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Microbial Toxins and Disease Development in Plants

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1. MICROBIAL TOXINS AND PLANT PATHOGENESIS

Microbial toxins are those that produced by algae and may be accumulated in edible aquatic organisms such as shellfish (phycotoxins) are also included in this standard. Mycotoxins and phycotoxins are both subclasses of contaminants.

Inherent natural toxicants that are implicit constituents of foods resulting from a genus, species or strain ordinarily producing hazardous levels of a toxic metabolite(s), i.e. phytotoxins are not generally considered within the scope of this standard. They are, however, within the terms of reference of the CCFAC and will be dealt with on a case-by-case basis.

Bacterial products include bacteria and their toxins (microbial toxins). Poisoning resulting from microbial toxins is, by convention, known as bacterial food poisoning. The illness is characterized by: (1) simultaneous poisoning of many persons at the same time; (2) history of ingestion of common food by all sufferers; and (3) similarity of signs and symptoms in a majority of cases.[1,2]

The term "microbial toxin" is usually reserved by microbiologists for toxic substances produced by microorganisms that are of high molecular weight and have antigenic properties; toxic compounds produced by bacteria that do not fit these criteria are referred to simply as poisons. Many of the former are proteins or mucoproteins and may have a variety of enzymatic properties. They include some of the most toxic substances, such as tetanus toxin, botulinus toxin, and diphtheria toxin. Bacterial toxins may be extremely toxic to mammals and may affect a variety of organ systems, including the nervous system and the cardiovascular system. A detailed account of their chemical nature and mode of action is beyond the scope of this volume. The range of poisonous chemicals produced by bacteria is also large. Again, such compounds may be used for beneficial purposes; for example, the insecticidal properties of *Bacillus thuringiensis*, due to a toxin, have been utilized in agriculture for some time.

Over millions of years of evolution, microbial toxins have developed activities and specificities that are many times greater than chemical drugs. These qualities make the toxins, with their highly complex multidomain structures and associated functions, superbly precise biochemical and clinical tools. One of the most fascinating aspects in the field of *C. botulinum* studies in recent years has been the development of the world's most potent naturally occurring toxin into a molecule of significant therapeutic utility.[3,4] The drive to use this toxin came from the medical field and it was the pioneering experiments of Alan Scott and others in the 1970s that led to the first use of botulinum toxin in a medical context. It occurred to Scott that the local paralytic effects induced by injection of botulinum toxin into muscle could be used therapeutically to relax specific muscles. His initial experiments were directed at treating strabismus (squint), with a preparation of botulinum neurotoxin A supplied by Schantz. The success of these initial experiments led Scott and others to explore the use of botulinum neurotoxin in a number of medical indications and ultimately led to the development of large-scale manufacturing facilities to meet the demand for the toxin.

The threat of purposeful transmission of airborne pathogenic microorganisms and microbial toxins as weapons of mass destruction (WMD) has increased the awareness of the importance of bioaerosols. The Centers for Disease Control and Prevention and the Department of Health and Human Services have listed several pathogenic microorganisms as select

agents. Of these, *Francisella tularensis, Yersinia pestis, Bacillus anthracis*, and *Variola virus*, the causative agents of tularemia, plague, anthrax, and smallpox, respectively, are ones that can be dispersed via aerosol and, therefore, are a concern for inhalation infection. Moreover, availability of new technologies, such as genetic engineering, may provide the tools to modify innocuous microorganisms or the transmission routes of other pathogens, involving a higher biosafety risk for the population[5,6] Continued researches and awareness by public health professionals are needed to recognize these diseases and minimize the risk of exposure of the population. For example, the uses of biosensors to detect airborne pathogens are being developed and installed in official buildings and strategic sites, that allow a rapid detection and intervention.

Microbial toxins are toxins produced by micro-organisms, including bacteria, fungi, protozoa, dinoflagellates, and viruses. Many microbial toxins promote infection and disease by directly damaging host tissues and by disabling the immune system. Endotoxins most commonly refer to the lipopolysaccharide (LPS) or lipooligosaccharide (LOS) that are in the outer plasma membrane of Gram-negative bacteria. The botulinum toxin, which is primarily produced by *Clostridium botulinum* and less frequently by other *Clostridium* species, is the most toxic substance known in the world. However, microbial toxins also have important uses in medical science and research.[7,8] Currently, new methods of detecting bacterial toxins are being developed to better isolate and understand these toxin. Potential applications of toxin research include combating microbial virulence, the development of novel anticancer drugs and other medicines, and the use of toxins as tools in neurobiology and cellular biology.

Bacteria toxins which can be classified as either exotoxins or endotoxins. Exotoxins are generated and actively secreted; endotoxins remain part of the bacteria. Usually, an endotoxin is part of the bacterial outer membrane, and it is not released until the bacterium is killed by the immune system. The body's response to an endotoxin can involve severe inflammation. In general, the inflammation process is usually considered beneficial to the infected host, but if the reaction is severe enough, it can lead to sepsis. Exotoxins are typically proteins with enzymatic activity that interfere with host cells triggering the symptoms associated with the disease. Exotoxins are also relatively specific to the bacteria that produce it; for example, diphtheria toxin is only produced by *Corynebacterium diphtheriae* bacteria and is required for the diphtheria disease. Some bacterial toxins can be used in the treatment of tumors. Endotoxins most commonly refer to the lipopolysaccharide (LPS) or lipooligosaccharide (LOS) that are in the outer plasma membrane of Gram-negative bacteria.[9,10] Exotoxins are typically proteins with enzymatic activity that interfere with host cells triggering the symptoms associated with the disease. Exotoxins are also relatively specific to the bacteria that produce it; for example, diphtheria toxin is only produced by *Corynebacterium diphtheriae* bacteria and is required for the diphtheria disease. Not all strains of a bacteria species are virulent; there are some strains of *Corynebacterium diphtheriae* that do not produce diphtheria toxin and are considered nonvirulent and nontoxigenic. Additional classifications used to describe toxins include enterotoxin, neurotoxin, leukocidin or hemolysin which indicate where in the host's body the toxin targets.. Enterotoxins target the intestines, neurotoxins target neurons, leukocidin target leukocytes (white blood cells), and hemolysins target red blood cells. Exotoxin activity can be separated into specific cytotoxic activity or broad cytotoxic activity based on whether the toxin targets specific cell types or various cell types and tissues, respectively. Lethal toxins refers to the group of toxins that are the obvious agents responsible for death associated with the infection.

Toxinosis is pathogenesis caused by the bacterial toxin alone, not necessarily involving bacterial infection (e.g. when the bacteria have died, but have already produced toxin, which are ingested). It can be caused by *Staphylococcus aureus* toxins, for example.[11,12]

There are over 200 *Clostridium* species in the world that live in mundane places such as soil, water, dust, and even our digestive tracts. Some of these species produce harmful toxins such as botulinum toxin and tetanus toxin among others. Most clostridium species that do have toxins typically have binary toxins with the first unit involved in getting the toxin into the cell and the second unit cause cellular stress or deformation. Clostridial toxins are widespread and aid in the production of many diseases in humans and other organisms. Clostridial toxins are known to aid in gastrointestinal diseases and there is a wide range of mechanisms that clostridial toxins take to invade or enter the cell of the host. Pore forming bacterial toxins are common and have a very interesting way of entering or invading the host's cell. The mechanism that clostridial toxins follow includes clostridia forming pores and then the pores inserting themselves into the cell membrane of cells. Clostridial toxins have the ability to damage or alter the cell membrane damaging the extracellular matrix of the organism.

Toxin A and toxin B are two toxins produced by *Clostridium difficile*. Toxin A and toxin B are glycosyltransferases that cause the antibiotic-associated pseudomembranous colitis and severe diarrhea that characterize disease presentation of *C. diff* infections.[13,14]

Botulinum neurotoxins (BoNTs) are the causative agents of the deadly food poisoning disease botulism, and could pose a major biological warfare threat due to their extreme toxicity and ease of production. They also serve as powerful tools to treat an ever expanding list of medical conditions that benefit from its paralytic properties, an example drug with BoNTs as the active ingredient is Botox. They also serve as powerful tools to treat an ever expanding list of medical conditions that benefit from its paralytic properties delivered through localized injections, an example drug with BoNTs as the active ingredient is Botox. Botulinum neurotoxins (BoNTs) are protein neurotoxins that are produced by the bacteria *Clostridium*. BoNTs are now largely being studied due to their ability to aid in chronic inflammatory diseases such as acne, multiple sclerosis, and for cosmetic purposes

Clostridium tetani produces tetanus toxin (TeNT protein), which leads to a fatal condition known as tetanus in many vertebrates (including humans) and invertebrates. While tetanus toxin is produced from *Clostridium tetani*, a spore forming bacteria found in soil, Tetanus is a paralytic disease that is global and commonly affects newborns as well as non-immunized individuals. Tetanus enters the body of organisms through wounds or skin breaks and can be found in manure, soil, and dust. Tetanus [15,16] mechanism includes tetanus preventing the transmission of glycine and γ-aminobutyric acid from inhibitory interneurons in the spinal cord, leading to spastic paralysis. Glycine is an important amino acid that is essential for adequate nervous system function aiding in cell communication throughout the body. When tetanus toxin enters the body it is taken up by cholinergic nerve endings traveling axonally into the brain and spinal cord, disrupting motor function in individuals. Although tetanus is a damaging toxin that has a multitude of symptoms it can be prevented through vaccination. *Clostridium perfringens* is an anaerobic, gram-positive bacteria that is often found in the large and small intestines of humans and other animals. *Clostridium perfringens* has the ability to reproduce quickly producing toxins relating to the cause of diseases. The pore-forming toxin perfringolysin has the ability to cause gangrene in calves with the presence of alpha toxin. Immune evasion proteins from *Staphylococcus aureus* have a significant conservation of protein structures and a range of activities that are all directed at the two key elements of host immunity, complement and neutrophils. These secreted virulence factors assist the bacterium in surviving immune response mechanisms.

Examples of toxins produced by strains of *S. aureus* include enterotoxins that cause food-poisoning, exfoliative toxins that cause scalded skin syndrome, and toxic-shock syndrome toxin (TSST) that underlies toxic shock syndrome. These toxin examples are classified as superantigens.[17,18]

Multi-drug resistant *S. aureus* strains also produce alpha toxin, classified as a pore-forming toxin, which can cause abscesses. Shiga toxins (Stxs), responsible for foodborne illnesses, are a classification of toxins produced by Shiga toxin-producing *Escherichia coli* (STEC) and *Shigella dysenteriae* serotype 1. Stx was first identified in *S. dysenteriae* and was later found to be produced by certain strains of *E. coli*.Stxs act through inhibiting protein synthesis of infected cells and can be divided into two antigenically different groups: Stx/Stx1 and Stx2. Stx1 is immunologically equivalent to Stx; however, it received a separate name to distinguish that it's produced by STEC not *S. dysenteriae*. Stx2 is produced only by STEC and is antigenically different from Stx/Stx1. The term shiga-like toxins was previously used to further distinguish the shiga toxins produced by *E. coli,* but nowadays, they are collectively referred to as shiga toxins.Within the STEC strains, a subgroup classified as enterohemorrhagic *E. coli* (EHEC) represent a class of pathogens with more severe virulence factors in addition to the ability to produce Stxs. EHEC infections result in more severe diseases of hemorrhagic colitis and hemolytic uremic syndrome. There are around 200 strains of STEC, and the wide range of diversity and virulence between them can be partly attributed to phage-mediated horizontal transfer of genetic material.

Anthrax disease in humans results from infection with toxin producing *Bacillus anthracis* strains that can be inhaled, ingested in contaminated food or drink, or obtained through breaks in the skin like cuts or scrapes. Domestic and wild animals can also be infected via inhalation or ingestion. Depending on the route of entry, disease can present initially as inhalation anthrax, cutaneous anthrax, or gastrointestinal anthrax, but eventually will spread throughout the body, resulting in death, if not treated with antibiotics. Anthrax toxin is composed of three domains: protective antigen (PA), edema factor (EF), and lethal factor (LF). EF is an adenylate cyclase that targets ATP. LF enzyme is a metalloprotease that

confers the lethal phenotype associated with anthrax disease. As LF is the agent responsible for the death of infected hosts, it is classified in the group of lethal toxins.[19,20]

Diphtheria toxin is produced by virulent *Corynebacterium diphtheriae* that infect the mucosal membranes of the throat and nasal cavity causing a gray, thickened lining of the throat, sore throat, weakness, mild fever, swollen glands of the neck, and difficulty breathing. Diphtheria toxin is an ADP-ribosyltransferase that inhibits protein synthesis which causes the symptoms associated with the disease. Diphtheria used to be a leading cause of childhood death until the creation of a vaccine. The diphtheria vaccine contains a diphtheria toxoid, antigenically identical yet inactivated and non-toxic. When the toxoid is introduced to the body in a vaccine, an immune response is mounted without sequelae associated with the toxigenicity.

Mycotoxins are secondary metabolites that are constructed by microfungi. Mycotoxins can be harmful because they can cause disease and death in humans and animals. They are found in many pharmaceuticals like antibiotics and growth developments. Mycotoxins can also play a role in chemical warfare agents, CWA, which are chemicals that contain toxins that are used to cause death, harm, or injuries to individuals that are considered enemies by the military during warfare.

Mycotoxins are synthesized by different types of moulds and are built by a wide group of toxins. Mycotoxins have a low molecular weight compound that is usually less than 1000 grams per mol. There are roughly 400 toxic mycotoxins that are constructed by 100 different fungi species that have been researched. Mycotoxins gain access into the body of a human or animal by food, they can contaminate many different types of agriculture during cultivation, harvesting, storage, and areas with high humidity. The Food and Agriculture Organization reported that about 25% of products produced by agriculture contain mycotoxins and this can lead to economic losses in the agricultural community. Levels of Mycotoxins secretion can rely on varying temperatures, the ideal temperature for Mycotoxins to grow is from 20 degrees Celsius to 37 degrees Celsius.[21,22] Mycotoxin production also relies heavily on water activity, the ideal range would be from 0.83 to 0.9 aw and higher. Humidity plays a key in the production of Mycotoxins as well. The higher the humidity levels, between 70% to 90%, and moisture levels, 20% to 25%, allow the Mycotoxins to grow more rapidly. Foods that Mycotoxins are found in cereal, spices, and seeds. They can also be found in eggs, milk, and meat from animals that have been contaminated during their feeding process. Since they are resistant to high temperatures and physical and chemical reception, it is considered unavoidable while cooking at high temperatures.

Trichothecenes is a mycotoxin that is produced from the fungi species, *Fusarium graminearum*. The T-2 toxin, Type A, and DON, Type B, are major mycotoxins that are responsible for toxicity in humans and animals. These two types come from an epoxide at the C12 and C13 positions in the trichothecenes. The T-2 toxin was found after civilians ate wheat that was contaminated by the *Fusarium* Fungi, during WWII from a biological weapon, the T-2 toxin was an outbreak and made humans develop symptoms like food poisoning, chills, nausea, dizziness, etc. The Trichothecenes mycotoxin affects animals by decreasing plasma glucose, blood cell and leukocyte counts. Pathological changes in the liver and stomach, as well as weight loss has been accounted for.

Zearalenone is a mycotoxin that is produced from *Fusarium graminearum* and *Fusarium culmorum* that are found in different types of foods and feeds. Zearalenone is a non-steroidal estrogenic mycotoxin that is found in farm animal's reproductive disorders and in humans it causes hypoestrogenic syndrome. Effects that come from Zearalenone include, enlarged uterus, improperly running reproductive tract, decreasing the fertility in women, and causes progesterone and estradiol levels to become not norma.[23,24] If Zearalenone is consumed during pregnancy, it can cause reduced fetal weight and decrease the chance of survival for the embryo.

Fumonisins, *Fusarium verticillioides*, is found in nature where fumonisin B1 has largely contaminated the area. These mycotoxins are hydrophilic compounds. Studies have shown that esophageal cancer can be related back to corn grain that contains fumonisins. Other effects from fumonisins are birth defects of the brain, spine, and spinal cord. In animals, problems with the pulmonary edema and hydrothorax swine's have been proven to have association with fumonisins.

Ochratoxin is a mycotoxin that is produced by *Aspergillus* species and *Penicillium* species. The most researched ochratoxin is the Ochratoxin A (OTA), which is a fungal toxin. This mycotoxin targets the OTA of kidneys and causes kidney disease in humans. Ochratoxin A is an immunosuppressive compound. Ochratoxin is a renal carcinogen, which has been found by animals containing OTA.

Aflatoxin is a mycotoxin that is produced from *Aspergillus flavus* and *Aspergillus parasititcus*. A type of Aflatoxin, AFB1 is the most common mycotoxin that is found in human food and animal feed. AFB1 targets the liver of both humans and animals. Acute aflatoxicosis can make humans and animals have symptoms like abdominal pain, vomiting, and even death.[25,26]

In the history of phytopathology, microbial toxins have been the objects of extensive studies as possible pathogenicity or virulence factors for the producer pathogens. The recent development of molecular genetic techniques provided an experimental basis to thoroughly test the role of these secondary metabolites in pathogenesis. Some of them did prove to be highly associated with disease initiation or enhanced virulence in certain plant-pathogen interactions. In this review, we describe recent progresses in the field of plant-pathogen interactions focusing on two toxins; i.e., tabtoxin from *Pseudomonas syringae* and trichothecenes from *Fusarium* and other fungi. These microbial toxins have convincingly been shown to play causal roles in plant disease development. Studies on the biosynthesis and resistance mechanisms of these producers are outlined, and the significance of this knowledge is discussed in relation to practical applications in agriculture.

Pathogenic fungi and bacteria often damage their host (plants) tissues by producing toxic metabolites, which induced various symptoms such as necrosis, chlorosis, wilting, water soaking and eventually the death of plants. These toxic metabolites also known is one of the weapons used by pathogen inducing disease condition in susceptible host plants. Many pathogens are known to produce toxins both *in vitro* and *in vivo* and these toxins have been implicated in the symptom development on the host tissues. Many of these phytotoxic metabolites have also been extracted from diseased plant tissues. Based on the reactions of host crops to the toxic metabolites of respective hosts, methods of rapid screening of germplasm for resistance to plant diseases have been developed. [27] Their application has successfully resulted in resistant lines in some tropical crops like cowpea, cassava, maize, yam, and soybean. Nowadays, these evaluation techniques are becoming an important complement to classical breeding methods. The knowledge of the inactivation of microbial toxins has led to the use of microbial enzymes to inactivate phytotoxins thereby reducing incidence and severity of disease induced by microbial toxins. Considering the increasing awareness of herbicide resistance, and the restriction of the use of chemical pesticides in agriculture against plant pathogens, novel compounds from microorganisms also provided new environmentally friendly bio-herbicides for the control of parasitic weeds that are normally difficult to control.

2. INFECTION IN PLANTS BY MICROBES

Although considered structurally simple, bacteria are extremely diverse from a metabolic standpoint and are found almost everywhere on Earth in vast numbers—from living in jet fuel and on the rims of volcanoes to thriving in hydrothermal vents deep on the ocean floor. There are both beneficial and pathogenic bacteria. Beneficial bacteria are involved in such diverse processes as digestion in animals, nitrogen fixation in the roots of certain legumes, the decomposition of animal and plant remains, and sewage disposal systems. Pathogenic bacteria, on the other hand, cause severe and often fatal diseases in humans, animals and plants. The first bacterial disease ever discovered was anthrax (caused by *Bacillus anthracis*) of cattle and sheep in 1876. The discovery of anthrax in cattle was immediately followed by the discovery of fire blight of pear and apple (caused by *Erwinia amylovora*) by T. J. Burrill from the University of Illinois (1877–1885). Another group of bacterial pathogens are difficult or impossible to culture in the laboratory and are called fastidious vascular bacteria. They grow in either the xylem or phloem tissues and interfere with the transport of water and nutrients in the plant. Many of them are vectored by sucking insects such as leafhoppers, planthoppers and psyllids. Studies of corn stunt provide evidence that once the insect vectors establish the infective particles in their bodies, the insects retain the ability to transmit them the rest of their lives. Until their discovery in 1967, most of the diseases now known to be caused by fastidious vascular bacteria were believed to be caused by viruses and were initially described by virologists.

The taxonomy of plant pathogenic bacteria is currently in flux based on recent advances on how bacteria are classified. Most plant pathogenic bacteria belong to the following genera: *Erwinia, Pectobacterium, Pantoea, Agrobacterium, Pseudomonas, Ralstonia, Burkholderia, Acidovorax, Xanthomonas, Clavibacter, Streptomyces, Xylella, Spiroplasma,* and *Phytoplasma.* Plant pathogenic bacteria cause many different kinds of symptoms that include galls and overgrowths, wilts, leaf spots, specks and blights, soft rots, as well as scabs and cankers. In contrast to viruses, which are inside host cells,

walled bacteria grow in the spaces [21,22] between cells and do not invade them. The means by which plant pathogenic bacteria cause disease is as varied as the types of symptoms they cause. Some plant pathogenic bacteria produce toxins or inject special proteins that lead to host cell death or they produce enzymes that break down key structural components of plant cells and their walls. An example is the production of enzymes by soft-rotting bacteria that degrade the pectin layer that holds plant cells together. Still others colonize the water-conducting xylem vessels causing the plants to wilt and die. *Agrobacterium* species even have the ability to genetically modify or transform their hosts and bring about the formation of cancer-like overgrowths called crown gall.

Bacteria that cause plant diseases are spread in many ways—they can be splashed about by rain or carried by the wind, birds or insects. People can unwittingly spread bacterial diseases by, for instance, pruning infected orchard trees during the rainy season. Water facilitates the entrance of bacteria carried on pruning tools into the pruning cuts. Propagation with bacteria-infected plant material is a major way pathogenic bacteria are moved over great distances. No matter how the bacterial pathogens are disseminated, they require a wound or natural opening, such as stomata, to get inside a plant host. Once inside they then kill host cells, by the means described above, so that they can grow. Between hosts they may grow harmlessly on plant surfaces and then can overwinter or survive unfavorable environmental periods or the absence of a susceptible host by either going dormant in infected tissue, infested soil or water, or in an insect vector.

Losses in crop yields due to disease need to be reduced in order to meet increasing global food demands associated with growth in the human population. There is a well-recognized need to develop new environmentally friendly control strategies to combat bacterial crop disease. Current control measures involving the use of traditional chemicals or antibiotics are losing their efficacy due to the natural development of bacterial resistance to these agents. In addition, there is an increasing awareness that their use is environmentally unfriendly. Bacteriophages, the viruses of bacteria, have received increased research interest in recent years as a realistic environmentally friendly means of controlling bacterial diseases. Their use presents a viable control measure for a number of destructive bacterial crop diseases, with some phage-based products already becoming available on the market. Phage biocontrol possesses advantages over chemical controls in that tailor-made phage cocktails can be adapted to target specific disease-causing bacteria. Unlike chemical control measures, phage mixtures can be easily adapted for bacterial resistance which may develop over time.

Bacterial diseases of plants are caused by six genera of bacteria, that is, *Agrobacterium*, *Corynebacterium*, *Erwinia*, *Pseudomonas*, *Streptomyces*, and *Xanthomonas*. The effective control of bacterial diseases in orchards is mostly based on cooper compounds which can cause phytotoxic effect, also some fungicides and different antibiotic preparations. The protection against bacterial diseases requires a new approach based on natural compounds (including botanical extracts and essential oils) and resistance inducers which applied with other substances can contribute to the integrated control of bacterial diseases

Among the bacterial diseases of plants, the most widespread and destructive losses are caused by the Gram-negative bacteria of the genus, *Erwinia*, *Pseudomonas*, and *Xanthomonas*. The genus X*anthomonas* is of great economic importance because of its broad host range. Collectively, members of the genus cause disease on at least 124 monocot species and 268 dicot species, including fruit and nut trees,[23] solanaceous and brassicaceous plants, and cereals. They cause a variety of symptoms like cankers, necrosis, blight, and spots, affecting a variety of plant parts, including leaves, stems, and fruits. The collectively broad host range of the genus contrasts strikingly with the typically narrow host range of individual species and pathovars, which typically also exhibit a marked tissue specificity, infecting either through stomates to colonize the intercellular spaces of the mesophyll parenchyma, via hydathodes (water pores at the leaf margin), or via wounds to spread systemically through the vascular system. The type of physiological function that is affected first depends on the cells and tissues of the host plant that become infected. Thus, the infection of xylem vessels interferes with the translocation of water, leading to vascular wilts and cankers, whereas infection of foliage interferes with the photosynthetic process as in leaf spots, blights, and pustules. Angular leaf-spot disease of cotton is caused by *X. campestris* pv. *malvacearum*. The disease is present wherever cotton is grown. The bacterium attacks the leaves as well as young cotton bolls. In rice, *X. campestris* pv. *oryzae* causes leaf blight disease. Bacterial blight or stripe of several cereals and streak of sorghum and maize is caused by *X. campestris* pv. *translucens*. *X.*

campestris pv. *juglandis* causes blight of walnuts. The bacterial pustule disease of soybean caused by *X. campestris* pv. *glycines* is known to cause considerable losses in yield (Figure 3). Gumming disease of sugarcane affecting yields of sugar is caused by *X. campestris* pv. *vasculorum*.

Among the bacterial diseases of plants, the most widespread and destructive losses are caused by the Gram-negative bacteria of the genus, *Erwinia*, *Pseudomonas* and *Xanthomonas*. The genus *Xanthomonas* is of great economic importance because of its broad host range. More than 145 species of plants are known to be infected, mainly by biotrophic pathogens, belonging to this genus. These pathogens cause many types of disease symptoms including spots, blights, cankers and vascular wilts. The type of physiological function that is affected first depends on the cells and tissues of the host plant that become infected. Thus the infection of xylem vessels interferes with the translocation of water leading to vascular wilts [24,25] and cankers, whereas infection of foliage interferes with the photosynthetic process as in leaf spots, blights, and pustules.

Angular leaf spot disease of cotton is caused by *X.c.* pv. *malvacearum*. The disease is present wherever cotton is grown. The bacterium attacks the leaves as well as young cotton bolls. In rice, *X.c.* pv. *oryzae* causes leaf blight disease. Bacterial blight or stripe of several cereals and streak of sorghum and maize is caused by *X.c.* pv. *translucens*. *X.c.* pv. *juglandis* causes blight of walnuts. In the case of soybean, bacterial pustule disease caused by *X.c.* pv. *glycines* is known to inflict considerable losses in yield. Gumming disease of sugarcane affecting yields of sugar is caused by *X.c.* pv. *vascularum*.

Bacterial pathogen–plant interactions involve an interplay of the various virulence factors, the hypersensitivity response and pathogenicity (*hrp*) and avirulence (*avr*) genes of the pathogen and the disease resistance genes in plants. The virulence factors comprise agents such as the hydrolytic enzymes, toxins, polysaccharides, and plant growth regulators secreted by the pathogen that damage or alter plant cells and provide optimal environment for the growth of the pathogen. On the other hand avirulence factors or the products of avirulence genes of the pathogen invoke hypersensitive response and death of the surrounding cells in the resistant host. This restricts the spread of the pathogen and in turn restricts its host range. *Hrp* genes in the pathogen regulate both the *avr*-induced hypersensitivity reaction as well as pathogenicity.

Unlike viruses, most bacterial diseases of plants do not require insects as vectors, relying instead on rain, wind, soil, seed dispersal or other means of transport and entry to plants. However, insect vectors contribute to the spread of some bacterial pathogens of plants. Fireblight is an important bacterial disease of pome fruits such as pears and apples in which flower-visiting insects may have an important role in disseminating the causal bacterium (*Erwinia amylovora*) among blossoms. Insects are not essential, however, for fireblight to spread within plants once the bacteria are established, and there is little vector specificity among flower-visiting insects. Bacteria that rot potatoes (*Erwinia caratovora*) may be transported from infested potato tubers to uninfested tubers by flies whose maggots feed on plant roots or seeds beneath the soil. There is much greater vector specificity in corn flea beetle transmission of the bacterium (*Erwinia stewartii*) that causes Stewart's wilt of corn and cucumber beetle transmission of the bacterium (*Erwinia tracheiphila*) that causes curcurbit wilt, an important disease of melons, squash, and cucumbers. The bacteria enter feeding wounds made by the beetle vectors, but not much is known of how the beetles introduce the bacteria into plants. Overwintering adult beetles provide an important way for these bacteria to survive the winter season without host plants.

Some bacterial pathogens are specialized parasites of plant vascular systems and require insect vectors for plant-to-plant movement and to enter and infect plants. These bacterial pathogens are specialized for vector transmission and for living in plant vascular systems. Examples are the mollicutes (bacteria that lack a rigid cell wall) that live exclusively in the nutrient-rich phloem tissues. A few bacterial pathogens with rigid cell walls, such as the bacterium that causes citrus greening disease, also specialize in living within plant phloem sap. The citrus greening bacterium is transmitted by psyllids (superfamily Psylloidea). The mollicute plant pathogens include phytoplasmas and spiroplasmas. Most of the helical-shaped spiroplasma pathogens of plants, such as the spiroplasma that causes citrus stubborn disease (*Spiroplasma citri*) and the corn stunt spiroplasma (*Spiroplasma kunkelii*), can be cultured on artificial media. So far, none of the phytoplasma (formerly known as mycoplasma-like organism) plant pathogens have been cultured. Examples of economically important phytoplasmas are aster yellows phytoplasma in lettuce, carrot, celery, and other flower and vegetable crops and X-disease phytoplasma in stone fruits such as peach or cherry. Lethal yellowing disease of palms has been a major factor in killing coconut palms in Africa and the Caribbean region. Both phytoplasmas and spiroplasmas are more specialized for parasitizing

insects rather than plants because they can successfully colonize, and more importantly, can be transmitted by only a few species of insects. The most important vectors of mollicute plant pathogens are leafhoppers and planthoppers, but psyllids are an important third group of Hemiptera that are vectors. The pear psylla (*Cacopsylla pyricola*) transmits the pear decline phytoplasma that causes the widespread pear decline disease. Typically, only one or a few species of insects within one of these families have been shown to transmit any particular mollicute. In contrast to their high degree of vector specificity, phytoplasmas and spiroplasmas can parasitize a typically wide range of plant species if the vectors can feed successfully on the plants.

Transmission appears to require that the mollicutes be taken up by vector feeding, penetrate the gut and multiply within the vector's body cavity, enter the salivary glands, and be expelled with saliva during vector feeding into functioning phloem tissues. Thus, it not surprising that vector transmission of various phytoplasmas or spiroplasmas requires a LP ranging from 1 to over 4 weeks. The length of the LP may be very sensitive to temperature, probably because the mollicutes must multiply within the vector for transmission to occur and multiplication is temperature sensitive.[26,27]

Vector-borne bacterial species that parasitize the water-conducting part of the plant's vascular system (xylem) are less numerous but cause some important plant diseases. One such pathogen is *Xylella fastidiosa*, best known as the cause of Pierce's disease of grapes, but other strains of this bacterium cause important other diseases of citrus, coffee, peach, and other crop and forest plants. Sucking insects in several families that feed primarily on xylem sap are *Xylella* vectors. This includes sharpshooter leafhoppers in the subfamily Cicadellinae of the leafhopper family Cicadellidae and spittlebugs (family Cercopidae). Vectors appear to transmit the bacterium from their foregut without any required LP, but continue to transmit for weeks or even months as adults. An immature vector (nymph) stops transmitting after molting its exoskeleton. Sumatra disease of cloves in Indonesia, caused by the xylem sap-inhabiting bacterium *Pseudomonas syzygii*, is spread by tube-building spittlebugs (family Machaerotidae), which are also xylem sap-feeders.

In crown gall, a disease caused by the bacterium *A. tumefaciens* on more than a hundred plant species, galls or tumors develop on the roots, stems , leaves, ears, tassels, and petioles of host plants. Crown gall tumors develop when crown gall bacteria enter fresh wounds on a susceptible host. Immediately after wounding, cells around the wound produce various phenolic compounds and are activated to divide. *Agrobacterium* bacteria do not invade cells but attach to cell walls, and, in response to phenolic compounds such as acetosyringone and other signals, they become activated and begin processing the DNA in their Ti plasmid (for tumor-inducing plasmid. During the intense cell division of the second and third days after wounding, the plant cells are somehow conditioned and made receptive to a piece of bacterial plasmid DNA (called T-DNA, for tumor DNA). Proteins coded by genes in the T-DNA virulence (Vir) region cut out a single strand of the T-DNA from the Ti plasmid and transfer it into the plant cell nucleus as a T-DNA–protein complex. The T-DNA then becomes integrated into the nuclear plant DNA (chromosomes) and some of its genes are expressed and lead to the synthesis of auxins and cytokinins, which transform normal plant cells into tumor cells. Tumor cells subsequently grow and divide independently of the bacteria, and their organization, rate of growth, and rate of division can no longer be controlled by the host plant.

The integrated T-DNA also contains genes that code for substances known as opines. Transformed plant cells produce opines, which can be used only by the intercellularly growing crown gall bacteria as a source of food. Although the increased levels of IAA and cytokinins of tumor cells are sufficient to cause the autonomous enlargement and division of these cells once they have been transformed to tumor cells, high IAA and cytokinin levels alone cannot cause the transformation of healthy cells into tumor cells. What other conditions or substances are involved in the transformation of healthy cells into tumor cells is not known.

In the knot disease of olive, oleander, and privet, another hyperplastic disease caused by the bacterium *Pseudomonas savastanoi*, the pathogen produces IAA, which induces infected plants to produce galls. The more IAA a strain produces, the more severe the symptoms it causes. Strains that do not produce IAA fail to induce the formation of galls. The bacterial genes for IAA production are in a plasmid carried in the bacterium, but some IAA synthesis is also carried out by a gene in the chromosome of the bacterium.

In the leafy gall disease of many plants caused by the bacterium *Rhodococcus fascians*, leafy galls are produced that consist of centers of shoot overproductions and shoot growth inhibition. The bacterium exists mostly at the surface of the plant tissues, but it can also grow internally in the plant. Auxin, cytokinins, and other hormonal substances are produced by the bacterium in cultured and by infected

tissues. Signals from bacteria involved in the development of symptoms initiate new cell divisions and formation of shoot meristem in tissues already differentiated. The bacterial signals originate in genes located on a linear plasmid and exert activities much more unique and more complex than those of cytokinins alone.[18,19]

3. CONTROL OF MICROBIAL DISEASES IN PLANTS

Plant diseases have caused severe losses to humans in several ways. Starvation and uprooting of families resulted from the Irish famine caused by potato late blight (caused by *Phytophthora infestans*). A valued resource was lost with the virtual elimination of the American chestnut by chestnut blight (caused by *Cryphonectria parasitica*). And direct economic loss such as the estimated one billion dollars lost in one year to American corn growers from southern corn leaf blight (caused by *Cochliobolus maydis*, anamorph *Bipolaris maydis*). Many plant diseases cause less dramatic losses annually throughout the world but collectively constitute sizable losses to farmers and can reduce the aesthetic values of landscape plants and home gardens.

The goal of plant disease management is to reduce the economic and aesthetic damage caused by plant diseases. Traditionally, this has been called plant disease control, but current social and environmental values deem "control" as being absolute and the term too rigid. More multifaceted approaches to disease management, and integrated disease management, have resulted from this shift in attitude, however. Single, often severe, measures, such as pesticide applications, soil fumigation or burning are no longer in common use. Further, disease management procedures are frequently determined by disease forecasting or disease modeling rather than on either a calendar or prescription basis. Disease management might be viewed as proactive whereas disease control is reactive, although it is often difficult to distinquish between the two concepts, especially in the application of specific measures.

This topic is a general overview of some of the many methods, measures, strategies and tactics used in the control or management of plant diseases. Specific management programs for specific diseases are not intended since these will often vary depending on circumstances of the crop, its location, disease severity, regulations and other factors. Most states have some agency such as the Agricultural Extension Service or State Department of Agriculture that formulates and promulgates disease management recommendations for that state. Involvement of these agencies is especially important where the practices include some regulated component such as pesticides or quarantines. Management procedures for some specific crops and diseases can be found in the APSnet Education Center online plant disease lessons.[20,21]

Plant disease management practices rely on anticipating occurrence of disease and attacking vulnerable points in the disease cycle (i.e., weak links in the infection chain). Therefore, correct diagnosis of a disease is necessary to identify the pathogen, which is the real target of any disease management program. A thorough understanding of the disease cycle, including climatic and other environmental factors that influence the cycle, and cultural requirements of the host plant, are essential to effective management of any disease.

The many strategies, tactics and techniques used in disease management can be grouped under one or more very broad principles of action. Differences between these principles often are not clear. The simplest system consists of two principles, prevention (prophylaxis in some early writings) and therapy (treatment or cure).

The first principle (prevention) includes disease management tactics applied before infection (i.e., the plant is protected from disease), the second principle (therapy or curative action) functions with any measure applied after the plant is infected (i.e., the plant is treated for the disease). An example of the first principle is enforcement of quarantines to prevent introduction of a disease agent (pathogen) into a region where it does not occur.

The second principle is illustrated by heat or chemical treatment of vegetative material such as bulbs, corms, and woody cuttings to eliminate fungi, bacteria, nematodes or viruses that are established within the plant material. Chemotherapy is the application of chemicals to an infected or diseased plant that stops (i.e., eradicates) the infection. Although many attempts have been made to utilize chemotherapy, few have been successful. In a few diseases of ornamental or other high value trees, chemotherapy has served as a holding action that must be repeated at intervals of one to several years.[22,23] For example, antibiotics have been infused into plants to reduce severity of phytoplasma diseases of palms (lethal yellowing) and pears (pear decline) and fungicides have been injected into elms to reduce severity of Dutch elm disease (caused by *Ophiostoma ulmi*) but in all cases the chemotherapeutant must be reapplied periodically. There also are some "systemic" fungicides such as the sterol biosynthesis inhibiting (SBI) and demethylation inhibiting (DMI) fungicides that diffuse into the plant tissues to some extent and

eliminate recently established infections. One early proposal by H. H. Whetzel included four general disease control principles, exclusion, eradication, protection and immunization (the latter principle is more appropriately called resistance since plants do not have an immune system in the same sense as animals). These principles have been expanded or altered to some extent by others. They are still valid and are detailed here but students should investigate other systems such as those proposed by Gäumann, Sharvelle, or the National Academy of Science and use the one(s) that they believe are applicable.

This principle is defined as any measure that prevents the introduction of a disease-causing agent (pathogen) into a region, farm, or planting. The basic strategy assumes that most pathogens can travel only short distances without the aid of some other agent such as humans or other vector, and that natural barriers like oceans, deserts, and mountains create obstacles to their natural spread. In many cases pathogens are moved with their host plants or even on nonhost material such as soil, packing material or shipping containers. Unfortunately, exclusion measures usually only delay the entry of a pathogen, although exclusion may provide time to plan how to manage the pathogen when it ultimately arrives. Karnal bunt (caused by *Tilletia indica*) of wheat is an example of a pathogen originally from India that was anticipated. Measures were established to prevent its introduction, but it finally found its way into the United States. Soybean rust (caused by *Phakopsora pachyrhiz*i) has been found recently in the southeastern U.S. and precautions have been undertaken to prevent further spread. Due to its destructiveness, South American leaf blight (SALB) (caused by *Microcyclus ulei*) is a feared disease in the major rubber producing region of Indonesia, and contingency plans have been proposed to chemically defoliate rubber trees by aerial application of herbicides if the pathogen is detected. It is hoped that this would prevent establishment of the pathogen in the region.[25,26]

In the United States, the Animal and Plant Health Inspection Service (APHIS), a division of the U.S. Department of Agriculture, is responsible for promulgating and enforcing plant quarantine measures. There are also state agencies that deal with local quarantines. Internationally, eight regional plant protection organizations (PPOs) were established in 1951 by the International Plant Protection Convention sponsored by the Food and Agricultural Organization of the United Nations. This was revised in 1997 and now includes nine regional PPOs. The European and Mediterranean Plant Protection Organization (EPPO) is the oldest of the regional PPOs. The regional PPOs have no regulatory authority such as APHIS or other governmental agency, but function to develop strategies against the introduction and spread of pests and to coordinate the use of phytosanitary regulations to ensure agreement among the different member countries. For more information on the role of regional PPOs

An important and practical strategy for excluding pathogens is to produce pathogen-free seed or planting stock through certification programs for seeds and vegetatively propagated plant materials such as potatoes, grapes, tree fruits, etc. These programs utilize technologies that include isolation of production areas, field inspections, and removal of suspect plants to produce and maintain pathogen-free stocks. Planting stock that is freed of pathogens can be increased by tissue culture and micropropagation techniques as well as be maintained in protective enclosures such as screenhouses to exclude pathogens and their vectors. Exclusion may be accomplished by something as simple as cleaning farming equipment to remove contaminated debris and soil that can harbor pathogens such as *Verticillium*, nematodes or other soilborne organisms and prevent their introduction into non-infested fields.

Another principle aims at eliminating a pathogen after it is introduced into an area but before it has become well established or widely spread. It can be applied to individual plants, seed lots, fields or regions but generally is not effective over large geographic areas. Two large attempts at pathogen eradication in the United States were the golden nematode (*Globodera rostochiensis*) program on Long Island, New York and the citrus canker (caused by *Xanthomonas axonopodis* pv. *citri* and pv. *aurantifolii*) program in Florida. However, neither of these attempts was a lasting success.

Eradication of the golden nematode involved removing infested soil, fumigating soil in infested fields and eventually abandoning infested potato fields for housing developments and other uses. Citrus canker eradication involved widespread removal and burning of diseased trees and, in some cases, destruction of entire citrus groves and nurseries. The disease appeared to be contained and the pathogen eradicated, but the disease has reappeared and new attempts at eradication are ongoing.

Eradication can also be on a more modest scale such as the removal of apple or pear branches infected by the fire blight bacterium (*Erwinia amylovora*) or pruning to remove blister rust cankers (caused by *Cronartium ribicola*) on white pine branches. Or, it can be the sorting and removal of diseased flower

bulbs, corms or rhizomes. Hot water seed-treatment of cereal seeds to kill smut mycelium in the seed and heat treatment to eliminate viruses from fruit tree budwood for grafting are other examples of pathogen eradication.

Two programs that are actually forms of protection and not pathogen eradication are barberry eradication for reducing stem rust (caused by *Puccinia graminis*) of wheat and *Ribes* eradication for preventing white pine blister rust. The strategy is that removing these alternate hosts breaks the disease cycles and prevents infection of the economically more valuable host. These two examples are mentioned here because they are frequently cited as eradication measures. However, stem rust can readily spread from wheat to wheat in many regions by the uredinial stage although elimination of the aecial host, barberry, may deter or diminish the development of pathogenic races of the rust. The white pine blister rust fungus is perennial in the pine host and eradication of the alternate host only protects noninfected trees but does not necessarily eliminate the pathogen from the area.[27]

Eradication may also be accomplished by destroying weeds that are reservoirs of various pathogens or their insect vectors. Elimination of potato cull piles (Figure 5) is an effective method of eradicating overwintering inoculum of the late blight pathogen. Soil fumigation has been a widely used eradication strategy. This technology involves introducing gas-forming chemicals such as carbon disulfide, methyl bromide, or chloropicrin into soil to kill target pathogens. However, undesirable side effects such as killing beneficial organisms, contamination of groundwater, and toxicity of these chemicals have resulted in less reliance on this approach for disease management. Volatile fumigants like methyl bromide are injected into soil and sealed with a plastic film. Some water-soluble fumigants like metam-sodium can be injected into the soil and the soil simply compacted to form a seal

Crop rotation is a frequently used strategy to reduce the quantity of a pathogen, usually soil-borne organisms, in a cropping area. Take-all of wheat (caused by *Gaeumannomyces graminis*) and soybean cyst nematode (*Heterodera glycines*). Burning is an effective means of eradicating pathogens and is often required by law to dispose of diseased elm trees affected by Dutch elm disease (DED).

Next principle depends on establishing a barrier between the pathogen and the host plant or the susceptible part of the host plant. It is usually thought of as a chemical barrier, e.g., a fungicide, bactericide or nematicide, but it can also be a physical, spatial, or temporal barrier. The specific strategies employed assume that pathogens are present and that infection will occur without the intervention of protective measures. For example, bananas are covered with plastic sleeves as soon as the fruit are set to protect the fruit from various pests including fruit decay fungi. Protection often involves some cultural practice that modifies the environment, such as tillage, drainage, irrigation, or altering soil pH. It may also involve changing date or depth of seeding, plant spacing, pruning and thinning, or other practices that allow plants to escape infection or reduce severity of disease. Raising planting beds to assure good soil water drainage is an example of cultural management of plant diseases such as root and stem rots.

Fungicides have been used for more than a hundred years and new fungicides continue to be developed. Bordeaux mixture, a basic copper sulfate fungicide, was the first widely used fungicide and is still used today in various forms. The earliest fungicides were simple elements like sulfur or metallic compounds of copper or mercury, and these are generally classed as inorganic fungicides. In the early to mid-1900s organic fungicides such as thiram, captan, and the bisdithiocarbamates were developed. These are broad-spectrum, contact or protectant fungicides that control a wide range of fungal diseases. Starting in the 1960s the "systemic" fungicides were developed. Most of these are not truly systemic in plants but have some limited mobility, usually translaminar, and often give some post-infection benefits. Some of the "systemic" fungicides move upward in the plant's vascular system, but currently only one (fosetyl-Al) has ambimobile distribution (both upward and downward) that would constitute a truly systemic fungicide. In addition to the SBI and DMI fungicides mentioned earlier, a recent group of systemic fungicides are the strobilurins.

Some fungicides have narrow ranges of activity and are used primarily for control of specific groups of diseases such as downy mildews, rusts, smuts or powdery mildews while others are active against a wider range of diseases.

One liability of these recent narrow-range fungicides is that they often have single-site modes of action, (that is, their site-specific activity is controlled by one or a few genes), and thus are especially prone to development of fungicide resistance in the pathogen. Several management strategies have been developed to combat fungicide resistance. These include using mixtures of single-site and multi-site fungicides, alternating applications of fungicides with different modes of action, applying fungicides only when needed instead of on either a calendar or prescription basis, and applying the recommended dosage and not

attempting to cut costs by reducing the recommended amount of fungicide applied.[26,27]

Fungicides can be applied by any of several methods: ground sprayers or through irrigation systems, but to be effective applications must be done properly. First, the fungicide must be legally registered for use on the plant involved and against the target disease. Several different chemicals may be registered for the same crop or disease. If the different fungicides are similar in effectiveness, cost, ease of application, and safety, then timing of application becomes the most critical factor. If applied too early much of the chemical will be wasted before it can be effective; if applied too late, it will be largely ineffective. The benefits of properly applied fungicides can often be striking. Many cultural practices can be modified to manage the occurrence, intensity or severity of plant diseases. These include selection of suitable growing sites for the crop, adequate tillage to bury pathogen-infested plant residues, rotation to nonsusceptible crops, selecting pathogen-free planting stocks, orientation of plantings to improve exposure to sun and air currents, pruning and thinning to eliminate sources of infection and improve aeration in and around susceptible plants, water management on both plants and in soil, adequate nutrition, proper cultivation to improve root growth and avoid plant injury, and sanitation procedures to eliminate sources of inoculum.

Biological control involves the use of one living organism to control another, and this management technology has received much attention in recent times. However, the number of biological agents registered for use is relatively small, success has been limited, and application has been largely restricted to intensively managed, high value crops such as greenhouse plants. Two examples of effective biological control are the use of the fungus *Peniophora gigantea* to inoculate tree stumps to prevent infection of adjacent trees by the wood decay fungus *Heterobasidion annosum*, and the application of the nonpathogenic (i.e., non-tumor-producing) bacterium *Agrobacterium radiobacter* to fruit trees before planting to prevent infection by the crown gall bacterium (*Agrobacterium tumefaciens*)

Use of disease-resistant plants is the ideal method to manage plant diseases, if plants of satisfactory quality and adapted to the growing region with adequate levels of durable resistance are available. The use of disease-resistant plants eliminates the need for additional efforts to reduce disease losses unless other diseases are additionally present. Resistant plants are usually derived by standard breeding procedures of selection and/or hybridization. A few disease-resistant lines have been obtained by inducing mutations with x-rays or chemicals. There is also interest in chemicals called “plant activators” that induce plant defense responses called systemic acquired resistance (SAR) and induced resistance. Recently, resistant plants have been developed through the use of genetic engineering (e.g., resistance to the *Papaya ringspot virus*).

Selection of resistant plants involves subjecting plants to high levels of disease pressure (Figure 18) and using the surviving plants as sources of disease resistance. Plants that survive this pressure often have genetic resistance that can be utilized directly by propagation or as sources of resistance to develop resistant plants that also have the requisite qualities for that crop. Hybridization is a tactic where a plant having the desired agronomic or horticultural qualities, but is susceptible to a disease, is crossed with a plant that is resistant but which may or may not have the other desirable characteristics such as size, yield, flavor, aesthetics, etc.

Disease escape occurs when susceptible plants do not become diseased for some reason. This may be due to some anatomical or physical character, such as the occurrence of leaf hairs, thick cuticle, or modified stomata, or they may be environmental, in which conditions are not conducive to disease development. Although disease escape based on some anatomical feature is useful occasionally, escape more often complicates the process of developing disease resistant plants.[25,26]

Development of disease-resistant plants has been relatively successful with annual and biennial plants, but less so with perennials, primarily because of the longer time required to develop and test the progeny. Woody perennials, such as ornamental, forest, and orchard trees, have been especially difficult for plant breeders to develop useful disease resistance. For example, chestnut blight and Dutch elm disease have devastated two valued native trees. In both cases there have been extensive attempts to develop resistant trees, usually by creating hybrids with exotic chestnut or elm trees, and some resistant selections have resulted. Unfortunately, these generally lack the desirable qualities, such as nut flavor or tree forms characteristic of the native trees. Another introduced disease that has impacted native trees is white pine blister rust. There has been an intense effort for more than 50 years to select and improve rust-resistant pines from the surviving population. These trees are now being planted for reforestation, but it will be another 50 or so years, when these trees have matured to produce a timber crop, before the success of this program is known.

Development of resistance has been most successful against the more specialized pathogens such as rust fungi , smut fungi, powdery mildew fungi, and viruses, but less so against general pathogens such as many blight, canker, root rot and leaf spotting pathogens. A major problem with genetically resistant plants is that host-differentiated pathogenic races can be selected, so that many breeding programs become continuous processes to develop disease resistant plant lines. Disease resistance conferred by a single major gene is sometimes called specific or qualitative resistance and is race-specific. This type of resistance is often unstable, and emergence of a pathogenic race that can attack that genotype can completely overcome this type of resistance. Quantitative resistance or general resistance derives from many different genes for resistance with additive effects to provide more stable (or durable) resistance to pathogens.

There are several strategies to minimize this race development and resistance failure. These include methods of gene deployment, where different genetic plant types are interspersed on a regional basis to avoid a genetic monoculture, or planting mixtures of cultivars having different genetic compositions to ensure that some component of the crop will be resistant to the disease.

A recent and controversial technique in developing disease resistant plants is the insertion of genes from other organisms into plants to impart some characteristic. For example, genes from the bacterium *Bacillus thuringiensis* have been inserted into plants to protect against insect attacks. Plants with these inserted genes are called genetically-modified organisms (GMOs), and have caused concern that unanticipated, and perhaps detrimental, characteristics, such as unforeseen allergens, may also be transferred to the new plants. However, unforeseen and undesirable qualities also can be transmitted by conventional plant breeding techniques. The potato cultivar Lenape was developed in part because of its resistance to *Potato virus A* and resistance to late blight tuber infection. After it was released it was discovered that the tubers contained very high levels of solanine, a toxic alkaloid. The wheat cultivar Paha had resistance to stripe rust (caused by *Puccinia striiformis*) but also was very susceptible to flag smut (caused by *Urocystis agropyri*). Both of these plant cultivars, developed by conventional breeding methods, were quickly taken out of production. There is much interest in the genetic engineering of disease-resistant plants and some success has been obtained with several virus diseases, the best known of which is papaya ringspot. This approach to plant disease management will likely expand, especially for widely grown crops such as wheat, corn, soybeans, rice, and the like, as social, legal, and economic obstacles are overcome.[27]

Integrated Disease Management (IDM) is a concept derived from the successful Integrated Pest Management (IPM) systems developed by entomologists for insect and mite control. In most cases IDM consists of scouting with timely application of a combination of strategies and tactics. These may include site selection and preparation, utilizing resistant cultivars, altering planting practices, modifying the environment by drainage, irrigation, pruning, thinning, shading, etc., and applying pesticides, if necessary. But in addition to these traditional measures, monitoring environmental factors (temperature, moisture, soil pH, nutrients, etc.), disease forecasting, and establishing economic thresholds are important to the management scheme. These measures should be applied in a coordinated integrated and harmonized manner to maximize the benefits of each component. For example, balancing fertilizer applications with irrigation practices helps promote healthy vigorous plants. However, this is not always easy to accomplish, and "disease management" may be reduced to single measures exactly the same as the ones previously called "disease control." Whatever the measures used, they must be compatible with the cultural practices essential for the crop being managed.

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