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Fighting for Their Lives: Plants and Pathogens

Plants are in constant contact with microbes, including viruses, bacteria, fungi, oomycetes, and nematodes, many of which have the potential to cause disease: that is, to become pathogenic. Globally, crop losses to pathogens are typically 10 to 30% of the potential harvest, but severe disease outbreaks can be even more damaging. Nevertheless, plant disease is the exception rather than the rule; the majority of plants are resistant to infection by the majority of microbes. What makes an organism into a successful pathogen? How do plants defend themselves against potential pathogens? What is the role of the environment in these interactions? How has the concept of plant disease been shaped by plant breeding and agricultural practices? In this article, we begin by considering what makes an interaction between organisms into a disease, as described by the disease triangle. We then look at the genetic interactions between plants and pathogens and the plant’s immune and defense responses. Both pathogenicity and defensive traits have been under intensive selective pressure for millions of years, as the organisms fight for their lives. We conclude with an examination of how plant diseases are managed or prevented, and throughout the article, we present case studies of pathogens or diseases to illustrate key concepts.

BRIEF HISTORY OF PLANT PATHOLOGY

The impact of plant diseases, their potential causes, and the treatment strategies to manage them have been documented for thousands of years. As early as 1500 BC, the ancient Indian text the Rigveda, and later the naturalist Varāhamihira (c. 500 AD), recorded that plant disease could be caused by cold climate, wind, and sun and pointed out similarities between human and plant diseases. The Bible makes references to blights and mildews, and the Greek philosopher Theophrastus (c. 300 BC, sometimes called the father of botany) wrote about cereal, tree, and legume diseases. He noted that disease susceptibility differed between plant species and that disease outbreaks in lowland areas were generally more severe than on hillsides. Despite these astute observations, the microscopic nature of most pathogens made it very difficult for ancient scientists to truly understand the nature of plant diseases. Nearly two thousand years after Theophrastus, advances in scientific approaches and microscopy, along with a devastating outbreak of potato late blight in the 1840s, led to the birth of plant pathology as a coherent and distinct discipline.

Case Study: Phytophthora infestans, the Plant Destroyer

Potatoes (Solanum tuberosum) and tomatoes (Solanum lycopersicum) are native to the Peruvian region of the Andes. They were imported into Europe by Spanish explorers in the late 16th century. During the early years of their cultivation in Europe, they were not afflicted by the late blight disease, which is caused by oomycete pathogen *P. infestans*. (*Phytophthora* is an oomycete, more closely related to brown algae than fungi, but its growth pattern resembles that of a fungus and for many years it was mistakenly classified as a fungus.) By the 1840s, *P. infestans* had been imported into Europe, probably in infected plant tissue. *P. infestans* is an aggressive pathogen (its name literally means plant destroyer), and European potatoes had little resistance to it, so the potato crop was almost entirely destroyed in many areas. Compounding the problems of crop loss were the social structures of the 19th century, particularly in Ireland and Scotland, which included large rural tenant farmer populations who relied on potatoes for sustenance and financial livelihood. The disease led to widespread famine and migration from these regions.

Miles Joseph Berkeley was an outstanding 19th century scientist with a keen interest in fungi and plant diseases. He observed, collected, and studied the infected potato plants and concluded that the oomycete *P. infestans* was the causal agent of the disease, but his conclusions were not readily accepted. In the 1860s, another eminent plant pathologist, Anton de Bary, confirmed Berkeley’s theory by transferring spores from an infected plant onto an uninfected plant, which became infected. (The control plant did not, ruling out the hypothesis that late blight was caused by dampness.)

Around this time, Louis Pasteur’s and Robert Koch’s studies showed unambiguously that bacteria can be causal agents of human and animal diseases, and bacteria and viruses also were found to be agents of plant disease. Koch’s Postulates, published in 1890, still are followed to identify a disease-causing agent. (Being macroscopic, nematodes were recognized as disease-causing agents earlier, but scientific investigations of nematodes didn’t progress until the 1850s, and phytoplasmas, tiny wall-less bacteria, were not discovered until the 1960s.)

By the beginning of the 20th century, scientists knew a lot about what causes plant disease. The early part of the 20th century focused on defining and characterizing diseases and disease-causing agents and developing tools with which to combat them. Plant breeders employed genetics, and agricultural chemists used chemicals to reduce the damaging effects of pathogens on food production. From the latter part of the 20th century to the present, molecular genetics techniques revealed the intricacies of pathogenicity, susceptibility, and immunity, opening the door to targeted disease suppression approaches.

WHAT MAKES AN INTERACTION BECOME A DISEASE?
THE DISEASE TRIANGLE

Three fundamental elements are required for plants to become diseased: a susceptible plant, a virulent pathogen, and a favorable climate.
environment. Human contributions, including the introduction of alien species to an environment, and the practice of planting genetically uniform crops, can contribute to the occurrence of plant diseases.

One side of the triangle is the pathogen: its abundance, and its genetically encoded virulence, which includes the ability to penetrate into the host and evade or suppress the host’s immune responses. Many of these virulence traits are conferred by the pathogen’s effector proteins, which are described further below.

The plant side of the triangle involves the plant’s overall health and vigor but also involves genetically encoded immunity, which as described below includes the ability to sense and respond to conserved microbial features as well as specific pathogen effectors.

The third side of the triangle is the environment. Environmental contributions to plant disease are important but complex and not fully understood. Temperature, moisture levels, and wind affect the viability, growth, and dispersal rate of pathogens as well as the susceptibility of their plant hosts. As examples, high winds can disperse fungal spores and cause physical damage to plants, facilitating pathogen entry. Outbreaks of diseases caused by Xanthomonas bacteria are more prevalent following typhoons (e.g., rice bacterial blight caused by Xanthomonas oryzae) or hurricanes (e.g., citrus canker caused by Xanthomonas axonopodis pv citri). Many fungal spores require free water to germinate and invade plant tissues, which is why wet weather is often correlated with disease outbreaks. Consecutive days of high humidity and moderate temperature, called blight weather, are highly conducive to outbreaks of late blight, and growers monitor these environmental indicators to decide when to treat their crops with fungicides. The local environment is also affected by agricultural practices, which are critical in managing disease, as described further below. Finally, the distribution of pathogens is being altered by climate change, meaning that disease outbreaks are occurring in previously unaffected regions.

Modern agricultural practices can also impact the incidence of disease. The application of fertilizers and other chemicals can make plants more susceptible to certain pathogens. Irrigation systems can leave standing water in soil or on leaves that can facilitate the spread of pathogens. The planting of large areas of a single, genetically uniform species, also known as monoculture, makes planting and harvesting easier and reduces production costs, but also means that when a pathogen finds a susceptible host, it can swiftly give rise to an epidemic.

**Case Study: The Southern Maize Leaf Blight Epidemic of 1970**

A classic example of all sides of the disease triangle coming together and resulting in an epidemic is the interaction between maize and the fungus Cochliobolus heterostrophus, causal agent of southern maize leaf blight. Traditionally, it had been a minor problem, but in the 1970 growing season in the United States, it caused devastating damage and crop losses and provided one of the most enduring lessons about plant breeding and the dangers of genetic uniformity.

Hybrid maize gives higher yields and is produced by crossing two different parental lines. Maize produces female flowers that give rise to cobs and seeds along the stem, and male, pollen-bearing flowers in a tassel at the top. Conventionally, hybrid maize was produced by detasseling the female parent to ensure that it would outcross with the pollen provided by a different plant. Breeders identified genetic male-sterility loci, which eliminate the need to detassel the female plants. When this trait is conferred by a mitochondrial gene showing cytoplasmic inheritance patterns, the trait is called cytoplasmic male sterility.

Beginning in the 1950s, Texas male-sterile cytoplasm (T-cms) maize (Zea mays) was grown for hybrid seed production. By 1970, almost 85% of hybrid seed produced in the United States was from T-cms maize. Simultaneously, a new, highly aggressive race of C. heterostrophus evolved, race T, which vigorously attacks T-cms maize but only causes mild symptoms on normal cytoplasm maize. Race T produces T-toxin, which binds to the inner mitochondrial membrane protein URF-13 that is only produced in T-cms maize, and generates pores that result in the termination of ATP production and cell death. This unique combination of host susceptibility and pathogen virulence was exacerbated by warm, wet weather throughout the maize growing season in 1970 and caused the worst agricultural disease epidemic in United States history. Yield reductions from southern maize leaf blight were observed in more than 30 maize-growing states, ranging from 50 to 100% and with an estimated loss of a billion (1970) dollars. Outcomes from this epidemic include increased awareness of the risks associated with planting large areas with genetically homogenous plants and a movement away from the use of vulnerable cytoplasmic male sterile maize.

**STRATEGIES OF PATHOGENICITY**

Most microorganisms are not capable of causing disease. Pathogenic organisms are widely distributed among nonpathogenic relatives, and phylogenetic studies suggest that pathogenicity traits have been gained and lost repeatedly. When a potential pathogen and plant meet, a series of biochemical interactions ensues. The outcome of these interactions determines whether the pathogen ultimately establishes a successful infection, or, more commonly, whether the host successfully fights it off through its myriad defense and immune responses. Here, we will introduce some of the habits of successful pathogens, with the caveat that there are few “absolutes” and “always” in biology.

**Finding the Host**

Many pathogens are dispersed passively, via wind or water droplets, and encounter a suitable host simply by chance. Others are carried between plants by vectors, which can be insects and other animals, or agricultural equipment. Many soil-borne pathogens can sense the presence of a nearby plant through sloughed off cells or chemicals secreted by the plant, such as amino or organic acids, or phenolic compounds released by wounded plant tissues. The pathogen can use the process of chemotaxis to move toward the source of these exudates. Although it may seem unusual that plants broadcast biochemical information about themselves into an environment full of potential
pathogens, some of these exuded compounds facilitate interactions with microbial symbionts, including nitrogen-fixing bacteria and mycorrhizal fungi. As an example, the zoospores of pathogenic Phytophthora sojae are attracted to daidzein and genistein, isoflavones released from soybean (Glycine max) roots that also attract symbiotic nitrogen-fixing rhizobia.

Host Attachment and Penetration

Once a pathogen has found a suitable host, it needs to gain entry. Plants are protected by physical barriers, including the cell wall and, in aerial tissues, the extracellular waxy cuticle. Many pathogens secrete extracellular polysaccharides that help them adhere to the host plant, and in some cases biofilms are formed that contribute to adhesion and/or protect the pathogens from plant defenses. Bacteria also can use hair-like structures called pili to adhere to the plant surface. Interfering with the process of adhesion is sometimes sufficient to prevent infection.

Pathogens can gain entry into the plant through wounds or natural openings, including stomatal pores or pores in the leaf margin called hydathodes. Viruses usually are carried by insect vectors and enter the plant through the wounds caused by insect feeding, often being deposited directly into the vascular tissues that facilitate their spread within the plant. Nematodes burrow into plant tissues and, if they evade the plant’s defenses, use a structure called a feeding stylet to pierce cells through which they extract nutrients and introduce effector proteins. Many pathogenic fungi and oomycetes adhere to the plant surface using adhesive proteins and penetrate host tissue by forming appressoria. Appressoria puncture through plant cell walls using high physical pressure, cell wall–degrading enzymes, or both. In some cases, the appressoria penetrate through stomata. Within the plant, hyphae or specialized organs called haustoria can form that are involved in the uptake of nutrients and that can export effectors that enhance the pathogen’s virulence or suppress the host’s defenses.

Growth within the Host: Biotrophs, Necrotrophs, and Hemibiotrophs

Once the host’s barriers have been breached, there can be several outcomes, based in part on the life history of the invading pathogen. Although plant pathogens come from diverse kingdoms, they can be placed into three groups by their mode of pathogenicity, or life history. Biotrophs are pathogens of living tissues and include nematodes, viruses, most bacteria, and some fungi and oomycetes. Biotrophs tend to have narrow host ranges and most can attack healthy host tissue at any state of development. Once established, the biotroph can reside within the host and exploit it as a source of nutrients without killing it. The genetic interactions between biotrophs and their host are complex, in that it is in the pathogen’s interest not to kill the host or trigger its catastrophic self-destruction. Although we don’t use the term biotroph for human pathogens, if we did, then HIV, Plasmodium spp (causal agents of malaria), and the fungal agents that cause ringworm would be biotrophs, residing in their living hosts for many years.

By contrast, necrotrophs have more of a destructive approach; they kill their host bit by bit and feed off the nutrients from the dead cells. Necrotrophs produce toxins and cell wall–degrading enzymes and cause extensive tissue maceration. Various bacterial, fungal, and oomycete pathogens employ this style of attack, such as the soft rot bacterial pathogen Pectobacterium carotovora and the gray mold fungus Botrytis cinerea, which is the gray mold often seen on strawberries (Fragaria spp), raspberries (Rubus spp), grapes (Vitis vinifera), and many other fruits. These pathogens overwhelm host defenses, frequently targeting weaker cells, such as those in young succulent or old senescent tissues. Necrotrophs kill, consume, and move to the next susceptible tissue. Human pathogens that use this approach include several types of necrotizing bacteria (aka flesh-eating bacteria) such as Staphylococcus aureus and Clostridium perfringens, which kill the host tissues through toxin production.

Some pathogens have a hybrid approach, initially being biotrophic but later necrotrophic; these are called hemibiotrophs. Fungal, oomycete, and bacterial hemibiotrophic pathogens exist, many of which establish an infection and proliferate as biotrophs, but then switch to an aggressively necrotrophic lifestyle. P. infestans is an example of a hemibiotroph that initially suppresses cell death in the host, but then once the pathogen has grown throughout the host, it begins producing proteins that rapidly kill cells and induce tissue necrosis.

Our understanding of what makes an organism an effective pathogen has been greatly enhanced by comparative genomic studies between pathogens and between pathogens and their closely related nonpathogenic relatives. For example, the genome sequence of barley powdery mildew (Blumeria graminis f. sp hordei), an obligate biotroph that can only grow and reproduce on a living plant, reveals that, compared with its relatives, it has lost many genes that are involved in nutrient uptake, production of cell wall–degrading enzymes, and production of defensive compounds; these latter two categories may enable it to avoid triggering plant defense responses. Other studies have indicated that many genes associated with pathogenicity are found in clusters within the genome and are often spread by horizontal gene transfer, even into organisms from other kingdoms (i.e., from fungi to oomycetes and from bacteria to fungi).

Case Study: Fungal Mimicry

A few species of plant pathogenic fungi have the ability to change their host’s morphology in extreme ways to facilitate the reproductive success of the pathogen. One example of this is the formation of pseudoflowers. Pseudoflowers are leaves that form with a morphology that resembles a flower (not necessarily the host’s own flower though). For example, Puccinia monoica induces its host (Arabis spp) to form a set of whorled, yellow leaves that produce floral aromas and nectar, thereby attracting pollinators to the pseudoflowers. The fungus releases gametes onto the surface of the pseudoflowers, which are picked up and distributed by pollinators. The ability to induce pseudoflowers has evolved several times in different fungal species. Another
type of mimicry involves the anther smut fungus *Microbotryum violaceum*. This fungus causes the host’s flowers to become sterile, but to produce anthers that display fungal spores in place of pollen. Through this mimicry, the fungus harnesses the activity of the plant’s pollinators to disperse its spores. At this point, the molecular bases for these odd pathogen-induced developmental defects are still being investigated.

**PLANT IMMUNE RESPONSES**

The accumulated insights of countless researchers have contributed to a relatively coherent picture of the molecular interactions through which plants recognize and respond to pathogens. Although it does not explain every interaction, nor apply equally to every pathogen, the zigzag model provides a conceptual framework with which to describe plant immune responses. The model has three parts. First is the initial recognition and response to the pathogen, called pattern-triggered immunity (PTI), in which the plant recognizes the pathogen through its conserved molecular structures and mounts a defense response. Some pathogens can overcome PTI by producing small molecules or proteins called effectors, which can dampen the plant immune response; this is called effector-triggered susceptibility. Finally, a second wave of plant immune responses is sometimes triggered in response to pathogen effectors, called effector-triggered immunity (ETI). Of course, this cycle can repeat; the pathogens can overcome ETI to gain the upper hand, and the plant can evolve a new mechanism by which to overcome the pathogen, over and over.

**PTI: The Enemy Is at the Gate**

Surveillance and recognition of the enemy is essential to defense. Plant receptors known as pattern recognition receptors (PRRs) bind to conserved microbial molecules, such as chitin found in fungal cell walls or the flagellin protein found in bacterial flagella. These conserved molecules, or signatures, are often referred to as pathogen-associated molecular patterns (PAMPs) or as microbial-associated molecular patterns. PRRs recognize pathogens through an extracellular domain, while the pathogen is on the outside of the plant cell. FLAGELLIN SENSING2, which recognizes the bacterial flagellin protein, and EF-TU RECEPTOR, which recognizes a bacterial elongation factor, are well-characterized PRRs. Both of these receptors include an extracellular leucine-rich repeat domain that recognizes the PAMP and a cytosolic protein kinase domain required for signal transduction. Structurally, they resemble the Toll-like receptors that act in animal cell immune responses. Chitin is recognized by a different type of PRR that has an extracellular LysM (chitin binding) domain and cytosolic kinase domain. Only a few PRR-PAMP pairs have been characterized, but identification of new combinations is an active area of research.

PAMP recognition initiates a signal transduction process sometimes including a mitogen-activated protein (MAP) kinase cascade that leads to the induction of defense responses. Induced defense responses include the production of pathogenesis-related proteins and reactive oxygen species, which can kill the pathogen, and the production of defensive phytoalexins. Plant cells are rich in secondary metabolites, many of which have documented roles in defense. Some of these are constitutively expressed and referred to as phytoanticipins, and some are induced by the presence of a pathogen and known as phytoalexins; a single compound can be a phytoanticipin in one plant and a phytoalexin in another depending on whether it is constitutive or induced. Defensive chemicals are diverse and often found in only a few species and include steroids, triterpenes, alkaloids, and flavonoids. Many pathogens that can successfully survive plant defense responses have evolved mechanisms to detoxify the induced phytoalexins or to interfere with their induction.

PRRs seem to recognize biotrophic and necrotrophic pathogens, but necrotrophs may also be revealed by the toxins they produce or the cellular damage they cause. Downstream of recognition, plant responses to biotrophic pathogens are also better understood. Biotrophic pathogens elicit the production of the defense hormone salicylic acid and a burst in production of reactive oxygen species, which serves as both a signal to induce further defense responses and as an antimicrobial agent. Salicylic acid and reactive oxygen species contribute to the transcriptional induction of defense genes. Cell surface responses at the site of pathogen attack are also common, such as the production of callose, a defensive polysaccharide. Often, but not always, PTI is sufficient to eliminate or arrest the invading pathogen.

**Effector-Triggered Susceptibility**

Successful pathogens can produce effectors to enhance their own pathogenicity and suppress the host’s immune response. Effectors are usually produced in a species-specific or racesspecific manner; they are rapidly evolving features. Most pathogens produce many different effectors, and while collectively these are important for pathogenicity, the loss of any one effector usually has little impact on virulence. Effectors are active outside of the pathogen and share the property of being transferred from the pathogen cell into the host tissue. Other than this property, effectors are extremely diverse in structure and function.

Some effectors act in the apoplast, but most are introduced into the plant cytoplasm or subcellular organelles. Pathogens have evolved various ways to transport their effector proteins from their own cell into the host cell. Many bacterial pathogens of plants and animals use a specialized secretion system called a Type III secretion system to introduce their effectors into the plant cell; effectors transported through this are referred to as Type III effectors and include some of the best characterized effectors. Plant pathogens also use Type II and Type IV secretion systems, albeit less commonly. Nematode effectors are encoded by parasitism genes and are introduced into the host cell through the feeding stylet. Fungi and oomycetes may introduce a haustorium into the host cell, which is within the cell wall but outside the host cell plasma membrane. In these cases, effectors are secreted from the haustorium into the extrahaustorial matrix and then can be taken up through the plant plasma membrane. Within the plant cell, effectors can be targeted to different cellular components, including the membrane, cytoplasm, mitochondria, chloroplast, or nucleus. Identifying and understanding the functions of the many diverse effectors is a highly active area of research.
Most pathogens produce many different effectors with different functions, which can contribute to enhancing the virulence of the pathogen or suppressing the plant’s defense responses. For example, the Cladosporium fulvum Avr2 effector is an inhibitor of Cys proteases, which acts in the apoplast and protects the fungus from attack by plant defensive proteases, and Ecp6 also acts in the apoplast where it binds to chitin in the fungal wall, effectively competing with the PRR and so helping the fungal pathogen to evade detection. Coronatine is produced by Pseudomonas syringae pv tomato and acts as a mimic of the plant hormone jasmonate-Ile, which promotes the opening of the plant’s stomata and facilitates bacterial entry. P. syringae also produces effectors that target the PRR signal transduction pathway, including effectors that promote proteolytic degradation of PRRs, inhibit their kinase activity, and irreversibly dephosphorylate the MAP kinases downstream of them. Other effectors block the plant’s defensive cell death response, alter hormonal signals to dampen the defense response, or even act as transcription factors to induce the expression of genes involved in cell proliferation, all of which enhance the viability of the pathogen. The transcription activator-like effectors, also known as TALEs, have a unique way of binding DNA that has great promise as a novel tool for biotechnology. Some Agrobacterium tumefaciens species can even mobilize a piece of DNA into the plant’s genome to alter the host’s metabolism and enhance the pathogen’s viability (Teaching Tools in Plant Biology 23 is an in-depth look at Agrobacterium tumefaciens).

Case Study: Pseudomonas syringae, a Model Pathogen

P. syringae is a bacterial pathogen that is responsible for bacterial speck diseases on several types of plants, including Arabidopsis thaliana. P. syringae is classified into pathovars (pathogenic varieties) that correspond to their hosts, and the various pathovars have had major roles in the uncovering of how bacteria pathogenize plants; this species has been one of the principle models for studies of plant pathogens. In fact, in the recently published list of the top 10 plant pathogenic bacteria in molecular plant pathology, the P. syringae pathovars came in at number one. The first bacterial effector gene was cloned from P. syringae, and the first effector, resistance (R) protein pair identified from it. Early studies of Type III secretion systems were conducted in P. syringae, and several components of the downstream defense signaling pathway were identified in Arabidopsis plants infected with P. syringae. Genomic sequence data for several pathovars are contributing to our understanding of the bacterial genes that underlie their host specificity.

Immune Receptors and Resistance Proteins

A pathogen that evolves a novel effector becomes a more successful pathogen and so provides strong selective pressure on the host plant population to evolve resistance mechanisms. Plants overcome effector-armed pathogens through R proteins, which are essentially intracellular immune receptors and are sometimes referred to as effector recognition receptors. They may recognize pathogen effectors directly, or they may act as guards that recognize abnormalities in host cell proteins. When R proteins sense pathogen effectors or their effects, they initiate a second defense mechanism, termed ETI.

Most known R proteins are nucleotide binding–leucine-rich repeat (NB-LRR) proteins. The Arabidopsis genome encodes 149 NB-LRR proteins, whereas rice (Oryza sativa) and poplar (Populus spp) have more than 400. At their N termini, these proteins usually have either a toll/interleukin-1 receptor domain or a coiled-coil domain. Most are located in the cytoplasm, although some have been found at the plasma membrane, in the apoplast, or the nucleus. Some R proteins are noncanonical, such as the Arabidopsis R protein RPW8.2 that confers resistance to powdery mildew, which has an N-terminal transmembrane domain and one to two coiled-coil domains, and is localized exclusively at the extrahaustorial membrane.

Although some resistance proteins recognize effectors directly, others recognize the effects of the effectors on host cell machinery. This latter strategy might be advantageous in that by surveying its own proteins, the plant doesn’t need a separate R protein for every effector a pathogen might produce; a single R protein can guard against many different effectors that target the same plant protein. In support of this model, the effector targets of evolutionarily diverse pathogens seem to converge on cellular hubs, that is, proteins that interact with many other proteins. Thus, critical cellular targets are both acted on by effectors and guarded by R proteins.

ETI

When resistance proteins recognize effectors or modified-self, they initiate an enhanced immune response known as ETI. This immune response is similar to but stronger, faster, and more prolonged than the PTI response, and it may be genetically more robust and resistant to interference by the pathogens. Although genetic studies have revealed many components of the signal transduction pathway downstream of R proteins, our understanding of these events remains incomplete. Current efforts include biochemical studies that are revealing the roles of protein complex formation in regulating R protein function and the role of subcellular localization and protein stability in the signal transduction pathways. The WRKY transcription factors are important modulators of the defense response, which can act as positive or negative regulators.

R protein activation can induce the production of the immune signals salicylic acid and reactive oxygen species, which contribute to the induction of defense response genes and also to the initiation of the hypersensitive response. The hypersensitive response is a form of programmed cell death mediated by a burst of reactive oxygen. It contributes to the plant defense against biotrophic pathogens by killing the infected host cell and associated pathogens; mutants deficient in the hypersensitive response can be more susceptible to diseases caused by biotrophs, and some biotrophic pathogen effectors suppress the hypersensitive response. The oxidative burst also increases cell wall cross-linking to seal off the infected tissues and help prevent pathogen spread. (Teaching Tools in Plant Biology 20 describes cell death and the hypersensitive response more completely).
Both PTI and ETI can induce the condition of systemic acquired resistance (SAR). SAR was described in the 1960s, with the observation that in a plant infected with a virus, leaves that were not themselves exposed to the virus nevertheless acquired resistance to subsequent viral challenge. SAR occurs in response to other pathogens as well. The debate as to the mobile signal that moves from the site of infection to more distant tissues is ongoing; several candidate signals have been proposed, and it is very likely that more than one signal is involved. Chromatin changes may also contribute to the resistant state.

Case Study: The Reemergence of Wheat Stem Rust

Many resistance proteins were first identified genetically, as R genes that confer resistance to a specific pathogen. Long before molecular biology revealed the nature of R genes, plant breeders had identified them phenotypically and were crossing them into susceptible plants to confer resistance. However, pathogens can evolve novel effectors that overcome R gene–mediated immunity; examples of this have occurred recently in the apple scab fungus and the wheat stem rust fungus.

Wheat (Triticum aestivum) is the largest food crop in the world. Biotrophic rust fungi, including wheat stem rust Puccinia graminis f. sp tritici, are among the most economically destructive plant pathogens. Stem rust spores are windblown and spread rapidly, and outbreaks frequently lead to nearly complete crop loss. Wheat stem rust most likely has been a problem since the earliest days of wheat cultivation; Romans prayed to the God of Rust to spare their grains, and it has been suggested that successive poor wheat harvests caused by rust may have contributed to the fall of the Roman Empire.

Wheat stem rust brought Norman Borlaug to Mexico in the 1960s, where among other successes he bred rust-resistant wheat carrying the R gene Sr31 (for Stem rust31), which proved so successful that wheat stem rust was not a threat for many years. Most cultivated wheat carries Sr31 along with other Sr genes. In the late 1990s, it became clear that a virulent rust strain was emerging in East Africa, which was named Ug99 after its formal identification in Uganda in 1999. Ug99 is able to overcome Sr31 and also several other Sr resistance genes. Most wheat varieties are susceptible to Ug99, so the only thing protecting the world’s wheat supply has been the rate of spread of the fungus. Since it was first discovered in 1999, Ug99 has spread throughout East and South Africa at an alarming rate, and in 2007 was identified in Iran, with the threat of its spread to the major wheat growing regions of Pakistan, India, and Australia. Since Ug99 was identified, an international team of scientists has worked at top speed to identify new R genes to protect wheat. Since 2010, wheat strains with heightened resistance have been planted in threatened regions, hopefully to slow and stop the spread of this devastating disease. It is important to note that this disease is particularly threatening to developing countries because, although fungicides are available that can combat the fungus, they are quite expensive. Ug99 will eventually reach developed countries, but the availability of fungicides will likely prevent famine in these regions.

PLANT RESPONSES TO NECROTROPHIC PATHOGENS

Necrotrophic pathogens are the largest class of plant pathogenic fungi and have significant economic consequences. Among the diseases caused by fungal necrotruchs are black spot of brassicas (Alternaria brassicicola), gray mold or botrytis blight (B. cinerea), fusarium blight (Fusarium graminearum), and southern maize leaf blight (C. heterostrophus). Seedling damping off is commonly caused by necrotrophic oomycete species of Pythium and Phytophthora. Bacterial necrotruchs include Pectobacterium carotovorum (formerly Erwinia), causal agent of soft rot. Plant responses to necrotruchs are different from those to biotrophs. For example, R protein–mediated immunity is not usually involved in defense against necrotruchs; if the plant did switch on this pathway, the downstream hypersensitive response could actually enhance the necrotruch’s growth. Therefore, it is not totally unexpected that some necrotruchs produce effectors (or toxins) that activate R protein–mediated defense and lead to cell death, essentially using the host’s defenses against it. With exceptions, plants also use different hormones in their responses to biotrophs and necrotruchs: salicylic acid for biotrophs and ethylene and jasmonate for necrotruchs.

SIGNAL INTEGRATION

The signaling events downstream of pathogen attack or resistance protein activation have been characterized in large part through studies of Arabidopsis. Genetic studies identified mutants with enhanced or suppressed defense responses, leading to the identification of major components of the signaling pathways. A key concept to emerge from these studies is that plant defense responses are informed by many different integrated signals. For example, biotroph–induced R protein–mediated signal pathways intersect with those of necrotrophic pathogens and with abiotic stress pathways, including water stress and elevated temperatures. It seems that instead of initiating defense responses independently, plants prioritize their responses contextually. For example, the hypersensitive response is suppressed when the plants are infected by both a biotroph and a necrotroph. P. syringae’s production of coronatine, which mimics jasmonate, exploits the integrated signaling network to suppress the salicylate-mediated defense responses, enhancing its pathogenicity. (Teaching Tools in Plant Biology 13 and 14 delve more deeply into defense hormone signaling pathways.)

PLANT RESPONSES TO VIRUSES

The responses of plants to viruses are surprisingly similar to their responses to other pathogens. One of the first R genes to be cloned, N, confers resistance to tobacco mosaic virus, and the concept of SAR emerged from studies of viral pathogens. However, because viruses replicate as free nucleic acids in the host cytoplasm, they are particularly vulnerable to small RNA–mediated defenses. In fact, the study of viral pathogens led to key insights into small RNA biology (see Teaching Tools in Plant Biology 5: The Small RNA World). As described below, viral resistance can be engineered through small RNA–mediated gene silencing.
Plant pathology includes a very applied aspect, and there are striking parallels between the management of plant and animal diseases. However, in contrast with human medicine, the goal of plant pathology is to maximize the health of the population, with little importance being placed on any one individual. Recognizing the symptoms of plant disease so that affected plants can be treated or removed is one of the most effective disease prevention strategies and is largely performed by the growers themselves. Agricultural extension services train growers to diagnose diseases and report local outbreaks. Cultural practices help to minimize disease outbreaks, but once an outbreak occurs, containment and destruction of the pathogen becomes paramount. In agricultural practices as in the hospital, pathogen spread must be avoided by rigorously cleaning equipment and incinerating contaminated tissues. The tools used in the development of antimicrobial agents are the same whether employed by a pharmaceutical company for human use or by an agricultural chemical company for use on plants. Other resources available to farmers are biological control agents and disease resistance breeding. As in medicine, economic costs are important considerations, as well as considerations of environmental and food safety.

An Ounce of Prevention Is Worth a Pound of Cure

The best plant disease practice is to avoid it entirely, which is of course impossible. In the age of air travel, pathogen dispersal occurs constantly, despite agricultural inspections at border crossings, inspections of food imports, and strict regulations controlling shipments of live plant materials. Quarantines can slow but not stop pathogen dispersals, as made clear by the introduction of the bacterium that causes Asiatic citrus canker (Xanthomonas axonopodis pv citr) into Florida in the early part of the 20th century, the introduction of a wheat fungus that causes Karnal bunt (Tilletia indica) into the United States in 1996, the spread of wheat stem rust variant Ug99 through Africa and into the Middle East, and the repeated introduction of Phytophthora ramorum (causal agent of sudden oak death) into North America and Europe during the past few years. Unfortunately, the increased emphasis on antiterrorism activities at United States border crossings over the past decade took resources from agricultural inspection services and is correlated with an increase in plant disease outbreaks.

One of the most effective ways to control disease is to remove dead plant matter from the growing area, which is often serves as the reservoir for the pathogen. Farm machinery and irrigation systems can also serve as pathogen reservoirs and need to be cleaned regularly. Rotating different crops through fields can minimize soil pathogen levels, so that pathogen populations decline between host plantings; the effectiveness of this strategy depends on the viability of the pathogen outside of a host. Prior to planting, soils can be chemically treated or heated (solarized) to further reduce pathogen levels. Many pathogens can live on and in seeds. Seed companies can be good sources of pathogen-free seeds, or seeds can be gently heat treated or bleached to reduce their pathogen loads.

The timing of planting and treatment of the growing area can help avoid pathogens or their associated vectors. For example, planting in well-drained soil can help avoid diseases caused by the oomycete Pythium that causes seedling damping off. In the early 1950s, T.C. Vanterpool demonstrated that amending soils by incorporating ammonium phosphate fertilizers or adding more organic matter could prevent Pythium diseases, possibly both by maintaining optimal seedling metabolism at critical developmental phases and by inducing the lysis and death of Pythium oospores. Control of insect vectors at peak germination times is particularly important in reducing the spread of viruses and fastidious prokaryotes (e.g., phytoplasmas). Physical barriers such as placing a plastic sleeve around bananas can decrease plant exposure to fungal pathogens and insects. Maintaining healthy plants is also important in avoiding diseases, as is avoiding standing water in soils and on leaf surfaces.

Case Study: Plant-Associated Human Pathogens

Since the early 1990s, outbreaks of food-borne human pathogens are regularly being traced back to contaminated plant foods. In 2011, two of the deadliest food-borne infectious illness outbreaks were caused by contaminated plants. In Germany, 50 people died from Escherichia coli–contaminated bean sprouts, whereas in the United States, 29 people died from Listeria–contaminated cantaloupes. Why are plants a source of human pathogens? What have we learned from these epidemics, and how can we prevent future ones? As with any epidemic, many factors are involved, including the evolution of a hypervirulent pathogen, changes in our dietary practices, and changes in how food is processed and transported.

The 2011 deaths in Germany were caused by a new, highly virulent, antibiotic-resistant strain of E. coli, O104:H4, which is a Shiga toxin-producing E. coli (STEC). Shiga toxin is an intestinal cytotoxin that originated in the causal agent of bacterial dysentery, Shigella dysenteriae; the ability to make the toxin moved into E. coli by horizontal gene transfer. STECs have been implicated in numerous outbreaks of food poisoning from poorly cooked meat as well as uncooked fruits and vegetables. Several recent epidemics have been traced to plants that were contaminated in the field, often by contaminated irrigation water or manure. Because cattle are unaffected by the Shiga toxin, they can carry STEC and disseminate it in their feces. Uncooked produce, such as lettuce, or the sprouted seeds responsible for the German outbreak, are excellent vectors for these pathogens, which are readily killed by cooking. The practice of packaging and shipping lettuces and other raw produce is also contributing to the problem; the more time that passes between harvesting and consumption, the more time for the pathogenic bacteria to multiply to hazardous levels. Because the pathogenic bacteria are frequently present inside the plant tissues, they are impossible to remove by washing; contamination must be avoided, or food must be cooked to kill the pathogen.
By contrast, the outbreak from cantaloupes in 2011 was almost certainly due to contamination after harvesting, and the outbreak could have been avoided with more careful hygiene practices. The increase in plant-based food poisoning over the past 20 years has led to more stringent regulations governing how fresh produce is grown, harvested, handled, stored, and transported, which has limited outbreaks, but a better understanding of how human pathogenic bacteria enter into plant tissues and persist is needed. Biocontrol measures might be an effective control strategy, for example, inoculating plants with nonharmful bacteria that could out-compete the pathogens. Irradiation as a pasteurization method could reduce the incidence of food-borne pathogens, but this method faces significant consumer resistance.

Eradicating Established Pathogens

Once a disease breaks out, chemical control measures can be used to eradicate it. Copper compounds have been some of the most widely used antimicrobial agents throughout history and until recently were one of the most important measures for control of plant pathogenic bacteria. Copper’s effects are pleiotropic and are not well understood, and these compounds must be used judiciously because they are also phytotoxic. Copper-resistant bacteria have become common since the 1980s, making copper compounds less effective. Various antibiotics have been employed to combat bacterial pathogens, but pathogen resistance to these compounds as well as concerns about their potential contributions to antibiotic resistance in human pathogens have limited their use.

Fungicides are tremendously important tools for crop protection. Ideal fungicides target only the fungi, not the plant host or animals. For example, some target the synthesis of chitin, a component of fungal cell walls. Others exploit variations between fungal and plant or animal metabolism. Systemic fungicides are widely used because they are moved throughout the plant to attack the pathogen internally as well as on the exposed surfaces. The development of novel, safe fungicides is challenging, so growers must take care to curtail fungicide use to slow the rate at which the pathogen populations become resistant. To prevent this phenomenon, growers can alternate fungicides that affect different metabolic processes such as sterol synthesis or cellular respiration.

Case Study: Tomato Spotted Wilt Virus

Tomato spotted wilt virus (TSWV) is responsible for more than a billion dollars in crop losses annually. TSWV affects over a thousand species in 85 families, from bean (Phaseolus vulgaris) to watermelon (Citrullus vulgaris) and amaryllis (Amaryllis spp) to zinnia (Zinnia spp), as well as an unknown number of wild, uncultivated species. Tomato and pepper (Capsicum annum) are susceptible, as well as peanut (Arachis hypogaea), making this one of the most agronomically important viruses. Symptoms are highly variable and range from discoloration to death. A common symptom is white or yellow concentric rings forming on the fruit. TSWV is primarily spread by at least seven different varieties of tiny insects called thrips. Because both the virus and insect have such broad host ranges, management of this disease is extremely difficult. One of the only available tools is applications of insecticides to reduce thrip infestations, but this tactic doesn’t result in complete control, which means that thrips are developing resistance to these insecticides. A few natural sources of resistance to the virus have been identified. For example, the tomato R gene Sw5 encodes a typical coiled-coil NB-LRR R protein that confers resistance to the virus. However, viruses that break Sw5 resistance have already been isolated, so screening for natural resistance is an ongoing priority. The induction of pathogen-derived resistance through expression of the viral coat protein or movement protein is proving to be a successful means of inducing resistance.

Case Study: Cyst and Root-Knot Nematodes

Nematodes, tiny roundworms of the phylum Nematoda, are one of the most abundant groups of animals and one of the most important types of plant pathogens. Many nematodes are free-living, some are human pathogens (including hookworms, pinworms, and Trichinella spiralis, causal agent of trichinosis), and some are plant pathogens. The plant pathogens are sedentary or migratory, and endo- or ecto-parasitic. The most damaging are the sedentary endo-parasites, which move into the root, remodel cells to form a feeding structure, eat, and reproduce. Included in this group are the root-knot nematodes (including Meloidogyne spp) and cyst nematodes (including Heterodera spp and Globodera spp). Globally, plant parasitic nematodes are estimated to cost well over $100 billion in crop losses annually. All plant parasitic nematodes feed through a stylet, which they introduce into the host plant cells. The stylet also introduces proteins into the host cell, many of which are necessary for suppressing the host’s defense responses and establishing the feeding cells. The feeding structures of cyst and root-knot nematodes differ in their formation. Root-knot nematodes cause the formation of a giant cell by inducing cell divisions without cytokinesis. Cyst nematodes induce the formation of syncytia by partially dissolving the cell walls from adjoining cells. Reprogramming root cells into these feeding structures involves changes in auxin transport or sensitivity conferred by the nematode effectors. Some nematodes also produce mimics of plant CLE-like regulatory peptides. Genes encoding cell wall-modifying enzymes are found in plant parasitic nematodes, but not other nematodes, and recent studies suggest that these genes have been acquired by horizontal gene transfer from bacteria.

Managing nematode damage to crop plants is challenging. For 50 years, methyl bromide fumigation of soils has been a widely used method to eliminate nematodes, but methyl bromide is being phased out because it is an atmospheric ozone-depleting agent. The search for effective, less harmful nematicides is ongoing. Practices such as crop rotation and leaving fields fallow are only somewhat effective because many nematodes have a broad host range, and the eggs can live in a dormant state in the soil for years. A few plants show genetic resistance to
nematodes, which is through NB-LRR proteins similar to those that confer resistance to microbial pathogens. Efforts to identify additional R genes and to introduce them into other plants are underway. Another promising approach is to engineer plants that introduce small RNA precursors into the nematodes to silence key parasitism or viability genes.

Biological Control of Pathogens

As early as the end of the 19th century, certain soils were recognized as being suppressive of plant diseases. This feature was later correlated to soil-borne microbes that suppress pathogenic microorganisms or promote plant defense. The application of these microbes as a tactic against pathogen is known as biocontrol.

Some biological control agents attack and kill or parasitize the plant pathogen; as examples, bacteriophage attack viruses that lyse and kill bacteria, the soil-borne fungus Coniothyrium minitans attacks several plant-pathogenic species of Sclerotinia, and fungi of the Trichoderma genus can be parasitic upon pathogenic fungi and oomycetes. Other biocontrol agents produce antibiotics, toxins including hydrogen cyanide, volatile organic compounds such as 1-butanol, or enzymes like chitinase that attack the pathogen. Some biocontrol agents compete with pathogenic microbes for space or nutrients, especially iron. Often a biocontrol species uses more than one of these mechanisms. Soil and foliar inoculations with biocontrol agents are proving to be successful methods of disease suppression in some circumstances and are under intensive study.

A classic example of biocontrol comes from studies of take-all of wheat, which is caused by the fungus Gaeumannomyces graminis var tritici. In a given field, the disease often increases in severity through several growing cycles but then abruptly declines; this latter effect is called take-all decline. Take-all decline appears to stem from the accumulated levels of antibiotics produced by beneficial Pseudomonas strains. One of these, 2,4-diacetylphloroglucinol, is broadly effective against many bacteria, fungi, and nematodes, as well as the protozoa that eat Pseudomonas.

One of the more elusive ways that biocontrol agents can work is called induced systemic resistance. Induced systemic resistance is a broad stimulation or priming of plant defenses that is similar to SAR but triggered by nonpathogenic organisms. Priming allows a plant to produce a more vigorous and rapid defense response. Primed plants have been shown to have increased levels of MAP kinases involved in defense signaling and in some cases to have epigenetic changes to defense genes that facilitate their expression. Bypassing the biocontrol agent completely by priming the plant’s defense system chemically is an exciting possibility for disease control.

Genetic Resistance to Pathogens

Quantitative disease resistance (QDR) refers to resistance conferred by many genes, each of which makes a small contribution. Often, QDR confers nonspecific resistance, essentially by strengthening the plant’s resistance to pathogens. QDR is particularly important in resistance to necrotrophic pathogens. In the past few years, some QDR genes have been identified and found to encode proteins ranging from putative transcription factors and protein kinases, to those involved in hormone synthesis or signaling, as well as completely novel proteins of unknown function. With advances in genome mapping and marker-assisted breeding, it should be feasible to combine many QDRs to produce plants with heightened immunity.

Other efforts toward genetic enhancement of immunity have been more directed and used a gene candidate approach. One of the most successful approaches has been the production of plants engineered to resist viruses through expression of viral coat proteins or small RNAs, which presumably cause silencing of the viral genomes. Other efforts have included the introduction of genes encoding defense proteins or the synthesis of defensive compounds (e.g., reactive oxygen or other antimicrobial compounds; see Collinge et al. [2010] for a comprehensive list). Efforts to eliminate fungal mycotoxins from foods are also being developed through introduction of detoxification genes into plants susceptible to mycotoxin-producing fungi (see below).

Case Study: Rainbow Papaya, the First Commercialized Transgenic Fruit Crop

Papaya ringspot virus occurs wherever papayas (Carica papaya) are grown and causes severe damage to the trees and the fruits. The virus is spread by aphids, and no effective control measures are available other than removing infected trees and abandoning highly affected sites. Papayas in Hawaii began to be infected in the 1940s and the disease nearly eliminated this crop. In the 1980s, a team led by the University of Hawaii and David Gonsalves from Cornell University attempted to engineer resistance to Papaya ringspot virus using a pathogen-derived resistance approach. They introduced a gene to express the viral coat protein in papaya cells and succeeded in regenerating whole plants. Approval was given for these plants to be field tested in 1991 and they proved to be resistant to the virus. After further studies, the genetically modified Rainbow papaya was deregulated in 1998, making it the first commercialized transgenic fruit crop. It currently accounts for more than 90% of Hawaii’s papaya acreage. In 2011, the Government of Japan approved Rainbow papaya for commercial shipment to Japan.

Case Study: Mycotoxins, Serious Food-Borne Fungal Toxins

Some pathogens do more than reduce yields; some pathogenic fungi contaminate plants with toxins known collectively as mycotoxins. There are hundreds of structurally diverse mycotoxins that share the characteristic of toxicity to humans or animals. Aflatoxins and fumonisins are two of the most important in terms of their toxicity and the number of people exposed.

Aflatoxins are secondary metabolites of Aspergillus species, some of which are highly toxic and carcinogenic. Aspergillus
infected plants in the field and is particularly problematic for improperly stored (e.g., moist) grains, nuts, and seeds. In many countries, stored grains and foods are routinely screened for the presence of aflatoxins, but these measures cannot protect people if they are not in place and cannot protect people who eat food that they produce themselves. It has been estimated that 4.5 billion people are regularly exposed to aflatoxins, causing inestimable health damage. Fatal outbreaks occur regularly and affect humans and livestock. Plants that are genetically less susceptible to aflatoxins have been identified or produced (e.g., by expression of antifungal proteins or increased defense responses), and some are being introduced into breeding programs. A biocontrol measure that can reduce aflatoxin infections by 85% or more involves spreading fields with heat-treated (unviable) seeds or hulls that have been coated with non-aflatoxin-producing fungi, which outcompete and suppress growth of these toxic fungi.

*Fusarium graminearum* and related species produce the mycotoxins deoxynivalenol (descriptively also known as vomitoxin) and zearalenone, which acts as an estrogen mimic. *Fusarium verticillioides* produce fumonisins, which have been implicated in neural tube birth defects and cancer in humans, as well as health problems in livestock. *Fusarium* mycotoxins persist through processing and have been identified in processed foods from baby biscuits to beer. Efforts to prevent fungal growth in the field and during post-harvest storage can help reduce the problem, but these efforts are costly and inadequate. Genetic sources of resistance to *Fusarium* are limited, but engineering plants to produce mycotoxin-detoxifying enzymes is promising.

The ergot alkaloids, produced from the *Claviceps* fungus, are responsible for the human disease of ergotism, which was once common (it has been calculated that there were 132 epidemics of ergotism in Europe between the 6th and 18th centuries) but is now rare. Ergot alkaloids warrant mention for two reasons. First, their therapeutic potential has been investigated for a variety of purposes, including the development of a truth serum (LSD) but also for the treatment of migraine headaches and Parkinson’s disease. Second, there has been speculation (and counterarguments) that an outbreak of ergotism was responsible for the infamous witchcraft trials of the 17th century in Salem, Massachusetts.

**SUMMARY AND ONGOING RESEARCH**

The study of plants and their pathogens stems from the impact that plant disease has on society and is in some ways a highly applied field of biology, not dissimilar from that of human medicine. Plant pathologists observe, diagnose, and treat their “patients” and practice prophylactic measures to prevent disease outbreaks. Major research thrusts include efforts to understand what confers pathogenicity upon an organism, and this is greatly accelerated by the recent accumulation of genomic sequence data for large numbers of pathogens. The ability to colonize and extract nutrients from living plants is more complex than simply breaking down dead plant materials. Pathogens are diverse but all must overcome the barriers and defenses that plants employ to protect themselves. In parallel with our increasing understanding of pathogens, we have a rapidly expanding understanding of how plants defend themselves. The models by which we interpret plant responses to pathogens increase in sophistication with astounding speed. For example, the first effector and *R* genes were cloned just ~25 and 20 years ago; before this, they were only known through genetic studies. As always, the more we know, the more we find we have to learn, and there are still major gaps in our understanding of plant disease processes. Furthermore, as a consequence of the continual arms race between pathogens and plants, accompanied by the added variable of rapid global change, there is no fixed end point to our investigations. The interactions between plants and their pathogens continues to be one of the most exciting and impactful areas of plant biology, and we agree with the thought expressed by Martinus Beijerinck, whose work helped to identify tobacco mosaic virus in the 1890s, “Fortunate are those who now start.”

**NOTE ABOUT TERMINOLOGY**

As we better understand the interactions between plants and their pathogens at the molecular level, some of the terminology that has long been associated with this discipline is being phased out in favor of more accurate or intuitive words. We have adopted the evolved terminology in this article, but here would like to introduce some of the terms that students will encounter as they explore some of the literature.

**Avirulence or Avr Genes**

This is a term that has its roots in classical genetic studies. A pathogen that carries an *Avr* gene was considered nonvirulent (avirulent) on a plant that carried a corresponding *R* gene; products of the *Avr* genes are effectors. The association between *Avr* genes and *R* genes are described as the familiar gene-for-gene interaction described by Harold Flor and found in most textbooks.

**Compatible and Incompatible Interactions**

These terms describe the host/pathogen interaction from the pathogen’s perspective. A compatible interaction indicates an interaction in which the host is susceptible to the pathogen and the pathogen succeeds, and in an incompatible interaction the host is resistant and the pathogen is unsuccessful.

**Host and Nonhost Resistance**

Nonhost resistance indicates that an entire species is not a host for a particular pathogen; for example, rice may display nonhost resistance to an *Arabidopsis* pathogen. By contrast, host resistance indicates that a particular variety resists a pathogen within a group of susceptible plants; wheat carrying the Sr31 gene displays host resistance to most varieties of stem rust fungi. Mechanistically there is some overlap between host and nonhost resistance, leading to confusion, so we have avoided these terms.
RECOMMENDED READING

(This is a representative list of sources to help the reader access a huge body of literature. We apologize in advance to those whose work is not included.)

General References


Strategies of Pathogenicity and Pathogen Effectors


**Plant Immune Responses**


Strategies to Prevent and Manage Disease


