, or specific pathogen elicitor molecules, and either of them may induce specific actions or formation of specific products by the other organism (Fig. 2-6).

Host components acting as signals for recognition by and activation of pathogens are numerous. They may include fatty acids of the plant cuticle that activate pro- duction by the pathogen of the cutinase enzyme, which breaks down cutin; galacturonan molecules of host pectin, which stimulate the production of pectin lyase enzymes by the fungus or bacterium; certain phenolic compounds, such as strigol, which stimulate activation and germination of propagules of some pathogens; and isoflavones and other phenolics, amino acids, and sugars released from plant wounds that activate a series of genes in certain pathogens leading to infection. A host plant may also send cues for recognition by some of its pathogens by certain of its surface characteristics such as ridges or furrows, hardness, or release of certain ions such as calcium.

Pathogen components that act as elicitors of recognition by the host plant and subsequent mobilization of plant defenses are still poorly understood. Elicitor molecules may be released from attacking pathogens before or during entry into the host, and they may have a narrow host range, e.g., the elicitins. Some elicitors may be components of the cell surface of the pathogen (e.g., β-glucans, chitin, or chitosan) that are released by the action of host enzymes (e.g., β-glucanase and/or chitinase) and have broad host ranges; some may be synthesized and released by the pathogen after it enters the host in response to host signals. The latter elicitors include the harpin proteins of bacteria that induce development of the hypersensitive response, certain hydroxy lipids, and certain peptides and carbohydrates that induce specific host defense responses such as the production of phytoalexins. Elicitors are considered as determinants of pathogen avirulence, as by their presence they elicit the hypersensitive (resistance) response and initiation of transcription of the plant genes that encode the various components of the defense response. These defense measures by the host plant, in turn, result in the pathogen appearing as avirulent.

When the initial recognition signal received by the pathogen favors growth and development, disease may be induced; if the signal suppresses pathogen growth and activity, disease may be aborted. However, if the initial recognition elicitor received by the host triggers a defense reaction, pathogen growth and activity may be slowed or stopped and disease may not develop; if the elicitor either suppresses or bypasses the defense reac- tion of the host, disease may develop.

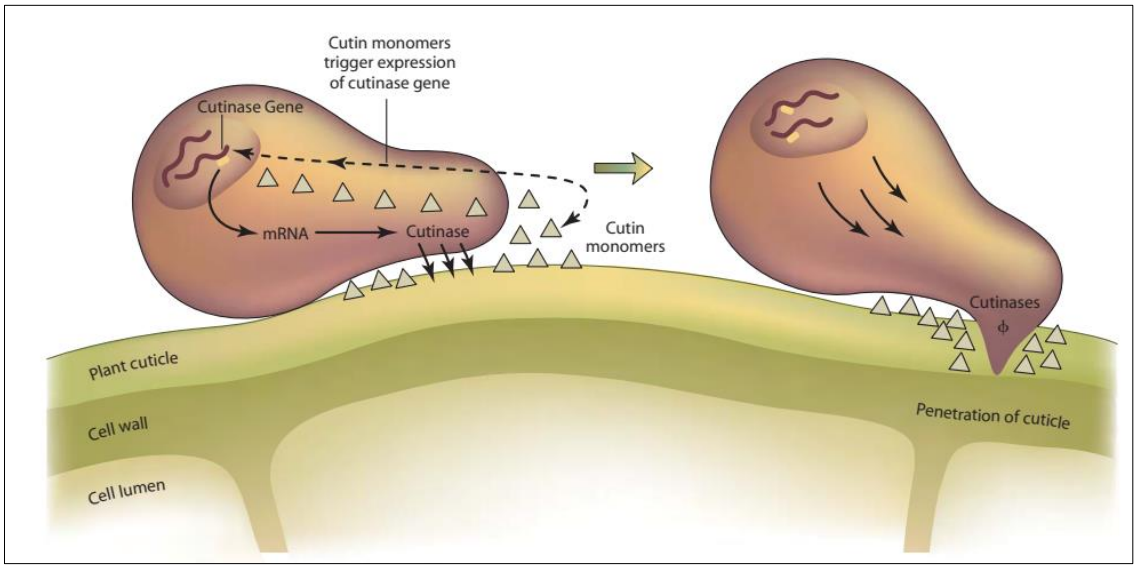


Types of reaction of plants to attacks by various pathogens in relation to the kind of resistance of the plant.

Plant pathogens are, generally, tiny microorganisms that cannot apply a "voluntary" force to a plant surface. Only some fungi, parasitic higher plants, and nematodes appear to apply mechanical pressure to the plant surface they are about to penetrate. The amount of pressure, however, may vary greatly with the degree of "presoftening" of a plant surface by enzymatic secretions of the pathogen. For fungi and parasitic higher plants to penetrate a plant surface, they must, generally, first adhere to it.

Hyphae and radicles are usually surrounded by mucilaginous substances, and their adhesion to the plant seems to be brought about primarily by the intermolecular forces developing between the surfaces of plant and pathogen on close contact with the adhesive substances and with one another. In some cases an adhesion pad forms from the spore when it comes in contact with a moist surface, and cutinase and cellulase enzymes released from the spore surface help the spore adhere to the plant surface. Spores of some fungi carry adhesive substances at their tips that, on hydration, allow spores to become attached to various surfaces.

After contact is established, the diameter of the tip of the hypha or radicle in contact with the host increases and forms the flattened, bulb-like structure called the appressorium. This increases the area of adherence between the two organisms and securely fastens the pathogen to the plant. From the appressorium, a fine growing point, called the penetration peg, arises and advances into and through the cuticle and cell wall. In some fungi, such as *Alternaria*, *Cochliobolus*, *Colletotrichum*, *Gaeumannomyces*, *Magnaporthe*, and *Verticillium*, penetration of the plant takes place only if melanin (dark pigment) accumulates in the appressorial cell wall. It appears that melanin produces a rigid structural layer and, by trapping solutes inside the appressorium, causes water to be absorbed. This increases the turgor pressure in the appressorium and, thereby, the physical penetration of the plant by the penetration peg. If the underlying host wall is soft, penetration occurs easily. When the underlying wall is hard, however, the force of the growing point may be greater than the adhesion force of the two surfaces and may cause separation of the appressorial and host walls, thus averting infection. Penetration of plant barriers by fungi and parasitic higher plants is almost always assisted by the presence of enzymes secreted by the pathogen at the penetration site, resulting in the softening or dissolution of the barrier. It was found, for example, that while appressoria of some powdery mildew fungi developed a maximum turgor pressure of 2–4 MPa, approximately sufficient to bring about host cell penetration, two cellulases were also present: one primarily at the tip of the appressorial germ tube and the other at the tip of the primary germ tube. While the penetration tube is passing through the cuticle, it usually attains its smallest diameter an appears thread-like. After penetration of the cuticle, the hyphal tube diameter often increases considerably. The penetration tube attains the diameter normal for the hyphae of the particular fungus only after it has passed through the cell wall (see Figs. 2-5 and 2-9 in Chapter 2). Nematodes penetrate plant surfaces by means of the stylet, which is thrust back and forth and exerts mechanical pressure on the cell wall (Fig. 2-10). The nematode first adheres to the plant surface by suction, which it develops by bringing its fused lips in contact with the plant. After adhesion is accomplished, the nematode brings its body, or at least the forward portion of its body, to a position vertical to the cell wall. With its head stationary and fixed to the cell wall, the nematode then thrusts its stylet forward while the rear part of its body sways or rotates slowly round and round. After several consecutive thrusts of the stylet, the cell wall is pierced, and the stylet or the entire nematode enters the cell.



Diagrammatic representation of cuticle penetration by a germinating fungus spore. Constitutive cuti- nase releases a few cutin monomers from the plant cuticle. These trigger expression of the cutinase genes of the fungus, leading to the production of more cutinase(s), which macerates the cuticle and allows penetration by the fungus.

Once a fungus or nematode has entered a cell, it generally secretes increased amounts of enzymes that presumably soften or dissolve the opposite cell wall and make its penetration easier. Mechanical force, however, probably is brought to bear in most such penetrations, although to a lesser extent. Considerable mechanical force is also exerted on host tissues from the inside out by some pathogenic fungi on formation of their fructifications in the tissues beneath the plant surface. Through increased pressure, the sporophore hyphae, as well as fruiting bodies, such as pycnidia and perithecia, push outward and cause the cell walls and the cuticle to expand, become raised in the form of blister-like proturberances, and finally break.

PREEXISTING STRUCTURAL AND CHEMICAL DEFENSES

Preexisting Defense Structures

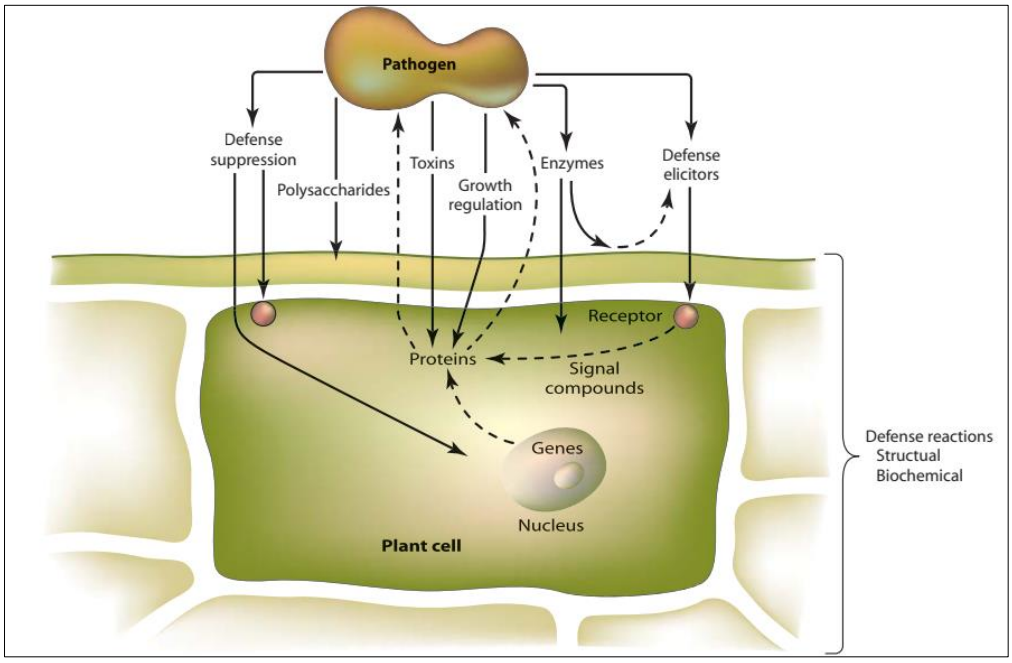
The first line of defense of a plant against pathogens is its surface, which the pathogen must adhere to and penetrate if it is to cause infection. Some structural defenses are present in the plant even before the pathogen comes in contact with the plant. Such structures include the amount and quality of wax and cuticle that cover the epidermal cells, the structure of the epidermal cell walls, the size, location, and shapes of stomata and lenticels, and the presence of tissues made of thick-walled cells that hinder the advance of the pathogen on the plant.

Waxes on leaf and fruit surfaces form a water-repellent surface, thereby preventing the formation of a film of water on which pathogens might be deposited and germinate (fungi) or multiply (bacteria). A thick mat of hairs on a plant surface may also exert a similar water-repelling effect and may reduce infection.

A thick cuticle may increase resistance to infection in diseases in which the pathogen enters its host only through direct penetration. Cuticle thickness, however, is not always correlated with resistance, and many plant varieties with cuticles of considerable thickness are invaded easily by directly penetrating pathogens.

The thickness and toughness of the outer wall of epidermal cells are apparently important factors in the resistance of some plants to certain pathogens. Thick, tough walls of epidermal cells make direct penetration by fungal pathogens difficult or impossible. Plants with such walls are often resistant, although if the pathogen is introduced beyond the epidermis of the same plants by means of a wound, the inner tissues of the plant are invaded easily by the pathogen.

Many pathogenic fungi and bacteria enter plants only through stomata. Although the majority of pathogens can force their way through closed stomata, some, like the stem rust of wheat, can enter only when stomata are open. Thus, some wheat varieties, in which the stomata open late in the day, are resistant because the germ tubes of spores germinating in the night dew desiccate due to evaporation of the dew before the stomata begin to open. The structure of the stomata, e.g., a very narrow entrance and broad, elevated guard cells, may also confer resistance to some varieties against certain of their bacterial pathogens.



The cell walls of the tissues being invaded vary in thickness and toughness and may sometimes inhibit the advance of the pathogen. The presence, in particular, of bundles or extended areas of sclerenchyma cells, such as are found in the stems of many cereal crops, may stop the further spread of pathogens such as stem rust fungi. Also, the xylem, bundle sheath, and sclerenchyma cells of the leaf veins effectively block the spread of some fungal, bacterial, and nematode pathogens that cause various "angular" leaf spots because of their spread only into areas between, but not across, veins. Xylem vessels seem to be involved more directly in the resistance and susceptibility to vascular diseases. For example, xylem vessel diameter and the proportion of large vessels were strongly correlated with the susceptibility of elm to Dutch elm disease caused by the fungus *Ophiostoma novoulni*.

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, hyper- sensitive cell death, ethylene, phytoalexins, pathogenesis-related proteins (hydrolytic enzymes, b-1,3-glucanases, chitinases), inhibitors (thionins, proteinase inhibitors, thaumatin-like proteins)

Preexisting Chemical Defenses

Although structural characteristics may provide a plant with various degrees of defense against attacking pathogens, it is clear that the resistance of a plant against pathogen attacks depends not so much on its structural barriers as on the substances produced in its cells before or after infection. This is apparent from the fact that a particular pathogen will not infect certain plant varieties even though no structural barriers of any kind seem to be present or to form in these varieties. Similarly, in resistant varieties, the rate of disease development soon slows down, and finally, in the absence of structural defenses, the disease is completely checked. Moreover, many pathogens that enter nonhost plants naturally or that are introduced into nonhost plants artificially, fail to cause infection, although no apparent visible host structures inhibit them from doing so. These examples suggest that defense mechanisms of a chemical rather than a structural nature are responsible for the resistance to infection exhibited by plants against certain pathogens.

Inhibitors Released by the Plant in Its Environment Plants exude a variety of substances through the surface of their aboveground parts as well as through the surface of their roots. Some of the compounds released by certain kinds of plants, however, seem to have an inhibitory action against certain pathogens. Fungitoxic exudates on the leaves of some plants, e.g., tomato and sugar beet, seem to be present in sufficient concentrations to inhibit the germination of spores of fungi *Botrytis* and *Cercospora*, respectively, that may be present in dew or rain droplets on these leaves. Similarly, in the case of onion smudge, caused by the fungus *Colletotrichum circinans*, resistant varieties generally have red scales and contain, in addition to the red pigments, the phenolic compounds protocatechuic acid and cate- chol. In the presence of water drops or soil moisture containing conidia of the onion smudge fungus on the surface of red onions, these two fungitoxic substances diffuse into the liquid, inhibit the germination of the conidia, and cause them to burst, thus protecting the plant from infection. Both fungitoxic exudates and inhibition of infection are missing in white-scaled, susceptible onion varieties (Fig. 6-2). It was noticed that applications of acibenzolar-S-methyl (ASM) on sun-flower reduced infection by the rust fungus *Puccinia helianthi* through the reduction of spore germination and appressorium formation. It was subsequently shown that ASM accomplished this by increasing the production and secretion by the plant on the leaf surface of coumarins and other toxic phenolics that inhibit spore germination and appressorium formation on the leaf surfaces on which they are present.

Inhibitors Present in Plant Cells before Infection

It is becoming increasingly apparent that some plants are resistant to diseases caused by certain pathogens because of one or more inhibitory antimicrobial com- pounds, known as phytoanticipins, which are present in the cell before infection. Several phenolic compounds, tannins, and some fatty acid-like compounds such as dienes, which are present in high concentrations in cells of young fruits, leaves, or seeds, have been proposed as responsible for the resistance of young tissues to pathogenic microorganisms such as Botrytis. For example, increased 9-hexadecanoic acid in cutin monomers in transgenic tomato plants led to resistance of such plants to powdery mildew because these cutin monomers inhibit the germination of powdery mildew spores. Many such compounds are potent inhibitors of many hydrolytic enzymes, including the pectolytic-macerating enzymes of plant pathogens. As the young tissues grow older, their inhibitor content and their resistance to infection decrease steadily. Strawberry leaves naturally contain (+)-catechin, which inhibits infection by *Alternaria alternata* by blocking the formation of infection hyphae from haustoria although it allows both spore germination and appressoria formation. Several other types of preformed compounds, such as the saponins (glycosylated steroidal or triterpenoid com- pounds) tomatine in tomato and avenacin in oats, not only have antifungal membranolytic activity, they actually exclude fungal pathogens that lack enzymes (saponinases) that break down the saponin from infecting the host. In this way, the presence or absence of saponin in a host and of saponinase in a fungus deter-mines the host range of the fungus.

In addition to the simple molecule antifungal com- pounds listed earlier, several preformed plant proteins have been reported to act as inhibitors of pathogen proteinases or of hydrolytic enzymes involved in host cell wall degradation, to inactivate foreign ribosomes, or to increase the permeability of the plasma membranes of fungi.

For example, in a number of plants there is a family of low molecular weight proteins called phytocystatins that inhibit cysteine proteinases carried in the digestive system of nematodes and are also secreted by some plant pathogenic fungi. Constitutively present or transgenically introduced phytocystatins in plants reduce the size of nematode females and the number of eggs produced by females, thereby providing effective or significant control of several plants to root knot, cyst, reniform, and lesion nematodes.

Another type of compounds, the lectins, which are proteins that bind specifically to certain sugars and occur in large concentrations in many types of seeds, cause lysis and growth inhibition of many fungi. However, plant surface cells also contain variable amounts of hydrolytic enzymes, some of which, such as glucanases and chitinases, may cause the breakdown of pathogen cell wall components, thereby contributing to resistance to infection. The importance of either of these types of inhibitors to disease resistance is not currently known, but some of these substances are known to increase rapidly upon infection and are considered to play an important role in the defense of plants to infection.

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 nonhost? 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 sensi- tive sites in the cell. Only plants that have such sensitive receptors or sites become diseased. Plants of other vari- eties 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 but not a resistant plant 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, however, 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, though, 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

INDUCED STRUCTURAL AND BIOCHEMICAL DEFENSES

Erwinia carotovora var. *atroseptica*, is less severe on potatoes with low-reducing sugar content than on pota- toes high in reducing sugars.

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 bio- chemical 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. 6-3).

Pathogen Elicitors

Various pathogens, especially fungi and bacteria, release a variety of substances in their immediate environment that act as nonspecific elicitors

of pathogen recognition by the host. Such nonspecific 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, 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 poly- saccharides 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 sub- sequent attacks with the fungus. The elicitor alone in germination of Spores and Seeds Almost all pathogens in their vegetative state are capable of initiating infection immediately. Fungal spores and seeds of parasitic higher plants, however, must first germinate (Figs. 2-4 and 2-5). Spores germinate by producing a typical mycelium that infects and grows into host plants or they produce a short germ tube that produces a specialized infectious structure, the haustorium (Figs. 2-4B–2-4D). In order to germinate, spores require a favorable temperature and also moisture in the form of rain, dew, or a film of water on the plant surface or at least high relative humidity. The moist conditions must last long enough for the pathogen to penetrate or else it desiccates and dies. Most spores can germinate immediately after their maturation and release, but others (so-called resting spores) require a dormancy period of varying duration before they can germinate. When a spore germinates it produces a germ tube, i.e., the first part of the mycelium, that can penetrate the host plant. Some fungal spores germinate by producing other spores, e.g., sporangia produce zoospores and teliospores produce basidiospores.

Spore germination is often favored by nutrients dif- fusing from the plant surface; the more nutrients (sugars and amino acids) exuded from the plant, the more spores germinate and the faster they germinate. In some cases, spore germination of a certain pathogen is stimulated only by exudates of plants susceptible to that particular pathogen. In other cases, spore germination may be inhibited

STAGES IN THE DEVELOPMENT OF DISEASE: THE DISEASE CYCLE

released into the surrounding water by the plant, by substances contained within the spores themselves, especially when the spores are highly concentrated (“quorum sensing”), and by saprophytic microflora present on or near the plant surface.

Fungi in soil coexist with a variety of antagonistic microorganisms that cause an environment of starvation and of toxic metabolites. As a result, spores of many soilborne fungi are often unable to germinate in some soils, and this phenomenon is called fungistasis, or their germ tubes lyse rapidly. Soils in which such events occur are known as suppressive soils. Fungistasis, however, is generally counteracted by root exudates of host plants growing nearby, and the spores are then able to germinate and infect.

After spores germinate, the resulting germ tube must grow, or the motile secondary spore (zoospore) must move, toward a site on the plant surface at which successful penetration can take place.The number, length, and rate of growth of germ tubes, or the number and mobility of motile spores, may be affected by physical conditions, such as temperature and moisture, by the kind and amount of exudates the plant produces at its surface, and by the saprophytic microflora.

The growth of germ tubes in the direction of successful penetration sites seems to be regulated by several factors, including greater humidity or chemical stimuli associated with such openings as wounds, stomata, and lenticels; thigmotropic (contact) responses to the topography of the leaf surface, resulting in germ tubes growing at right angles to cuticular ridges that generally surround stomata and thus eventually reaching a stoma; and nutritional responses of germ tubes toward greater concentrations of sugars and amino acids present along roots. The direction of movement of motile spores (zoospores) is also regulated by similar factors, namely chemical stimuli emanating from stomata, wounds, or the zone of elongation of roots, physical stimuli related to the structure of open stomata, and the nutrient gradient present in wound and root exudates.

Seeds germinate by producing a radicle, which either penetrates the host plant directly or first produces a small plant that subsequently penetrates the host plant by means of specialized feeding organs called haustoria. Most conditions described earlier as affecting spore germination and the direction of growth of germ tubes also apply to seeds. Haustoria are also produced by many fungi.

Hatching of Nematode Eggs

Nematode eggs also require conditions of favorable temperature and moisture to become activated and hatch. In most nematodes, the egg contains the first juvenile stage before or soon after the egg is laid. This

juvenile immediately undergoes a molt and gives rise to the second juvenile stage, which may remain dormant in the egg for various periods of time. Thus, when the egg finally hatches, it is the second-stage juvenile that emerges, and it either finds and penetrates a host plant or undergoes additional molts that produce further juvenile stages and adults.

Once nematodes are in close proximity to plant roots, they are attracted to roots by certain chemical factors associated with root growth, particularly carbon dioxide and some amino acids. These factors may diffuse through soil and may have an attractant effect on nematodes present several centimetres away from the root. Nematodes are generally attracted to roots of both host and nonhost plants, although there may be some cases in which nematodes are attracted more strongly to the roots of host plants.

Penetration

Pathogens penetrate plant surfaces by direct penetration of cell walls, through natural openings, or through wounds. Some fungi penetrate tissues in only one of these ways, others in more than one. Bac- teria enter plants mostly through wounds, less frequently through natural openings, and never directly through unbroken cell walls. Viruses, viroids, mollicutes, fastidious bacteria, and protozoa enter through wounds made by vectors, although some viruses and viroids may also enter through wounds made by tools and other means. Parasitic higher plants enter their hosts by direct penetration. Nematodes enter plants by direct penetration and, sometimes, through natural openings.

Penetration does not always lead to infection. Many organisms actually penetrate cells of plants that are not susceptible to these organisms and that do not become diseased; these organisms cannot proceed beyond the stage of penetration and die without producing disease.

Direct Penetration through Intact Plant Surfaces

Direct penetration through intact plant surfaces is probably the most common type of penetration by fungi, oomycetes, and nematodes and the only type of penetration by parasitic higher plants. None of the other pathogens can enter plants by direct penetration.

Of the fungi that penetrate their host plants directly, the hemibiotrophic, i.e., nonobligate parasitic ones, do so through a fine hypha produced directly by the spore or mycelium.

PARASITISM AND DISEASE DEVELOPMENT

obligately parasitic ones do so through a penetration peg produced by an appressorium. The fine hypha or appressorium is formed at the point of contact of the germ tube or mycelium with a plant surface. The fine hypha grows toward the plant surface and pierces the cuticle and the cell wall through mechanical force and enzymatic softening of the cell wall substances. Most fungi, however, form an appressorium at the end of the germ tube, with the appressorium usually being bulbous or cylindrical with a flat surface in contact with the surface of the host plant. Then, a penetration peg grows from the flat surface of the appressorium toward the host and pierces the cuticle and the cell wall. The penetration peg grows into a fine hypha generally much smaller in diameter than a normal hypha of the fungus, but it regains its normal diameter once inside the cell. In most fungal diseases the fungus penetrates the plant cuticle and the cell wall, but in some, such as apple scab, the fungus penetrates only the cuticle and stays between the cuticle and the cell wall.

Parasitic higher plants also form an appressorium and penetration peg at the point of contact of the radicle with the host plant, and penetration is similar to that in fungi. Direct penetration in nematodes is accomplished by repeated back-and-forth thrusts of their stylets. Such thrusts finally create a small opening in the cell wall; the nematode then inserts its stylet into the cell or the entire nematode enters the cell.

Penetration through Wounds

All bacteria, most fungi, some viruses, and all viroids can enter plants through various types of wounds. Some viruses and all mollicutes, fastidious vascular bacteria, and protozoa enter plants through wounds made by their vectors. The wounds utilized by bacteria and fungi may be fresh or old and may consist of lacerated or killed tissue. These pathogens may grow briefly on such tissue before they advance into healthy tissue. Laceration or death of tissues may be the result of environmental factors such as wind breakage and hail; animal feeding, e.g., by insects and large animals; cultural practices of humans, such as pruning, trans- planting, and harvesting; self-inflicted injuries, such as leaf scars; and, finally, wounds or lesions caused by other pathogens. Bacteria and fungi penetrating through wounds germinate or multiply in the wound sap or in a film of rain or dew water present on the wound. Subsequently, the pathogen invades adjacent plant cells or it secretes enzymes and toxins that kill and macerate the nearby cells.

The penetration of viruses, mollicutes, fastidious bacteria, and protozoa through wounds depends on the deposition of these pathogens by their vectors in fresh wounds created at the time of inoculation. All four types of pathogens are transmitted by certain types of insects. Some viruses are also transmitted by certain nematodes, mites, and fungi. Some viruses and viroids are transmitted through wounds made by human hands and tools. In most cases, however, these pathogens are carried by one or a few kinds of specific vectors and can be inoculated successfully only when they are brought to the plant by these particular vectors.

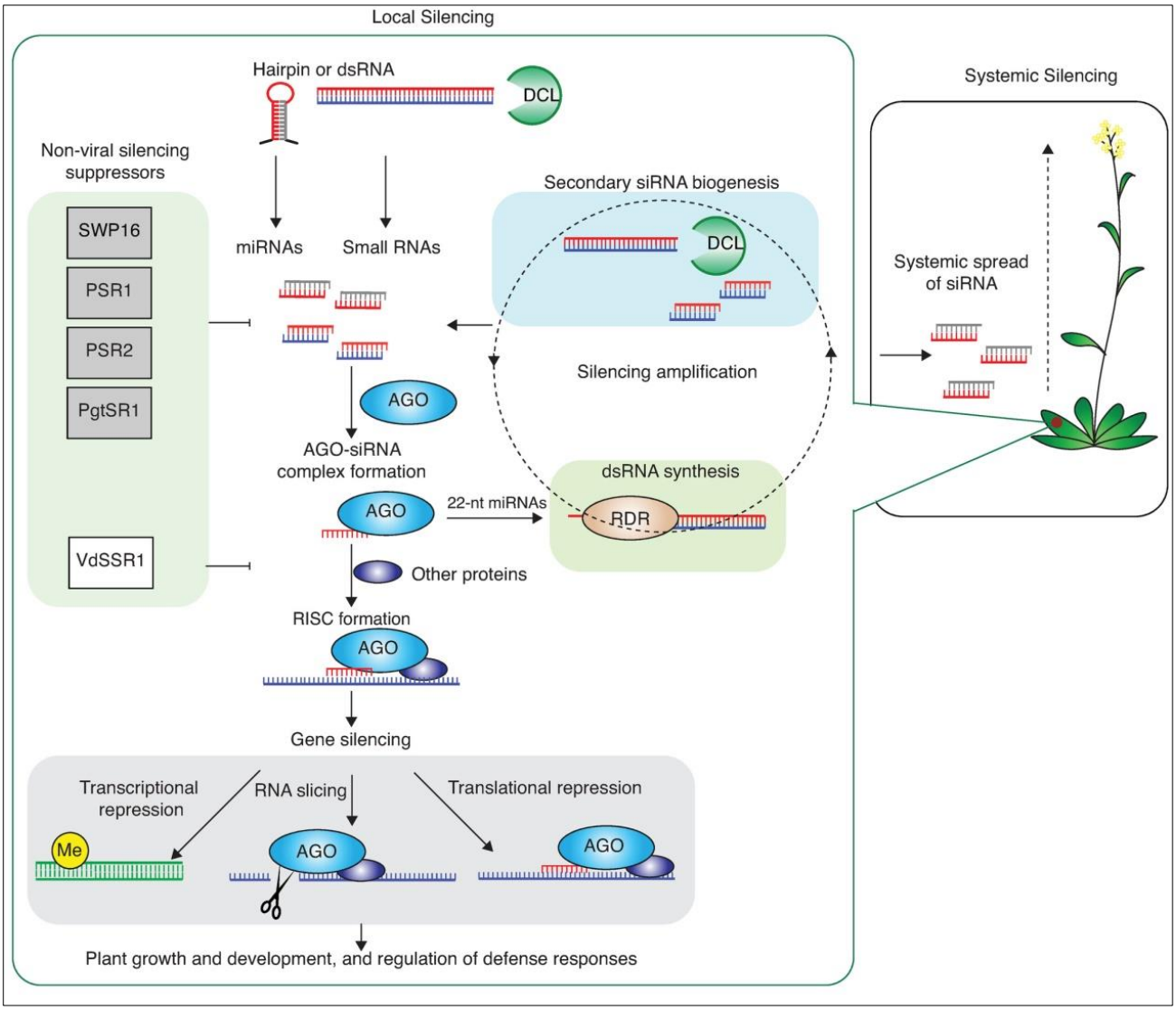
Penetration through Natural Openings

Many fungi and bacteria enter plants through stomata, and some enter through hydathodes, nectarthodes, and lenticels. Stomata are most numerous on the lower side of leaves. They measure about 10–20 by 5–8 mm and are open in the daytime but are more or less closed at night. Bacteria present in a film of water over a stoma and, if water soaking occurs, can swim through the stoma easily and into the substomatal cavity where they can multiply and start infection. Fungal spores generally germinate on the plant surface, and the germ tube may then grow through the stoma. Frequently, however, the germ tube forms an appressorium that fits tightly over the stoma, and usually one fine hypha grows from it into the stoma. In the sub- stomatal cavity the hypha enlarges, and from it grow one or several small hyphae that actually invade the cells of the host plant directly or by means of haustoria. Although some fungi can apparently penetrate.

Suppressors of Plant Defense Responses

It has been shown that at least some plant pathogenic fungi, e.g., *Puccinia graminis f. sp. tritici*, which causes stem rust of wheat, and *Mycosphaerella pinodes*, which causes a leaf spot on pea, produce substances called suppressors that act as pathogenicity factors by sup- pressing the expression of defense responses in the host plant. The defense suppressor of the wheat stem rust fungus has been found in the fungus germination fluid and in the intercellular fluid of rust-infected wheat leaves. This suppressor interacts with the wheat cell plasma membrane and reduces binding of the pathogen's 67-kDa glycoprotein elicitor of host defenses to the plasma membrane. In this way, the suppressor molecule suppresses the activity of phenylalanine lyase (PAL) and the normal development of defense responses. The pea- infecting fungus produces two suppressors in the spore germination fluid. Both suppressors are glycopeptides, counteract the elicitor of phytoalexin biosynthesis, and temporarily suppress the expression of all defense reactions of the host plant. The *Mycosphaerella* suppressors seem to reduce the proton-pumping activity of the host cell membrane ATPase and thereby temporarily lower the ability of the cell to function and to defend itself. A different mechanism of suppression of plant defense responses has been reported in the ergot disease of rye caused by the fungus *Claviceps purpurea*. In that disease the fungus produces the enzyme catalase, which reacts with and neutralizes the hydrogen peroxide that is produced as one of the first defense responses of plants against infecting pathogens. The fungal catalase con- centration is greatest at hyphal walls and hyphal sur- faces and is secreted by the fungus into the host apoplast at the host–pathogen interface, where the host H_2O_2 is produced. By inactivating active oxygen species produced by the host through catalase, the fungus sup- presses the host defenses. Compounds present in the cell, despite the fact that no such compound can be found in infected cells. How viruses cause disease remains, therefore, pretty much a mystery but some facts are beginning to emerge.

One of the most important proteins coded by viruses that plays an important role in their pathogenicity and virulence is their coat protein. In addition to protecting the viral nucleic acid from external damaging factors, the coat protein plays important roles in practically everything pertaining to viral replication and dis- semination. Thus, the coat protein plays a role in host recognition, uncoating and release of the nucleic acid, assistance in replication of the nucleic acid, movement of the virus between cells and organs, movement of the virus via a vector between plants, and modification of symptoms. Again, little is known on the mechanisms by which the coat protein affects these functions.



Silencing suppressors of plant pathogens. The diagram illustrates the general gene silencing pathway in plants initiated by double-stranded (ds) RNA, the biogenesis of primary and secondary small interfering RNAs (siRNAs), RNA-induced silencing complex (RISC) formation, and gene silencing by transcriptional repression, RNA slicing, or translational repression. Biogenesis of secondary siRNAs is triggered by some 22-nucleotide (nt) micro-RNAs (miRNAs) and includes the formation of dsRNA by cellular RNA-dependent RNA polymerases. Systemic silencing involves the systemic spread of small interfering RNAs (siRNAs). Nonviral silencing suppressors described to date interfere with the biogenesis or activity of endogenous plant siRNAs. VdSSR1 inhibits the nuclear export of AGO1–miRNA complexes.

Another viral protein that has been studied extensively is the so-called movement protein, which enables viruses to move between cells and/or through the phloem system of the plant by altering the properties of plasmodesmata. However, some movement proteins not only open movement channels for the virus, they also block a defense molecule, the suppressor of virus silencing by the plant cell activated by the viral infection. Some viroids seem to form complexes with certain host proteins that help the viroids pass through plasmodesmata and with plant lectins that help viroids move through the phloem of host plants.

Whatever the Plant Defense or Resistance, It Is Controlled by Its Genes

One concept that must be made clear at the outset is that whatever the kind of defense or resistance a host plant employs against a pathogen or against an abiotic agent, it is ultimately controlled, directly or indirectly, by the genetic material (genes) of the host plant and of the pathogen.

Nonhost Resistance

A plant may find it easy to defend itself, i.e., to stay resistant (immune) when it is brought in contact with a pathogenic biotic agent to which the plant is not a host. This is known as nonhost resistance and is the most common form of resistance (or defense from attack) in nature. For example, apple trees are not affected by

pathogens of tomato, of wheat, or of citrus trees because the genetic makeup of apple is in some way(s) different from that of any other kinds of host plants, which, of course, are attacked by their own pathogens. However, apple can be attacked by its own pathogens, which, in turn, do not attack tomato, wheat, citrus, or anything else. Similarly, the fungus that causes powdery mildew on wheat (Blumeria graminis f. sp. tritici) does not infect barley and vice versa, the fungus that causes powdery mildew on barley (B. graminis f. sp. hordei) does not infect wheat, and so on. All such unsuccessful plant/ pathogen interactions are thought to represent nonhost resistance. It has been shown recently however, that in at least some related pairings, e.g., the wheat, powdery mildew fungus inoculated on barley, the fungus produces haustoria and the host reacts by producing hydrogen peroxide (H_2O_2), cell wall appositions under the appressoria, and a hypersensitive response in which epidermal cells die rapidly in response to fungal attack.

Partial, Polygenic, Quantitative, or Horizontal Resistance

Each plant, of course, is attacked by its own pathogens, but there is often a big difference in how effectively the plant can defend itself (how resistant the plant is) against each pathogen. Even when conditions for infection and disease development are favorable, a plant, upon infection with a particular pathogen, may develop no disease, only mild disease, or severe disease, depending on the specific genetic makeup of the plant and of the pathogen that attacks it. Many genes are involved in keeping a plant protected from attack by pathogens. Many of these genes provide for the general upkeep and well-being functions of plants, but plants also have many genes whose main functions seem to be the protection of plants from pathogens. Some of the latter plant genes code for chemical substances that are toxic to pathogens or neutralize the toxins of the pathogens, and these substances may be present in plants regardless of whether the plant is under attack or not. Plants also have genes that produce and regulate the formation of structures that can slow down or stop the advance of a pathogen into the host and cause disease. These structures can also be present in a plant throughout its life or they may be produced in response to attack by one of several pathogens or following injury by an abiotic agent. Preexisting defense structures or toxic chemical substances, and many of those formed in response to attack by a pathogen or abiotic agent, are important in the defense of most plants against most pathogens.

When a pathogen attacks a host plant, the genes of the pathogen are activated, produce, and release all their weapons of attack (enzymes, toxins, etc.) against the plants that they try to infect. With the help of different combinations of preexisting or induced toxic chemical substances or defense structures, most plants manage to defend themselves partially or nearly completely. Such plants show sufficient resistance that allows them to survive the pathogen attacks and to produce a satisfactory yield. This type of defense or resistance is known as polygenic, general, or quantitative resistance because it depends on many genes for the presence or formation of the various defense structures and for preexisting or induced production of many substances toxic to the pathogen. This type of resistance is present at different levels against different pathogens in absolutely all plants and is also known as partial, quantitative, horizontal, multigenic, field, durable, or minor gene resistance.

Most plants depend on general resistance against their pathogens, especially nonobligate parasites, e.g., the semi biotrophic or nectrotrophic oomycetes Pythium and Phytophthora, the fungi *Botrytis*, *Fusarium*, *Sclerotinia*, and *Rhizoctonia*, and most bacteria, nematodes, and so on. In at least some polygenic plant pathogen combinations, such as the early blight of tomato caused by the necrotrophic fungus *Alternaria solani*, the more resistant the varieties are, the higher the constitutive concentration and the more rapid the accumulation in them of pathogen-induced pathogenesis related (PR) proteins, than in susceptible varieties. These PR proteins include some of the specific antifungal isozymes of chitinase and β-1,3-glucanase. Also, total enzyme preparations from resistant varieties were able to release elicitors of the hypersensitive response (HR) (see later) from purified fungal cell walls, whereas enzymes from susceptible varieties could not. Furthermore, partially purified chitinases from tomato leaves could release HR elicitors from germinating *A. solani* spores but not from mature intact cell walls. This suggests that, perhaps, constitutively produced hydrolytic enzymes may act as a mechanism of elicitor release in tomato resistance to the early blight disease. Quantitative resistance has also been shown to increase in transgenic plants carrying introduced R genes and matching avirulence genes, even though the latter do not express the hypersensitive cell death.

Race-Specific, Monogenic, R Gene, or Vertical Resistance

In many plant–pathogen combinations, especially those involving biotrophic oomycetes (downy mildews), fungi (powdery mildews, rusts), and many other fungi, e.g., *Cochliobolus, Magnaporthe, Cladosporium*, many bacteria, nematodes, and viruses, defense (resistance) of a host plant against many of its pathogens is through the presence of matching pairs of juxtaposed genes for disease in the host plant and the pathogen. The host plant carries one or few resistance genes (R) per pathogen capable of attacking it, while each pathogen carries matching genes for avirulence (A) for each of the R genes of the host plant. As explained in some detail later, the avirulence gene of the pathogen serves to trigger the host R gene into action. This then sets in motion a series of defense reactions that neutralize and eliminate the specific pathogen that carries the corresponding (matching) gene for avirulence (A), while

the attacked and a few sur- rounding cells die. This type of defense or resistance is known as race-specific, hypersensitive response (HR), major gene, R gene, or vertical resistance. However, some R genes, e.g., Xa21 of rice, do not induce a visible HR.

CONCLUSION

In plant pathology, certain traditional approaches are used to detect and identify the plant pathogens. These approaches are: (i) Symptomatology, (ii) Host Range, (iii) Morphology of the Pathogen, and (iv) Isolation of Pathogen onto Selective Culture Media. The host–pathogen interaction is described as the molecular, cellular, organismal, or population-level sustenance of bacteria or viruses within host organisms. This phrase is widely used to describe to bacteria that cause disease, however they may not infect all hosts. As a result, the concept has been expanded to include how infections persist within their host, regardless of whether they cause disease. The host–pathogen interaction is defined as how microbes or viruses sustain themselves within host organisms on a molecular, cellular, organismal or population level.

This term is most commonly used to refer to disease causing microorganisms although they may not cause illness in all hosts.[1] Because of this, the definition has been expanded to how known pathogens survive within their host, whether they cause disease or not. On the molecular and cellular level, microbes can infect the host and divide rapidly, causing disease by being there and causing a homeostatic imbalance in the body, or by secreting toxins which cause symptoms to appear. Viruses can also infect the host with virulent DNA, which can affect normal cell processes (transcription, translation, etc.), protein folding, or evading the immune response.

Conflicts of Interest: The authors declare no conflict of interest

SELECTED REFERENCES

- Alfano, J. R., & Guo, M. (2003). The *Pseudomonas syringae* Hrp (type III) protein secretion system: Advances in the new millenium. In “Plant-Microbe Interactions” (G. Stacey and N. T. Keen, eds.), 6, 227–258.
- Bai, J., Choi, S. H., Ponciano, G., Leung, H., & Leach, J. E. (2000). Xanthomonas oryzae pv. oryzae avirulence genes contribute differently and specifically to pathogen aggressiveness. *Molecular plant-microbe interactions*, *13*(12), 1322-1329.
- Barras, F., van Gijsegem, F., & Chatterjee, A. K. (1994). Extracellular enzymes and pathogenesis of soft-rot Erwinia. *Annual review of phytopathology*, *32*(1), 201-234.
- Belding, R. D., Sutton, T. B., Blankenship, S. M., & Young, E. (2000). Relationship between apple fruit epicuticular wax and growth of Peltaster fructicola and Leptodontidium elatius, two fungi that cause sooty blotch disease. *Plant Disease*, *84*(7), 767-772.
- Blanchette, R. A. (1991). Delignification by wood-decay fungi. *Annual review of phytopathology*, *29*(1), 381-403.
- Bostock, R. M., Wilcox, S. M., Wang, G., & Adaskaveg, J. E. (1999). Suppression ofMonilinia fructicolacutinase production by peach fruit surface phenolic acids. *Physiological and molecular plant pathology*, *54*(1-2), 37-50.
- Brown, H. P., Panshing, A. J., & Forsaith, C. C. (1949). “Textbook of Wood Technology,” Vol. 1. McGraw-Hill, New York.
- Burr, T. J., & Otten, L. (1999). Crown gall of grape: biology and disease management. *Annual review of phytopathology*, *37*(1), 53-80.
- Callaway, A., Giesman-Cookmeyer, D., Gillock, E. T., Sit, T. L., & Lommel, S. A. (2001). The multifunctional capsid proteins of plant RNA viruses. *Annual review of phytopathology*, *39*(1), 419-460.
- Cao, H., Baldini, R. L., & Rahme, L. G. (2001). Common mechanisms for pathogens of plants and animals. *Annual review of phytopathology*, *39*(1), 259-284.
- Carzaniga, R., Fiocco, D., Bowyer, P., & O'Connell, R. J. (2002). Localization of melanin in conidia of Alternaria alternata using phage display antibodies. *Molecular plant-microbe interactions*, *15*(3), 216-224.
- Chen, Z., Kloek, A. P., Boch, J., Katagiri, F., & Kunkel, B. N. (2000). The Pseudomonas syringae avrRpt2 gene product promotes pathogen virulence from inside plant cells. *Molecular Plant-Microbe Interactions*, *13*(12), 1312-1321.
- Comai, L. U. C. A., Surico, G., & Kosuge, T. (1982). Relation of plasmid DNA to indoleacetic acid production in different strains of Pseudomonas syringae pv. savastanoi. *Microbiology*, *128*(9), 2157-2163.
- Culver, J. N. (2002). Tobacco mosaic virus assembly and disassembly: determinants in pathogenicity and resistance. *Annual review of phytopathology*, *40*(1), 287-308.
- Cutler, D. F., Alvin, K. L., & Price, C. E., eds. (1982). “The Plant Cuticle.” Linn. Soc. Symp. Ser. No. 10. Academic Press, London.
- Daly, J. M., & Deverall, B. J. eds. (1983). “Toxins in Plant Pathogenesis.” Academic Press, New York.
- Dardick, C. D., Golem, S., & Culver, J. N. (2000). Susceptibility and symptom development in Arabidopsis thaliana to Tobacco mosaic virus is influenced by virus cell-to-cell movement. *Molecular Plant-Microbe Interactions*, *13*(10), 1139-1144.
- Daub, M. E., & Ehrenshaft, M. (2000). The photoactivated Cercospora toxin cercosporin: contributions to plant disease and fundamental

biology. *Annual review of phytopathology*, *38*(1), 461-490.

- Davis, E. L., Hussey, R. S., Baum, T. J., Bakker, J., Schots, A., Rosso, M. N., & Abad, P. (2000). Nematode parasitism genes. *Annual review of phytopathology*, *38*(1), 365-396.
- Denny, T. P. (1995). Involvement of bacterial polysaccharides in plant pathogenesis. *Annual review of phytopathology*, *33*(1), 173-197.
- Dow, M., Newman, M. A., & Von Roepenack, E. (2000). The induction and modulation of plant defense responses by bacterial lipopolysaccharides. *Annual review of phytopathology*, *38*(1), 241-261.
- Doyle, E. A., & Lambert, K. N. (2002). Cloning and characterization of an esophageal-gland-specific pectate lyase from the root-knot nematode Meloidogyne javanica. *Molecular Plant-Microbe Interactions*, *15*(6), 549-556.
- Durbin, R. D. (1991). Bacterial phytotoxins: Mechanisms of action. *Experientia*, *47*(8), 776-783.
- Fridborg, I., Grainger, J., Page, A., Coleman, M., Findlay, K., & Angell, S. (2003). TIP, a novel host factor linking callose degradation with the cell-to-cell movement of Potato virus X. *Molecular plant-microbe interactions*, *16*(2), 132-140.
- Fritig, B., & LeGrand, M. (1993). "Mechanisms of Plant Defense Responses." Kluwer, Dordrecht, The Netherlands.
- Fry, B. A., & Loria, R. (2002). Thaxtomin A: evidence for a plant cell wall target. *Physiological and Molecular Plant Pathology*, *60*(1), 1-8.
- Gevens, A., & Nicholson, R. L. (2000). Cutin composition: A subtle role for fungal cutinase? Physiol. *Mol. Plant Pathol*, *57*, 43–45.
- Gheysen, G., & Fenoll, C. (2002). Gene expression in nematode feeding sites. *Annual review of phytopathology*, *40*(1), 191-219.
- Glenn, A. E., Gold, S. E., & Bacon, C. W. (2002). Fdb1 and Fdb2, Fusarium verticillioides loci necessary for detoxification of preformed antimicrobials from corn. *Molecular Plant-Microbe Interactions*, *15*(2), 91-101.
- Goethals, K., Vereecke, D., Jaziri, M., Van Montagu, M., & Holsters, M. (2001). Leafy gall formation by Rhodococcus fascians. *Annual review of phytopathology*, *39*(1), 27-52.
- Gold, S. E. (2003). *Ustilago* pathogenicity. In "Plant-Microbe Interactions" (G. Stacey and N. T. Keen, eds.), 6, 147–172.
- Gold, S. E., García-Pedrajas, M. D., & Martínez-Espinoza, A. D. (2001). New (and used) approaches to the study of fungal pathogenicity. *Annual review of phytopathology*, *39*(1), 337-365.
- Goodman, R. N., Kiraly, Z., & Zaitlin, M. (1967)." The Biochemistry and Physiology of Infectious Plant Disease." Van Nostrand- Reinhold, Princeton, NJ.
- Graniti, A. (1989). "Phytotoxins and Plant Pathogenesis." Springer-Verlag, Berlin.
- Hammerschmidt, R. (1999). Phytoalexins: what have we learned after 60 years?. *Annual review of phytopathology*, *37*(1), 285-306.
- Harper, G., Hull, R., Lockhart, B. E. N., & Olszewski, N. (2002). Viral sequences integrated into plant genomes. *Annual review of Phytopathology*, *40*(1), 119-136.
- He, S. Y. (1998). Type III protein secretion systems in plant and animal pathogenic bacteria. *Annu. Rev. Phytopathol*, *36*, 363–392.
- Henson, J. M., Butler, M. J., & Day, A. W. (1999). The dark side of the mycelium: melanins of phytopathogenic fungi. *Annual review of phytopathology*, *37*(1), 447-471.
- Hiriart, J. B., Aro, E. M., & Lehto, K. (2003). Dynamics of the VIGS-mediated chimeric silencing of the Nicotiana benthamiana ChlH gene and of the tobacco mosaic virus vector. *Molecular plant-microbe interactions*, *16*(2), 99-106.
- Hooykaas, P. J., & Beijersbergen, A. G. (1994). The virulence system of Agrobacterium tumefaciens. *Annual review of phytopathology*, *32*(1), 157-181.
- Hussey, R. S. (1989). Disease-inducing secretions of plant-parasitic nematodes. *Annual review of phytopathology*, *27*(1), 123-141.
- Jahr, H., Dreier, J., Meletzus, D., Bahro, R., & Eichenlaub, R. (2000). The endo-β-1, 4-glucanase CelA of Clavibacter michiganensis subsp. michiganensis is a pathogenicity determinant required for induction of bacterial wilt of tomato. *Molecular Plant-Microbe Interactions*, *13*(7), 703-714.
- Keen, N. T. (2000). A century of plant pathology: a retrospective view on understanding host-parasite interactions. *Annual review of phytopathology*, *38*(1), 31-48.
- Kolattukudy, P. E. (1985). Enzymatic penetration of the plant cuticle by fungal pathogens. *Annual Review of Phytopathology*, *23*(1), 223-250.
- Kosuge, T., & Nester, E. W. eds. (1984). "Plant-Microbe Interactions: Molecular and Genetic Perspective," 1. Macmillan, New York.
- Kuriger, W. E., & Agrios, G. N. (1977). Cytokinin levels and kinetin-virus interactions in tobacco ringspot virus infected cowpea plants. *Phytopathology*, *67*, 604-609.
- Manulis, S., & Barash, I. (2003). Molecular basis for transformation of an epiphyte into a gall-forming pathogen as exemplified by Erwinia herbicola pv. gypsophilae. *Plant-microbe interactions: Vol. 6*, 19-52.
- Marcell, L. M., & Beattie, G. A. (2002). Effect of leaf surface waxes on leaf colonization by Pantoea agglomerans and Clavibacter michiganensis. *Molecular plant-microbe interactions*, *15*(12), 1236-1244.

- Markham, J. E., & Hille, J. (2001). Host-selective toxins as agents of cell death in plant–fungus interactions. *Molecular plant pathology*, *2*(4), 229-239.
- Martin-Hernandez, A. M., Dufresne, M., Hugouvieux, V., Melton, R., & Osbourn, A. (2000). Effects of targeted replacement of the tomatinase gene on the interaction of Septoria lycopersici with tomato plants. *Molecular plant-microbe interactions*, *13*(12), 1301-1311.
- Morrissey, J. P., Wubben, J. P., & Osbourn, A. E. (2000). Stagonospora avenae secretes multiple enzymes that hydrolyze oat leaf saponins. *Molecular plant-microbe interactions*, *13*(10), 1041-1052.
- Navarre, D. A., & Wolpert, T. J. (1999). Effects of light and CO2 on victorin-induced symptom development in oats. *Physiol. Mol. Plant Pathol*, *55*, 237–242.
- Otani, H., Kohnobe, A., Kodama, M., & Kohmoto, K. (1998). Production of a host-specific toxin by germinating spores ofAlternaria brassicicola. *Physiological and Molecular Plant Pathology*, *52*(5), 285-295.
- Owens, R. A., Blackburn, M., & Ding, B. (2001). Possible involvement of the phloem lectin in long-distance viroid movement. *Molecular plant-microbe interactions*, *14*(7), 905-909.
- Petrini, O., & Ouellette, G. B. (1994). "Host Wall Alterations by Parasitic Fungi." APS Press, St. Paul, MN.Prusky, D., McEvoy, J. L., Leverentz, B. (2001). Local modulation of host pH by *Colletotrichum* species as a mechanism to increase virulence. *Mol. Plant-Microbe Interact*, *14*, 1105–1113.
- Pryce-Jones, E. M. I. L. Y., Carver, T. I. M., & GURR, S. J. (1999). The roles of cellulase enzymes and mechanical force in host penetration by Erysiphe graminis f. sp. hordei. *Physiological and Molecular Plant Pathology*, *55*(3), 175-182.
- Quidde, T., Osbourn, A. E., & Tudzynski, P. (1998). Detoxification of α-tomatine byBotrytis cinerea. *Physiological and Molecular Plant Pathology*, *52*(3), 151-165.
- Rantakari, A., Virtaharju, O., Vähämiko, S., Taira, S., Palva, E. T., Saarilahti, H. T., & Romantschuk, M. (2001). Type III secretion contributes to the pathogenesis of the soft-rot pathogen Erwinia carotovora: partial characterization of the hrp gene cluster. *Molecular plant-microbe interactions*, *14*(8), 962-968.
- Ream, W. (1989). Agrobacterium tumefaciens and interkingdom genetic exchange. *Annual Review of Phytopathology*, *27*(1), 583-618.
- Robison, M. M., Griffith, M., Pauls, K. P., & Glick, B. R. (2001). Dual role for ethylene in susceptibility of tomato to Verticillium wilt. *Journal of Phytopathology*, *149*(7-8), 385-388.
- Schäfer, W. (1994). Molecular mechanisms of fungal pathogenicity to plants. *Annual review of phytopathology*, *32*(1), 461-477.
- Schell, M. A. (2000). Control of virulence and pathogenicity genes of Ralstonia solanacearum by an elaborate sensory network. *Annual review of phytopathology*, *38*(1), 263-292.
- Schouten, A., Tenberge, K. B., Vermeer, J., Stewart, J., Wagemakers, L., Williamson, B., & Van Kan, J. A. (2002). Functional analysis of an extracellular catalase of Botrytis cinerea. *Molecular plant pathology*, *3*(4), 227-238.
- Siedow, J. N., Rhoads, D. M., Ward, G. C., & Levings III, C. S. (1995). The relationship between the mitochondrial gene T-urf 13 and fungal pathotoxin sensitivity in maize. *Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease*, *1271*(1), 235-240.
- Sijmons, P. C., Atkinson, H. J., & Wyss, U. (1994). Parasitic strategies of root nematodes and associated host cell responses. *Annual review of phytopathology*, *32*(1), 235-259.
- Singh, U. S., Singh, P. R., & Kohmoto, K. (1994). "Pathogenesis and Host Specificity in Plant Diseases: Histopathological, Biochemical, Genetic and Molecular Bases," 1–3. Pergamon/Elsevier, Tarrytown, NY.
- Stall, R. E., & Hall, C. B. (1984). Chlorosis and ethylene produc- tion in pepper leaves infected by Xanthomonas campestris pv. vesicatoria. *Phytopathology*, *74*, 373–375.
- Sugui, J. A. (1997). Association of *Pestalotia malicola* with the plant cuticle: Visualization of the pathogen and detection of cutinase and non-specific esterase. Physiol. Mol. *Plant Pathol*, *52*, 213–221.
- Taylor, J. L. (2003). Transporters involved in commuication, attack or defense in plant-microbe interactions. In "Plant-Microbe Interactions" (G. Stacey and N. T. Keen, eds.), 6, 97–146.
- Tenllado, F., Barajas, D., Vargas, M., Atencio, F. A., González-Jara, P., & Díaz-Ruíz, J. R. (2003). Transient expression of homologous hairpin RNA causes interference with plant virus infection and is overcome by a virus encoded suppressor of gene silencing. *Molecular plant-microbe interactions*, *16*(2), 149-158.
- Valette-Collet, O., Cimerman, A., Reignault, P., Levis, C., & Boccara, M. (2003). Disruption of Botrytis cinerea pectin methylesterase gene Bcpme1 reduces virulence on several host plants. *Molecular Plant-Microbe Interactions*, *16*(4), 360-367.
- van Wezel, R., Liu, H., Tien, P., Stanley, J., & Hong, Y. (2001). Gene C2 of the monopartite geminivirus tomato yellow leaf curl virus-China encodes a pathogenicity determinant that is localized in the nucleus. *Molecular plant-microbe interactions*, *14*(9), 1125-1128.
- Vereecke, D., Temmerman, W., & Jaziri, M. (2003). Toward an understanding of the *Rhodococcus*

fascians -plant interaction. In "Plant-Microbe Interactions" (G. Stacey and N. T. Keen, eds.), 6, 53–80.

- Wolpert, T. J., Dunkle, L. D., & Ciuffetti, L. M. (2002). Host-selective toxins and avirulence determinants: what's in a name?. *Annual review of phytopathology*, *40*(1), 251-285.
- Wu, Y., & Hohn, B. (2003). Cellular transfer and chromosomal integration of T-DNA during Agrobacterium tumefaciens-mediated plant transformation. *Plant-microbe interactions: Vol. 6*, 1-18.
- Yamada, T. (1993). The role of auxin in plant-disease development. *Annual review of phytopathology*, *31*(1), 253-273.
- Zhang, J. X., Bruton, B. D., Miller, M. E., & Isakeit, T. (1999). Relationship of developmental stage of cantaloupe fruit to black rot susceptibility and enzyme production by Didymella bryoniae. *Plant disease*, *83*(11), 1025-1032.

Cite This Article: Senthilmurugan Palanisamy, Atul Singh, Potunuri Hema Prashanthi Lakshmi, Bhuvana C.R, Glory, K.B (2024). Host Recognition for Survival of Plant Pathogens. *East African Scholars J Agri Life Sci*, *7*(10), 116-130.