

## 1: Systemic acquired resistance - Wikipedia

*Plant and Animal Pathogens With respect to farm animals, only a small number of viral diseases are capable of inflicting major economic damage. Examples include foot-and-mouth disease (FMD) in cattle and pigs, classical swine fever and African swine fever in pigs, and avian influenza and Newcastle disease in poultry.*

Bacteria as plant pathogens. The Plant Health Instructor. Vidaver and Patricia A. Plant associated bacteria may be beneficial or detrimental. All plant surfaces have microbes on them termed epiphytes, and some microbes live inside plants termed endophytes. Some are residents and some are transient. Bacteria are among the microbes that successively colonize plants as they mature. Individual bacterial cells cannot be seen without the use of a microscope, however, large populations of bacteria become visible as aggregates in liquid, as biofilms on plants, as viscous suspensions plugging plant vessels, or colonies on petri dishes in the laboratory. Figure 1 Plant pathogenic bacteria cause many serious diseases of plants throughout the world Vidhyasekaran; Figure 2, but fewer than fungi or viruses, and they cause relatively less damage and economic cost Kennedy and Alcorn Most plants, both economic and wild, have innate immunity or resistance to many pathogens. However, many plants can harbor plant pathogens without symptom development asymptomatic. Figure 2 return to top Frontiers: Current and Future Areas of Inquiry The most exciting and current areas of research on plant-associated bacteria are the result of new intellectual discoveries, analyses and fields of study, new techniques and new instrumentation unavailable even a decade ago. For example, genomic sequencing, or the ordered reading of thousands of nucleotides constituting the deoxyribonucleic acid DNA of an organism, is now relatively common. Along with sequencing and its enormous data accumulation has arisen the field of bioinformatics to enable communication among scientists and analyses of the data, particularly comparative and evolutionary studies. Even supposedly simple steps like annotation of a gene, or its name or function, remains a challenge. For maximal usefulness, such as unequivocal identification of an organism and determination of the number of its virulence genes and their locations, a fully annotated genome sequence is needed Fraser et al. Analyzing the expression of DNA through intermediate steps with microarrays is a powerful emerging tool Hinds et al. Similarly, methods for characterizing the entire protein complement of an organism proteomics are becoming available Graves and Hayward These new and evolving techniques are enabling the study of virulence the disease severity and pathogenicity the ability to cause disease, strain descendants of a single isolation in culture identification and typing similarity or difference analysis relative to other strains, evolution and spread of bacteria, gene expression and gene regulation. These discoveries are occurring both with natural bacterial variants and mutants constructed in the laboratory. The expectation is to exploit these findings for improved disease management. Mechanisms of pathogenicity of bacterial plant pathogens are becoming well known Ahlemeyer and Eichenlaub, Burger and Eichenlaub Virulence and pathogenicity genes may be harbored in different replicons independent replicating units, such as spread throughout the chromosome or in specialized areas termed genomic or pathogenicity islands Arnold et al. Population development must normally occur for many bacteria to survive and infect plants. Infectious doses normally are in the millions of cells. In several cases, and perhaps all, the cells communicate chemically with one another quorum sensing and with other species. These chemical sensing molecules are under intensive study Federle and Bassler In some cases, and perhaps most, microorganisms organize in dense growths to form biofilms that tightly adhere to surfaces, serving as protectants against the elements and enabling cells to produce a favorable environment for survival and spread. Some structures used by bacteria to insert chemical compounds into plant cells are well studied, such as the so-called Type III secretion system five types are currently known. The Type III system operates somewhat like a syringe and plunger to transport pathogen-produced proteins that effect disease or trigger defense Pociano et al. These mechanisms have sometimes shown surprising and unexpected similarity to those found in animal and human pathogens Cao et al. There are even a few strains of bacteria that cross kingdoms: The genetic basis for such novelty is of immense interest and significance regarding the basis of infectious disease. Many challenges remain in transformation of certain plant varieties and species, as well as predictable and stable expression of transgenes

Challenges and opportunities for the future in plant microbiology abound. The best is yet to come. For example, one of the current challenges is providing healthy plants for humans during long-term space travel and exploration. On the plant side, many avenues are being explored. Understanding and manipulating resistance in host plants is extremely important. Host resistance may be due to one or several resistance genes or R genes to specific pathogens harboring virulence genes. If the virulence genes trigger a host defense response, they are termed avirulence avr genes. If the resistance is more general, a variety of preformed defense mechanisms, both structural and chemical, may be involved with induced chemicals as well local or systemic acquired resistance. Studies of pathogen interactions in model systems, particularly *Arabidopsis thaliana*, are enabling clearer understanding of susceptibility and resistance applicable to more complex plants. Sequencing of major plant genomes is underway as well, with rice being completed. Multiple alleles and chromosomes, as well as complex traits are challenges in understanding and managing host resistance. Compiling information from sequencing and functional analysis of both pathogens and major crop plants is expected to bring new insights useful for sustained disease management. The forgoing depends on a basic knowledge of these bacteria, which follows.

History Individual bacteria were first seen by humans about years ago when they were magnified by the first microscope. The disease caused by *Erwinia amylovora*, now widespread throughout much of the temperate world, remains a limiting factor in growth of healthy apple and pear trees (Figure 4). Arthur was able to isolate a bacterium from diseased plants, culture it, and then inoculate the same host to reproduce a naturally occurring disease. (Figure 4) Bacteria as plant pathogens can cause severe economically damaging diseases, ranging from spots, mosaic patterns or pustules on leaves (Figure 5) and fruits, or smelly tuber rots to plant death. Some cause hormone-based distortion of leaves and shoots called fasciation (Figure 6), or crown gall, a proliferation of plant cells producing a swelling at the intersection of stem and soil (Figure 7) and on roots.

## 2: Category:Plant pathogens and diseases - Wikipedia

*Viral diseases in plants-the causal organism and the disease. Bacterial diseases in plants - the causal organism and the disease.*

View Larger Image Pathogen induced diseases Pathogen in a broad sense is anything that can infect and can cause a disease. It is a type of infectious agent that can be a virus, bacteria, prion, fungus, viroid or parasite that causes diseases in the host and the host can be a fungus, a plant, an animal or any microorganism also. Soon after the recognition and entry into the host cells, the damaged cell of the host disintegrates; the virus-lacking envelope is released rapidly. Viruses with an envelope are released slowly without immediate disintegration of the host cell. Apart from being released slowly or rapidly there is a possibility where viruses become long term residents of the host. Herpes simplex virus that follows such possibility and causes fever and blisters. In animal Herpes virus infects and damages the epithelial tissues; it then spreads to the nerve cells and become inactive. During this period, it resides in nerves near the skin. They get reactivated by the emotional or physical stress, chemical or microbial infections. They damage the epithelial cells of the skin upon reactivation. Retrovirus One defining characteristic of retrovirus is the use of viral reverse transcriptase. Retroviruses carry this enzyme in their genome and use it and convert its single stranded RNA into a double stranded DNA. A key feature of the retrovirus reverse transcriptase is the lack of a proofreading ability, an ability that is found in cellular polymerase enzymes. Errors in this transcription process result in slightly different genetic codes or mutants. Adenovirus Once the virus has successfully gained entry into the host cell, the endosome acidifies, which alters the virus topology by causing capsid components to disassociate. These changes as well as the toxic nature of the pentons of their structure results in the release of virion into the cytoplasm. With the help of cellular microtubules, the virus is transported to the nuclear pore complex, whereby the adenovirus particle disassembles. Viral DNA is then subsequently released, which can enter the nucleus via the nuclear pore. The DNA then associates with histone molecules. Thus, viral gene expression can occur and new virus particles can be generated. DNA replication process divides adenovirus life cycle into two phases: In both phases, a primary transcript, i. There are four stages to the adenovirus replication cycle First the cell binds to surface receptors on the target cell and enter the cell via endocytosis. The virion then sheds its endosome coat and enters into the nucleus. In the nucleus the virion releases its genes, which are then read by the machinery inside of the host cell. The adenovirus has both early and late genes. The early genes code for non-structural, regulatory proteins, while the late genes code for replication substrates and machinery. After transcription, these genes are translated, the new progeny are assembled and then they exit through cell lysis. Responses Elicited by Disease Cell wall reinforcement callose, lignin, suberin, cell wall proteins. Antimicrobial chemicals and proteins. Endophyte assistance from plant roots: The viral RNA is infectious by itself, but the addition of a protein coat protects the RNA from enzymes that would destroy it. The protein coat poses a problem, that it must be removed once the virus gets inside a cell. As with many viruses, TMV has chemicals that cause the protein to change when the environment changes. The capsid protein has several clusters of acidic amino acids that are stable outside of cells, where calcium levels are high, but repel each other in the low-calcium conditions inside the cell. This is to loosen the first few capsid proteins, releasing the end of the RNA. TMV then uses ribosomes as the supporter to finish the process. As the ribosomes move down the strand, creating the first set of virus proteins, they displace the remaining capsid proteins. If the RNA and capsid proteins are mixed together, they spontaneously form functional viruses. A two-step process is used for their assembly. First, the TMV protein forms a two-layer disk, with 17 proteins in each ring. A special initiation sequence in the RNA then binds in the hole at the center. The remaining subunits then stack on this structure, elongating until the RNA is covered. There is a great advantage to this two-step process: Diseases caused by pathogens Trypanosoma gambiense are the causative organism for the African sleeping sickness. Different species of the trypanosoma family namely T. Gambiense reside in blood, lymph and cerebrospinal fluid. Reservoir host- Antelopes, Pigs and Buffaloes. Parasite reproduces by longitudinal binary fission. They respire anaerobic and excrete by diffusion. There are three forms namely stumpy, intermediate

and slender. Pathogenicity During first stage, parasite resides in blood but no significant symptoms appear. After an incubation period of days, the parasite enters the lymph nodes and multiplies in cells of lymph glands and causes swelling. There is an increase in number of WBCs and enlargement of liver and spleen. The symptoms include nausea, headache, unconsciousness and irregular fever. Morphology of the malaria parasite *Plasmodium falciparum* inside the infected erythrocyte Early trophozoite. Mature schizont with merozoites and clumped pigment. Macrogametocyte with bluish cytoplasm and compact chromatin. Microgametocyte with pinkish cytoplasm and dispersed chromatin. Pertussis It is also known as whooping cough, a highly contagious disease caused by the bacterium *Bordetella pertussis*. Symptoms After two-day incubation period, pertussis in infants and young children are characterized initially by mild respiratory infection symptoms such as coughing, sneezing and runny nose catarrhal stage. Coughing fits may be followed by vomiting due to the sheer violence of the fit. In severe cases, the vomiting induced by coughing fits can lead to malnutrition and dehydration. The fits that do not occur on their own can also be triggered by yawning, stretching, laughing or yelling. Triggering fits gradually diminish over one to two months during the convalescent stage. Other complications of the disease include pneumonia, encephalitis, pulmonary hypertension and secondary bacterial super-infection. Transmission Adults and adolescents are the primary reservoirs for pertussis. It is spread by contact with airborne discharges from the mucous membranes of infected people, who are most contagious during the catarrhal stage. Because the symptom during the catarrhal stage is non-specific, pertussis is usually not diagnosed until the appearance of the characteristic cough of the paroxysmal stage.

## 3: Pathogen - Wikipedia

*P. Gladieux, T. Giraud, in Genetics and Evolution of Infectious Diseases (Second Edition), Comparing Animal and Plant Pathogens. Pathogenic fungi are mostly intracellular pathogens, indicating that at some point during the interaction between the host and the invading species the pathogen lives inside the host cell.*

Resources Like human beings and other animals, plants are subject to diseases. There are many branches of science that participate in the control of plant diseases. Among them are biochemistry, bio-technology, soil science, genetics and plant breeding, meteorology, mycology fungi, nematology nematodes, virology viruses, and weed science. Chemistry, physics, and statistics also play a role in the scientific maintenance of plant health. The study of plant diseases is called plant pathology. The most common diseases of cultivated plants are bacterial wilt, chestnut blight, potato late blight, rice blast, coffee rust, stem rust, downy mildew, ergot, root knot, and tobacco mosaic. This is a small list of the more than 50, diseases that attack plants. Diseases can be categorized as annihilating, devastating, limiting, or debilitating. As the term suggests, annihilating diseases can totally wipe out a crop, whereas a devastating plant disease may be severe for a time and then subside. Debilitating diseases weaken crops when they attack them successively over time and limiting diseases reduce the viability of growing the target crop, thereby reducing its economic value. Plant diseases are identified by both common and scientific names. The scientific name identifies both the genus and the species of the disease-causing agent. For the past 50 years, the ability to combat plant diseases through the use of modern farm management methods, fertilization of crops, irrigation techniques, and pest control have made it possible for the United States to produce enough food to feed its population and to have surpluses for export. However, the use of pesticides, fungicides, herbicides, fertilizers and other chemicals to control plant diseases and increase crop yields also poses significant environmental risks. Air, water, and soil can become saturated with chemicals that can be harmful to human and ecosystem health.

History of plant pathology While early civilizations were well aware that plants were attacked by diseases, it was not until the invention of the first microscope that people began to understand the real causes of these diseases. There are references in the Bible to blights, blasts, and mildews. Aristotle wrote about plant diseases in BC and Theophrastus BC theorized about cereal and other plant diseases. During the Middle Ages in Europe, ergot fungus infected grain and Shakespeare mentions wheat mildew in one of his plays. After Anton von Leeuwenhoek constructed a microscope in 1673, he was able to view organisms, including protozoa and bacteria, not visible to the naked eye. In the eighteenth century, Duhumel de Monceau described a fungus disease and demonstrated that it could be passed from plant to plant, but his discovery was largely ignored. About this same time, nematodes were described by several English scientists and by the treatment of seeds to prevent a wheat disease was known. In the nineteenth century, Ireland suffered a devastating potato famine due to a fungus that caused late blight of potatoes. At this time, scientists began to take a closer look at plant diseases. Heinrich Anton DeBary, known as the father of modern plant pathology, published a book identifying fungi as the cause of a variety of plant diseases. Until this time, it was commonly believed that plant diseases arose spontaneously from decay and that the fungi were caused by this spontaneously generated disease. DeBary supplanted this theory of spontaneously generated diseases with the germ theory of disease. Petri, Pierre Millardet, Erwin F. Smith, Adolph Mayer, Dimitri Ivanovski, Martinus Beijerinck, and Hatsuzo Hashimoto, made important discoveries about specific diseases that attacked targeted crops. During the twentieth century advances were made in the study of nematodes. Stanley was awarded a Nobel Prize for his work with the tobacco mosaic virus. By 1935, virus particles could be seen under the new electron microscope. In the 1940s fungicides were developed and in the 1950s nematicides were produced. In the 1960s Japanese scientist Y. Doi discovered mycoplasmas, organisms that resemble bacteria but lack a rigid cell wall, and in 1971, T. Diener discovered viroids, organisms smaller than viruses.

Causes of plant disease Plant diseases can be infectious transmitted from plant to plant or noninfectious. Noninfectious diseases are usually referred to as disorders. Common plant disorders are caused by deficiencies in plant nutrients, by waterlogged or polluted soil, and by polluted air. Too little or too much water or improper nutrition can cause plants to grow poorly. Plants can also be stressed by weather that is too

hot or too cold, by too little or too much light, and by heavy winds. Pollution from automobiles and industry, and the excessive application of herbicides for weed control can also cause noninfectious plant disorders. Infectious plant diseases are caused by pathogens, living microorganisms that infect a plant and deprive it of nutrients. Bacteria, fungi, nematodes, mycoplasmas, viruses and viroids are the living agents that cause plant diseases. Nematodes are the largest of these agents, while viruses and viroids are the smallest. None of these pathogens are visible to the naked eye, but the diseases they cause can be detected by the symptoms of wilting, yellowing, stunting, and abnormal growth patterns.

**Bacteria** Some plant diseases are caused by rod-shaped bacteria. The bacteria enter the plant through natural openings, like the stomata of the leaves, or through wounds in the plant tissue. Other common symptoms of bacterial disease include rotting and swollen plant tissues. Bacteria can be spread by water, insects, infected soil, or contaminated tools. Bacterial wilt attacks many vegetables including corn and tomatoes, and flowers. Crown gall, another bacterial plant disease, weakens and stunts plants in the rose family and other flowers. Fireblight attacks apple, pear, and many other ornamental and shade trees. Fungi can grow on living or dead plant tissue and can survive in a dormant stage until conditions become favorable for their proliferation. Fungal spores, which act like seeds, are spread by wind, water, soil, and animals to other plants. Warm, humid conditions promote fungal growth. Many fungi can attack a variety of plants, but some are specific to particular plants. The list of fungi and the plants they infect is a long one. Black spot attacks roses, while brown rot damages stone fruits. Damping off is harmful to seeds and young plants. Downy mildew attacks flowers, some fruits, and most vegetables. Gray mold begins on plant debris and then moves on to attack flowers, fruits, and vegetables. Oak root fungus and oak wilt are particularly damaging to oaks and fruit trees. Peach leaf curl targets peaches and nectarines. Powdery mildew, rust, sooty mold, and southern blight attack a wide variety of plants, including grasses. Texas root rot and water mold root rot can also infect many different plants. Verticillium wilt targets tomatoes, potatoes, and strawberries.

**Viruses and viroids** The viruses and viroids that attack plants are the hardest pathogens to control. Destroying the infected plants is usually the best control method, since chemicals to inactivate plant viruses and viroids have not proven effective. While more than plant viruses have been identified, new strains continually appear because these organisms are capable of mutating. The symptoms of viral infection include yellowing, stunted growth in some part of the plant, and plant malformations like leaf rolls and uncharacteristically narrow leaf growth. The mosaic viruses can infect many plants. Plants infected with this virus have mottled or streaked leaves; infected fruit trees produce poor fruit and a small yield.

**Nematodes** Nematodes are tiny microscopic animals with wormlike bodies and long, needlelike structures called stylets that suck nutrients from plant cells. They lay eggs that hatch as larvae and go through four stages before becoming adults. Nematodes have a day life cycle, but they can remain in a dormant state for more than 30 years. Nematicides are chemicals used to control nematode infestations. Marigolds are resistant to nematodes and are often planted to help eliminate them from infected soil. Nematodes primarily attack plant roots, but they may also destroy other parts of the plant either internally or externally. They thrive in warm, sandy, moist soil and attack a variety of plants including corn, lettuce, potatoes, tomatoes, alfalfa, rye, and onions. However, all nematodes are not harmful to plants. Some are actually used to control other plant pests such as cutworms, armyworms, and beetle grubs.

**Other causes of plant diseases** Mycoplasmas are single-celled organisms that lack rigid cell walls and are contained within layered cell membranes. They are responsible for the group of plant diseases called yellow diseases and are spread by insects such as the leafhopper. Parasitic plants, such as mistletoe, cannot get their nutrients from the soil, but must attach themselves to other plants and use nutrients from the host plant to survive. They weaken the wood of their host trees and deform the branches.

**Disease cycles** An equilateral disease triangle is often used to illustrate the conditions required for plant diseases to occur. The base of the triangle is the host and the two equal sides represent the environment and the pathogen. When all three factors combine, then disease can occur. Pathogens need plants in order to grow because they cannot produce their own nutrients. When a plant is vulnerable to a pathogen and the environmental conditions are right, the pathogen can infect the plant causing it to become diseased. The key to success in growing plants, whether in the home garden or commercially, is to change one or more of the three factors necessary to produce disease. Disease-resistant plants and enrichment of soil nutrients are two ways of

altering the disease triangle. Weather is one environmental factor in the plant disease triangle that is impossible to control. When weather conditions favor the pathogen and the plant is susceptible to the pathogen, disease can occur. Weather forecasting provides some help; satellites monitor weather patterns and provide farmers with some advance warning when conditions favorable to disease development are likely to occur. Battery-powered microcomputers and microenvironmental monitors are placed in orchards or fields to monitor temperature, rainfall, light levels, wind, and humidity. These monitors provide farmers with information that helps them determine the measures they need to take to reduce crop loss due to disease. Control Control of plant disease begins with good soil management.

*When these induced responses are triggered rapidly and coordinately during a given plant-pathogen interaction, the plant is resistant to disease. A susceptible plant responds more slowly with an onset of defense mechanisms after infection.*

General considerations Nature and importance of plant diseases Plant diseases are known from times preceding the earliest writings. Fossil evidence indicates that plants were affected by disease million years ago. The Bible and other early writings mention diseases, such as rusts, mildews, and blights, that have caused famine and other drastic changes in the economy of nations since the dawn of recorded history. Other plant disease outbreaks with similar far-reaching effects in more recent times include late blight of potato in Ireland 1845-1850; powdery and downy mildews of grape in France and Italy; coffee rust in Ceylon now Sri Lanka; starting in the 1860s; Fusarium wilts of cotton and flax; southern bacterial wilt of tobacco early 1900s; Sigatoka leaf spot and Panama disease of banana in Central America 1860s-1870s; black stem rust of wheat, 1840s-1850s; southern corn leaf blight in the United States; Panama disease of banana in Asia, Australia, and Africa to present; and coffee rust in Central and South America, to present. Such losses from plant diseases can have a significant economic impact, causing a reduction in income for crop producers and distributors and higher prices for consumers. Loss of crops from plant diseases may also result in hunger and starvation, especially in less-developed countries where access to disease-control methods is limited and annual losses of 30 to 50 percent are not uncommon for major crops. In some years, losses are much greater, producing catastrophic results for those who depend on the crop for food. Major disease outbreaks among food crops have led to famines and mass migrations throughout history. The devastating outbreak of late blight of potato caused by the water mold *Phytophthora infestans* that began in Europe in 1845 brought about the Great Famine that caused starvation, death, and mass migration of the Irish. This water mold thus had a tremendous influence on economic, political, and cultural development in Europe and the United States. During World War I, late blight damage to the potato crop in Germany may have helped end the war. Diseases are a normal part of nature Plant diseases are a normal part of nature and one of many ecological factors that help keep the hundreds of thousands of living plants and animals in balance with one another. Plant cells contain special signaling pathways that enhance their defenses against insects, animals, and pathogens. One such example involves a plant hormone called jasmonate (jasmonic acid). In the absence of harmful stimuli, jasmonate binds to special proteins, called JAZ proteins, to regulate plant growth, pollen production, and other processes. In the presence of harmful stimuli, however, jasmonate switches its signaling pathways, shifting instead to directing processes involved in boosting plant defense. Genes that produce jasmonate and JAZ proteins represent potential targets for genetic engineering to produce plant varieties with increased resistance to disease. Humans have carefully selected and cultivated plants for food, medicine, clothing, shelter, fibre, and beauty for thousands of years. Disease is just one of many hazards that must be considered when plants are taken out of their natural environment and grown in pure stands under what are often abnormal conditions. Many valuable crop and ornamental plants are very susceptible to disease and would have difficulty surviving in nature without human intervention. Cultivated plants are often more susceptible to disease than are their wild relatives. This is because large numbers of the same species or variety, having a uniform genetic background, are grown close together, sometimes over many thousands of square kilometres. A pathogen may spread rapidly under these conditions. Plant diseases can be broadly classified according to the nature of their primary causal agent, either infectious or noninfectious. Infectious plant diseases are caused by a pathogenic organism such as a fungus, bacterium, mycoplasma, virus, viroid, nematode, or parasitic flowering plant. An infectious agent is capable of reproducing within or on its host and spreading from one susceptible host to another. Noninfectious plant diseases are caused by unfavourable growing conditions, including extremes of temperature, disadvantageous relationships between moisture and oxygen, toxic substances in the soil or atmosphere, and an excess or deficiency of an essential mineral. Because noninfectious causal agents are not organisms capable of reproducing within a host, they are not transmissible. In nature, plants may be affected

by more than one disease-causing agent at a time. A plant that must contend with a nutrient deficiency or an imbalance between soil moisture and oxygen is often more susceptible to infection by a pathogen, and a plant infected by one pathogen is often prone to invasion by secondary pathogens. The combination of all disease-causing agents that affect a plant make up the disease complex. Knowledge of normal growth habits, varietal characteristics, and normal variability of plants within a species—as these relate to the conditions under which the plants are growing—is required for a disease to be recognized. The study of plant diseases is called plant pathology. Pathology is derived from the two Greek words pathos suffering, disease and logos discourse, study. Plant pathology thus means a study of plant diseases. Disease development and transmission

**Pathogenesis and saprogenesis** Pathogenesis is the stage of disease in which the pathogen is in intimate association with living host tissue. Three fairly distinct stages are involved: One of the important characteristics of pathogenic organisms, in terms of their ability to infect, is virulence. Many different properties of a pathogen contribute to its ability to spread through and to destroy the tissue. Among these virulence factors are toxins that kill cells, enzymes that destroy cell walls, extracellular polysaccharides that block the passage of fluid through the plant system, and substances that interfere with normal cell growth. Not all pathogenic species are equal in virulence—that is, they do not produce the same amounts of the substances that contribute to the invasion and destruction of plant tissue. Also, not all virulence factors are operative in a particular disease. For example, toxins that kill cells are important in necrotic diseases, and enzymes that destroy cell walls play a significant role in soft rot diseases. Many pathogens, especially among the bacteria and fungi, spend part of their life cycle as pathogens and the remainder as saprotrophs. During this stage, some fungi produce their sexual fruiting bodies; the apple scab *Venturia inaequalis*, for example, produces perithecia, flask-shaped spore-producing structures, in fallen apple leaves. Other fungi produce compact resting bodies, such as the sclerotia formed by certain root- and stem-rotting fungi *Rhizoctonia solani* and *Sclerotinia sclerotiorum* or the ergot fungus *Claviceps purpurea*. These resting bodies, which are resistant to extremes in temperature and moisture, enable the pathogen to survive for months or years in soil and plant debris in the absence of a living host. Page 1 of

*REVIEWS Fig. 1. The pathogen-induced and transgenically triggered hypersensitive response (HR) in plants. (a) Shows virus-induced HR lesions.*

Freeman and Gwyn A. The Plant Health Instructor. Although lacking an immune system comparable to animals, plants have developed a stunning array of structural, chemical, and protein-based defenses designed to detect invading organisms and stop them before they are able to cause extensive damage. Humans depend almost exclusively on plants for food, and plants provide many important non-food products including wood, dyes, textiles, medicines, cosmetics, soaps, rubber, plastics, inks, and industrial chemicals. Understanding how plants defend themselves from pathogens and herbivores is essential in order to protect our food supply and develop highly disease-resistant plant species. This article introduces the concept of plant disease and provides an overview of some defense mechanisms common among higher plants. A close examination of plant anatomy is presented, as well as some of the ecological relationships that contribute to plant defense and disease resistance. Special care has been taken to illustrate how products used in everyday life are derived from substances produced by plants during defense responses. Disease can be caused by living biotic agents, including fungi and bacteria, or by environmental abiotic factors such as nutrient deficiency, drought, lack of oxygen, excessive temperature, ultraviolet radiation, or pollution. In order to protect themselves from damage, plants have developed a wide variety of constitutive and inducible defenses. Constitutive continuous defenses include many preformed barriers such as cell walls, waxy epidermal cuticles, and bark. These substances not only protect the plant from invasion, they also give the plant strength and rigidity. In addition to preformed barriers, virtually all living plant cells have the ability to detect invading pathogens and respond with inducible defenses including the production of toxic chemicals, pathogen-degrading enzymes, and deliberate cell suicide. Plants often wait until pathogens are detected before producing toxic chemicals or defense-related proteins because of the high energy costs and nutrient requirements associated with their production and maintenance. In a similar way, many pathogens establish intimate connections with their hosts in order to suppress plant defenses and promote the release of nutrients. Pathogens that keep their host alive and feed on living plant tissue are called biotrophs. Examples of biotrophic pathogens include the powdery mildew fungus *Blumeria graminis* and the bacterial rice pathogen *Xanthomonas oryzae*. Other pathogens resort to brute force like thieves who blast open a bank vault with explosives. These pathogens often produce toxins or tissue-degrading enzymes that overwhelm plant defenses and promote the quick release of nutrients. These pathogens are called necrotrophs, and examples include the gray mold fungus *Botrytis cinerea* and the bacterial soft-rot pathogen *Erwinia carotovora*. Some pathogens are biotrophic during the early stages of infection but become necrotrophic during the latter stages of disease. These pathogens are called hemibiotrophs and include the fungus *Magnaporthe grisea*, the causative agent of rice blast disease. Powdery mildew on a maple leaf Figure 2. Rice blast disease on rice leaves Figure 3. Blackleg soft-rot on a potato tuber Most biotrophic and hemibiotrophic pathogens can only cause disease on a relatively small group of host plants because of the slightly different set of specialized genes and molecular mechanisms required for each host-pathogen interaction. The host range refers to the plant species on which a pathogen is capable of causing disease. For example, bromo mosaic virus BMV infects grasses such as barley but not legumes. A plant species that does not show disease when infected with a pathogen is referred to as a non-host plant species for that pathogen. Organisms that do not cause disease on any plant species, such as the saprophytic bacterial species *Pseudomonas putida*, are referred to as non-pathogens. When a pathogen is capable of causing disease on a particular host species, two outcomes are possible: A compatible response is an interaction that results in disease, while an incompatible response is an interaction that results in little or no disease at all. Although a particular plant species may be a susceptible host for a particular pathogen, some individuals may harbor genes that help recognize the presence of the pathogen and activate defenses. For example, some tomato cultivars show disease when infected with the bacterial pathogen *Pseudomonas syringae* a compatible response, but others cultivar Rio Grande, for example are capable of recognizing the bacteria and limiting

disease via resistance an incompatible response. Disease resistance exists as a continuum of responses ranging from immunity the complete lack of any disease symptoms to highly resistant some disease symptoms to highly susceptible significant disease symptoms. Tomato leaves exposed to the bacterial pathogen *P*. The leaf on the left is diseased, and the leaf on the right is resistant. Surveillance and Detection of Microbial Pathogens Figure 5. Bacterial flagella are often recognized by plants during basal resistance. Plants have developed multiple layers of sophisticated surveillance mechanisms that recognize potentially dangerous pathogens and rapidly respond before those organisms have a chance to cause serious damage. These surveillance systems are linked to specific pre-programmed defense responses. Basal resistance, also called innate immunity, is the first line of pre-formed and inducible defenses that protect plants against entire groups of pathogens. Basal resistance can be triggered when plant cells recognize microbe-associated molecular patterns MAMPs including specific proteins, lipopolysaccharides, and cell wall components commonly found in microbes. The result is that living plant cells become fortified against attack. Non-pathogens as well as pathogens are capable of triggering basal resistance in plants due to the widespread presence of these molecular components in their cells. Pathogens have developed countermeasures that are able to suppress basal resistance in certain plant species. If a pathogen is capable of suppressing basal defense, plants may respond with another line of defense: The HR is characterized by deliberate plant cell suicide at the site of infection. Although drastic compared to basal resistance, the HR may limit pathogen access to water and nutrients by sacrificing a few cells in order to save the rest of the plant. The HR is typically more pathogen-specific than basal resistance and is often triggered when gene products in the plant cell recognize the presence of specific disease-causing effector molecules introduced into the host by the pathogen. Bacteria, fungi, viruses, and microscopic worms called nematodes are capable of inducing the HR in plants. HR lesion on an Arabidopsis leaf. Once the hypersensitive response has been triggered, plant tissues may become highly resistant to a broad range of pathogens for an extended period of time. This phenomenon is called systemic acquired resistance SAR and represents a heightened state of readiness in which plant resources are mobilized in case of further attack. Researchers have learned to artificially trigger SAR by spraying plants with chemicals called plant activators. These substances are gaining favor in the agricultural community because they are much less toxic to humans and wildlife than fungicides or antibiotics, and their protective effects can last much longer. In addition to the hypersensitive response, plants can defend themselves against viruses by a variety of mechanisms including a sophisticated genetic defense system called RNA silencing. Plants can recognize these foreign molecules and respond by digesting the genetic strands into useless fragments and halting the infection. Plants that are infected with viruses will often exhibit chlorosis and mottling, but disease symptoms may eventually disappear if RNA silencing is successful, a process called recovery. In addition, the plant may retain a template of the digested genetic strand that can be used to quickly respond to future attack by similar viruses, a process analogous to the memory of vertebrate immune systems. Plants can distinguish between general wounding and insect feeding by the presence of elicitors contained in the saliva of chewing insects. In response, plants may release volatile organic compounds VOCs , including monoterpenoids, sesquiterpenoids, and homoterpenoids. These chemicals may repel harmful insects or attract beneficial predators that prey on the destructive pests. For example, wheat seedlings infested with aphids may produce VOCs that repel other aphids. Lima beans and apple trees emit chemicals that attract predatory mites when damaged by spider mites, and cotton plants produce volatiles that attract predatory wasps when damaged by moth larvae. Feeding on one part of the plant can induce systemic production of these chemicals in undamaged plant tissues, and once released, these chemicals can act as signals to neighboring plants to begin producing similar compounds. Production of these chemicals exacts a high metabolic cost on the host plant, so many of these compounds are not produced in large quantities until after insects have begun to feed. Structural Defenses The Plant Cell All plant tissues contain pre-formed structural barriers that help limit pathogen attachment, invasion and infection. The cell wall is a major line of defense against fungal and bacterial pathogens. It provides an excellent structural barrier that also incorporates a wide variety of chemical defenses that can be rapidly activated when the cell detects the presence of potential pathogens. All plant cells have a primary cell wall, which provides structural support and is essential for turgor pressure, and many also form a secondary cell wall that develops

inside of the primary cell wall after the cell stops growing. The primary cell wall consists mostly of cellulose, a complex polysaccharide consisting of thousands of glucose monomers linked together to form long polymer chains. These chains are bundled into fibers called microfibrils, which give strength and flexibility to the wall. The cell wall may also contain two groups of branched polysaccharides: Cross-linking glycans include hemicellulose fibers that give the wall strength via cross-linkages with cellulose. Soft-rot pathogens often target pectins for digestion using specialized enzymes that cause cells to break apart: Comparison of cell wall types. The primary cell walls of red pepper cells A are relatively thin compared to the thick secondary cell walls of oak wood B. The walls of pear fruit stone cells C are so thick that the cell lumen is barely visible. Many cell walls also contain lignin, a heterogeneous polymer composed of phenolic compounds that gives the cell rigidity. Cutin, suberin, and waxes are fatty substances that may be deposited in either primary or secondary cell walls or both and outer protective tissues of the plant body, including bark. Cell walls contain proteins and enzymes that actively work to reshape the wall during cell growth yet thicken and strengthen the wall during induced defense. When a plant cell detects the presence of a potential pathogen, enzymes catalyze an oxidative burst that produces highly reactive oxygen molecules capable of damaging the cells of invading organisms. Reactive oxygen molecules also help strengthen the cell wall by catalyzing cross-linkages between cell wall polymers, and they serve as a signal to neighboring cells that an attack is underway. Plant cells also respond to microbial attack by rapidly synthesizing and depositing callose between the cell wall and cell membrane adjacent to the invading pathogen. Callose deposits, called papillae, are polysaccharide polymers that impede cellular penetration at the site of infection, and these are often produced as part of the induced basal defense response. Some plant cells are highly specialized for plant defense. There are many classes of idioblasts including pigmented cells, sclereids, crystalliferous cells, and silica cells. Pigmented cells often contain bitter-tasting tannins that make plant parts undesirable as a food source. Young red wines often contain high levels of tannins that give wine a sharp, biting taste. Sclereids are irregularly-shaped cells with thick secondary walls that are difficult to chew: Stinging nettles *Urtica dioica* produce stinging cells shaped like hypodermic needles that break off when disturbed and inject highly irritating toxins into herbivore tissues. Some stinging cells contain prostaglandins, hormones that amplify pain receptors in vertebrate animals and increase the sensation of pain. Crystalliferous cells contain crystals of calcium oxalate that may tear herbivore mouthparts when chewed and can be toxic if ingested. Members of the genera *Philodendron* and *Dieffenbachia* are very common tropical house plants that contain large amounts of these cells. Humans and pets who chew the leaves of these plants may experience a burning sensation in the mouth and throat that is often accompanied by swelling, choking, and an inability to speak. For these reasons, species of *Dieffenbachia* are commonly called dumb cane. Grasses and sedges contain rows of silica cells in their epidermal layers which give strength and rigidity to the growing leaf blades and deter feeding by chewing insects. Plant Tissues and Specialized Appendages The epidermis constitutes the outermost protective tissue system of leaves, floral parts, fruits, seeds, stems, and roots of plants until they undergo considerable secondary growth. It is the first line of defense against invading pathogens and consists of both specialized and unspecialized cells.

**6: Overview of Plant Defenses**

*The past two decades have seen an increasing number of virulent infectious diseases in natural populations and managed landscapes. In both animals and plants, an unprecedented number of fungal and fungal-like diseases have recently caused some of the most severe die-offs and extinctions ever.*

Matsuura Received Dec 5; Accepted Dec This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. This article has been cited by other articles in PMC. Abstract Disease control is largely based on the use of fungicides, bactericides, and insecticides—chemical compounds toxic to plant invaders, causative agents, or vectors of plant diseases. However, the hazardous effect of these chemicals or their degradation products on the environment and human health strongly necessitates the search for new, harmless means of disease control. There must be some natural phenomenon of induced resistance to protect plants from disease. Elicitors are compounds, which activate chemical defense in plants. Various biosynthetic pathways are activated in treated plants depending on the compound used. Commonly tested chemical elicitors are salicylic acid, methyl salicylate, benzothiadiazole, benzoic acid, chitosan, and so forth which affect production of phenolic compounds and activation of various defense-related enzymes in plants. Their introduction into agricultural practice could minimize the scope of chemical control, thus contributing to the development of sustainable agriculture. This paper chiefly highlights the uses of elicitors aiming to draw sufficient attention of researchers to the frontier research needed in this context. Introduction Plants are challenged by a variety of biotic stresses like fungal, bacterial, or viral infections. This lead to a great loss to plant yield. There are various options available for the farmers to protect their crop from the disease. Some options include development of resistant cultivars, biological control, crop rotation, tillage, and chemical pesticides. Nearly all chemical pesticides or fungicides have a direct antibiotic principle. But their use at commercial level is uneconomical, application is cumbersome, and some are proved to be carcinogenic. Therefore, considerable efforts have been accomplished to devise environmental-friendly strategies for the check of plant diseases and thus to save mankind from health hazard [ 1 ]. Plants can activate separate defense pathways depending on the type of pathogen encountered [ 2 ]. Jasmonic acid JA and ethylene dependent responses seem to be initiated by necrotrophs, whereas salicylic acid SA dependent response is activated by biotrophic pathogens. The mechanisms responsible for this differential recognition and response may involve crosstalk among these three different signal transduction pathways: JA, ethylene, and SA. The better understanding of plant signalling pathways has led to the discovery of natural and synthetic compounds called elicitors that induce similar defense responses in plants as induced by the pathogen infection [ 3 ]. Different types of elicitors have been characterized, including carbohydrate polymers, lipids, glycopeptides, and glycoproteins. In plants, a complex array of defense response is induced after detection of microorganism via recognition of elicitor molecules released during plant-pathogen interaction. Following elicitor perception, the activation of signal transduction pathways generally lead to the production of active oxygen species AOS , phytoalexin biosynthesis, reinforcement of plant cell wall associated with phenyl propanoid compounds, deposition of callose, synthesis of defense enzymes, and the accumulation of pathogenesis-related PR proteins, some of which possess antimicrobial properties [ 4 ]. AOS lead to hypersensitive response HR [ 5 ] in plants which is a localized or rapid death of one or few cells at the infection site to delimit the pathogen growth. Following the activation of HR, uninfected distal parts of the plant may develop resistance to further infection, by a phenomenon known as systemic acquired resistance SAR , which is effective against diverse pathogens, including viruses, bacteria, and fungi [ 6 ]. Host Pathogen Interaction Resistance in plant species is often divided into host- or nonhost-specific resistance. Host-specific resistance involves interactions between specific host and pathogen genotypes, which give a pathogen race-specific resistance. Nonhost resistance, shown by a whole plant species against a specific parasite or pathogen, is the most common form of resistance in plants towards the majority of potential pathogens [ 7 ]. The biochemical changes that occur during infection are very similar in host and nonhost resistant plants [ 8 ].

Disease spreads only in susceptible plants compatible interactions which are unable to recognize the pathogen or respond too slowly [ 2 ]. The hypersensitive response is triggered by the plant when it recognizes a pathogen. The identification of a pathogen typically occurs when avirulence Avr gene products, secreted by pathogen, bind to or indirectly interact with the product of a plant resistance R gene gene for gene model. When both the R gene and corresponding Avr genes are present, recognition occur, which lead to active resistance of the plant and avirulence of the pathogen. If either Avr gene in the pathogen or R gene in the host is absent or is mutated, no recognition will occur and outcome will be a compatible reaction and disease [ 9 ]. As a result of putative binding of these two partners, a signal transduction cascade is activated and lead to the activation of a variety of plant defense responses. The defense responses are associated with restriction of pathogen growth. R gene products are highly polymorphic and many plants produce several different types of R gene products, enabling them to act as a receptor of Avr proteins produced by many different pathogens [ 7 ].

**Hypersensitive Response HR** Direct physiological contact between the host and infecting parasite is obviously necessary for the activation of HR. The HR was first described by Stakman [ 10 ] to describe rapid host cell death in resistant wheat plants upon infection by rust fungi. Hypersensitivity is a rapidly developing defense reaction induced in incompatible host by a plant pathogen, which results in the death of a limited number of host cells and a concomitant localization of the pathogen. Some investigators have described the HR as resembling the process of apoptosis, the principal manifestation of programmed cell death in many animal cell types [ 11 ]. This definition has now expanded to include defense gene expression in addition to cell death [ 7 ]. The HR is analogous to the innate immune response found in animals. HR provides resistance to biotrophic pathogens that obtain their energy from living cells [ 12 ]. Phase 1 is rapid, transient, and nonspecific, whereas phase 2 occurs later and yields a much higher concentration of ROS [ 14 ]. This specific, biphasic response is proposed to be an important component of plant defense [ 15 ] because in compatible interactions only the first phase is induced [ 16 ]. The two distinct phases of the oxidative burst are seen only when an R gene and an Avr gene are both present, for example, with transgenic tomato plants differing only in the presence or absence of the R gene, Pto, and the bacterial pathogen, *Pseudomonas syringae* pv. This confirms that the second phase of the oxidative burst is associated with disease resistance [ 17 ]. These initial reactions are the prerequisite for initiation of the signalling network that will trigger the overall defense response [ 19 ].

**Sources of ROS** ROS are toxic intermediates that are generated through the sequential one electron reduction steps of molecular oxygen [ 20 ]. Various enzyme systems have been proposed as the source of ROS in plants. An NADPH oxidase system similar to that of mammalian systems or a pH-dependent cell wall peroxidase may be two sources of oxidative burst [ 21 ]. For the protection from oxidative damage, plant cells contain both oxygen radical detoxifying enzymes such as catalase, peroxidase, and superoxide dismutase, and nonenzymatic antioxidants such as ascorbate peroxidase and glutathione-S-transferase [ 55 ]. These enzymes play a crucial role in the protection of plant cells from oxidative damage at the sites of enhanced ROS generation [ 56 ]. The cooperative function of these antioxidants plays an important role in scavenging ROS and maintaining the physiological redox status of organisms [ 57 ].

**Systemic Acquired Resistance SAR** Host plants can be protected against further pathogen attack if they have survived earlier infection by phytopathogenic viruses, bacteria, or fungi. It appears that the first infecting pathogen immunizes the plant against further infections by homologous pathogens, even though the plant may not carry gene determining cultivar-specific resistance. The readiness of the plant to repel subsequent pathogen attacks spread throughout the whole plant. This response is called systemic acquired resistance SAR. The development of SAR is often associated with various cellular defense responses, such as synthesis of PR proteins, phytoalexins and accumulation of AOS, rapid alterations in cell wall, and enhanced activity of various defense related enzymes [ 58 ].

**Sequence of Events Associated with the Establishment of SAR** The onset of SAR in noninfected plant organs is triggered by the phloem mobile signal which is released following pathogen infection. The signal travels throughout the plant and transduced in target tissues. Following signal transduction, resistance is maintained for several days and weeks and this is likely due to de novo gene expression. The biochemical changes that occur during SAR can be divided into two phases, that is, initiation and maintenance. Physiological changes during initiation phase may be transient and short lived, but during

maintenance a quasisteady state should exist. Elicitors and Their Mode of Action Originally the term elicitor was used for molecules capable of inducing the production of phytoalexins, but it is now commonly used for compounds stimulating any type of plant defense [ 59 – 61 ]. Eventually, the induction of defense responses may lead to enhanced resistance. This broader definition of elicitors includes both substances of pathogen origin exogenous elicitors and compounds released from plants by the action of the pathogen endogenous elicitors [ 59 , 62 ]. Elicitors are classified as physical or chemical, biotic or abiotic, and complex or defined depending on their origin and molecular structure Table 1. Table 1 List of various types of plant elicitors. Type of elicitors and their examples Physical elicitors.

## 7: Plant pathology - Wikipedia

*Microbial pathogens and herbivores that cause disease or inflict damage to plants are ubiquitous in nature. To withstand and counteract invasions by these, plants have evolved several overlapping.*

These organisms include fungi, bacteria, and viruses. Therefore, on a statistical basis alone, you are likely to encounter fungal diseases much more often than those caused by other types of pathogens. We will now proceed to a description of the main characteristics of fungi. Fungi include the molds and mildews that we are all familiar with in Florida. At one time fungi were considered to be types of plants. Indeed, mycology, the scientific study of fungi, is still done today in botany departments. However, in modern biology, fungi are not considered plants. They are placed in their own Kingdom Mycota, for the serious biologists out there, with equivalent status to the familiar Animal and Plant Kingdoms. Sometimes, growth of fungi is so profuse that a large enough mass mycelia - multicelled microscopic strands will accumulate to be seen with the naked eye. A good example is the growth of the target spot fungus on this ripe tomato fruit. Figure 7 Target spot on ripe tomato fruit. Most of the time, however, careful examination with a microscope is needed to see fungi and ultimately identify them. Fungi consist of multi-celled microscopic strands. Often, spores, or the reproductive structures of fungi are readily visible, as seen in this photomicrograph of the fungus *Botrytis*. Figure 8 It is the peculiar size, shape, coloration, etc. Figure 9 Sometimes, we encounter important pathogenic fungi that do not readily form spores. A good example is the root-infecting fungus, *Rhizoctonia*, shown here. We take note of the distinctive right-angle branching of the fungal threads mycelia in making an identification of *Rhizoctonia*.

**Plant Pathogenic Bacteria** Our next group of pathogens is the bacteria. These are smaller than fungi. Though fungi cause more diseases than bacteria, bacterial diseases are generally more difficult to control. Again, bacteria are not plants. They are one-celled microorganisms, requiring good, powerful light microscopes to be seen. Though some bacteria produce resistant spores, no plant pathogenic bacteria do so. Details of bacteria are best seen at the very high magnifications of electron microscopes. Figure 10 Note the whip-like appendages flagella of the bacteria in this photomicrograph. Figure 10a Bacteria depend on outside agents for dispersal. They do not spread on the wind as many fungi do. Many bacterial diseases can be spread readily simply by touching an infected plant and then by touching a healthy plant. Like human bacteria, plant pathogenic bacteria are extremely contagious. Bacteria cannot penetrate the cuticle of plants but must enter the plant through a wound or natural opening.

**Plant Viruses** Viruses are by far the smallest of the pathogens considered in this program. The term "organism" may not be appropriate for a virus. They must have a living host in order to reproduce replicate. Electron microscopes are needed to see viruses. Even the best light microscopes are not good enough to see such tiny particles. Figure 11 This is a picture of the rod-shaped virus particles of the tobacco mosaic virus, the first plant-infecting virus discovered. Figure 12 Viruses usually are vectored or carried from infected to healthy plants by insects. Can you identify the insect in this picture?

## 8: pathogen induced disease

*We show that expression of piox is rapidly induced in response to various cellular signals mediating plant responses to pathogen infection and that activation of piox expression is most likely related to the oxidative burst that takes place during the cell death processes examined.*

Pathogenic fungi Fungi comprise a eukaryotic kingdom of microbes that are usually saprophytes consume dead organisms but can cause diseases in humans, animals and plants. Fungi are the most common cause of diseases in crops and other plants. The typical fungal spore size is 1–40 micrometers in length. Prion According to the prion theory, prions are infectious pathogens that do not contain nucleic acids. These abnormally folded proteins are found characteristically in some diseases such as scrapie , bovine spongiform encephalopathy mad cow disease and Creutzfeldt–Jakob disease. Human parasites Some eukaryotic organisms, including a number of protozoa and helminths , are human parasites i. Algal[ edit ] Examples of algae acting as a mammalian pathogen are known as well, notably the disease protothecosis. Protothecosis is a disease found in dogs, cats, cattle, and humans caused by a type of green alga known as prototheca that lacks chlorophyll. Treatment and health care[ edit ] Bacteria are usually treated with antibiotics while viruses are treated with antiviral compounds. Eukaryotic pathogens are typically not susceptible to antibiotics and thus need specific drugs. Infection with many pathogens can be prevented by immunization. A small amount of pathogens are used in vaccines to make immunity stay alert and strengthen defense on the insides to prepare for a larger quantity of the virus ever getting inside. Hygiene is critical for the prevention of infection by pathogens. Sexual interactions[ edit ] Many pathogens are capable of sexual interaction. Among pathogenic bacteria sexual interaction occurs between cells of the same species by the process of natural genetic transformation. Transformation involves the transfer of DNA from a donor cell to a recipient cell and the integration of the donor DNA into the recipient genome by recombination. Examples of bacterial pathogens capable of natural transformation are Helicobacter pylori , Haemophilus influenzae , Legionella pneumophila , Neisseria gonorrhoeae and Streptococcus pneumoniae. Meiosis involves the intimate pairing of homologous chromosomes and recombination between them. Examples of eukaryotic pathogens capable of sex include the protozoan parasites Plasmodium falciparum , Toxoplasma gondii , Trypanosoma brucei , Giardia intestinalis , and the fungi Aspergillus fumigatus , Candida albicans and Cryptococcus neoformans. This process involves pairing of homologous genomes and recombination between them by a process referred to as multiplicity reactivation. Examples of viruses that undergo this process are herpes simplex virus , human immunodeficiency virus , and vaccinia virus.

## 9: Role of Elicitors in Inducing Resistance in Plants against Pathogen Infection: A Review

*In plants, a complex array of defense response is induced after detection of microorganism via recognition of elicitor molecules released during plant-pathogen interaction.*

Determine the impacts of high temperatures, limited water availability, and elevated carbon dioxide CO<sub>2</sub> concentrations on: A plant-grazing insect pest interactions and signaling; and B plant pathogen relationships with host plants. C Elucidate chemical mechanisms responsible for regulation of nematode repellence and attraction to hosts and development of nematodes. Determine plant physiological mechanisms that mediate the effects of elevated atmospheric CO<sub>2</sub>, temperature, and limited water availability on plant-grazing insect pest interactions and plant pathogen interactions. Experiments will be carried out in controlled environment chambers. Studies for objectives 1a, b and 2 will focus on maize cultivar Golden Queen as the host crop plant, on European corn borer as the insect herbivore, and on *Fusarium graminearum* as the fungal pathogen. The main focus will be on determining the impact of treatments on generation of plant volatiles that may contribute to changes in plant defense mechanisms and signaling, or generation of plant toxins, but measurements will also be made to assess the impacts on plant productive capacity as well. Experiments will determine the effects of elevated carbon dioxide concentrations and limited water availability, individually and jointly, on the induction of plant defense chemicals in response to planned infestation of maize plants with European corn borer or with infections of F. The second experiments will determine the effects on interactions of elevated carbon dioxide and high temperatures associated with predicted climate change. Studies on sub-objective 1c will focus on plant and nematode produced compounds that repel, attract or inhibit development of *Meloidogyne* root knot nematodes. We will collect volatiles and water soluble exudates from plants and nematodes and conduct bioassays to determine repellence, attractiveness and developmental regulators. Chemicals will be purified by chromatographic methods coupled with bioassays. Synthesized compounds will be tested in laboratory and field assays to determine efficacy. One anticipated effect of increasing atmospheric carbon dioxide CO<sub>2</sub> is increased frequency and duration of drought. Plants respond to abiotic and biotic stresses by activating signal transduction cascades to coordinate physiological responses necessary for adaptation and survival. Abiotic stress affects some of the same hormonal signals stimulated by herbivory and can interfere with defense responses. Therefore, the combined effects of elevated CO<sub>2</sub> and drought on biotic stress induced volatile production was measured on Golden Queen sweet corn at ambient ppm and elevated ppm CO<sub>2</sub>. Three-week-old plants were subjected to drought for seven days. Leaf volatile production was not significantly altered by abiotic stress alone. Next the production of volatiles induced by Fall Armyworm feeding on the fifth leaf for 24 hours was measured. Less volatiles were produced by drought stressed plants and by plants grown at elevated CO<sub>2</sub>. An Herbivory Response Peptide was used to stimulate volatile production. Leaves from different environmental conditions were excised and placed in this peptide solution for 16 hours before collecting volatiles. Drought stressed leaves treated with this peptide emitted less volatiles than well-watered leaves. Similarly, peptide treated leaves grown at elevated CO<sub>2</sub> emitted less volatiles than peptide treated leaves grown at ambient CO<sub>2</sub>. In contrast to volatiles emitted, the internal leaf volatile concentrations were not lower in drought stressed or elevated CO<sub>2</sub> leaves, nor were the transcript levels of caryophyllene TPS23 and bergamotene TPS10 terpene synthases significantly attenuated by the environmental stresses. Results indicate that induced corn volatile emission may be hindered by physiological changes caused by climate change. In a second study, Golden Queen sweet corn was exposed to three levels of drought stress none, moderate, and severe with and without Fall Armyworm. Third instar larvae fed on leaves for 24 hours. Volatile organic compounds from the leaves were collected and analyzed. After 24 hours of feeding, leaves of non-stressed plants emitted more volatiles, including monoterpenes and sesquiterpenes, and volatile production decreased as drought stress increased. Without herbivory, there was less volatile production. Under severe drought stress, Fall Armyworm feeding induced more volatiles than with no Fall Armyworm. The reduction of emissions with increased drought stress is possibly related to stomatal closure. Under drought stress, plant roots also produced less volatiles. Further research is needed to determine if this

reduction was caused directly from drought or was due to escape of volatiles through larger air-space pores of the dryer soil. Reductions of volatile emissions imply that plants under drought stress have less defenses against herbivory and may be more vulnerable in future climates. Corn photosynthetic capacity was affected by severe drought but no significant interaction was found between Fall Armyworm feeding and photosynthetic capacity of the corn plant. Accomplishments 01 Plant produced attractants for entomophagous nematodes. Ultraviolet UV radiation exclusion by polycarbonate greenhouses causes abnormal soybean internode elongation. Climate change studies focusing on soybean seed set and yield responses to elevated temperatures were conducted in controlled environments of an 8-room polycarbonate greenhouse. ARS researchers and University of Florida collaborators at Gainesville, Florida discovered that two soybean cultivars produced abnormally long internodes that increased mainstem lengths by about 3-fold, but seed set and yield were affected little. Spectral radiometer measurements showed essentially no transmission through polycarbonate materials below nm wavelengths; however greenhouse glass, special acrylics, and certain thin film claddings transmitted UV-A nm and sometimes UV-B nm well. Other studies are few but outdoor UV exclusion experiments in India showed that soybean, guar bean, cowpea, and some types of mung bean exhibited internode elongation, but not corn and other types of mung bean in the U.S. These findings should provide essential information for a selection of appropriate greenhouse or growth chamber materials for research or for commercial production of certain plants, or to lead to use of supplemental UV lighting. Furthermore, these findings should stimulate additional fundamental research into UV photoreceptors that govern genetic control of plant photomorphogenic responses. Biological control is an attractive alternative to soil fumigation with methyl bromide. Or potential replacements, that not only will kill unwanted plant parasitic organisms but also beneficial organisms that in a natural situations often can control a pest to acceptable levels. Larvae of the root weevil *Diaprepes abbreviatus* are serious pests on numerous agricultural plants and infestations are therefore routinely treated with pesticides or soil fumigation. We found that beneficial entomopathogenic nematodes were attracted by damage induced root volatiles and thus selection of suitable root stock, or cultivars, could reduce the need for chemical control of root feeding pests. We have now demonstrated in field trials that the citrus produced attractant, pregeijerene, strongly attracted several species of beneficial native Entomopathogenic nematode EPN species and thus appear to be a potential key signal for below ground chemical control. We have earlier discovered that many nematode species utilize pheromones to regulate numerous behaviors, such as mating and entry into resting stage. We have now discovered that dispersal by nematode infectious stages is also regulated by pheromones, and furthermore that multiple entomopathogenic as well as plant pathogenic nematode species can distinguish and respond to other nematode signals. These signals can be used to improve the host searching behavior of commercially reared entomopathogenic nematodes when applied to insect infested fields. 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Greenhouse gas fluxes of drained organic and flooded mineral agricultural soils in the United States. Managing agricultural greenhouse gases. Leaf photosynthesis and carbohydrates of CO<sub>2</sub>-enriched maize and grain sorghum exposed to a short period of soil water deficit during vegetative development. Testing effects of climate change in crop models. Handbook of climate change and agroecosystems. Impact of elevated carbon dioxide and temperature on growth and sugar yield of the C4 sugarcane. Current Topics in Plant Biology. Interspecific nematode signals regulate dispersal behavior. Subterranean, herbivore-induced plant volatile increases biological control activity of multiple beneficial nematode species in distinct habitats. Approach from AD Experiments will be carried out in controlled

environment chambers. ARS scientists at Gainesville, Florida embarked on new research on the impact of abiotic stresses related to Global Climate Change on plant defense responses induced by biotic stresses of insect herbivory and plant pathogens. Increase in atmospheric carbon dioxide CO<sub>2</sub> is anticipated to alter rainfall, which could decrease the yield and quality food crops. Drought stress alone can impair plant productivity, but in nature, plants are exposed to both abiotic stresses and biotic attacks. *Fusarium verticillioides* is an important fungal pathogen of corn that causes seedling decay, stalk and ear rot, and produces carcinogenic mycotoxins. Consumption of mycotoxins through contaminated food can cause serious illness that affect both humans and animals. Water stress and herbivore damage promotes the production of these mycotoxins in infected plants. At the site of *F.* Phytoalexin production is stimulated through the jasmonic acid-ethylene signaling pathway. Jasmonic acid production was suppressed in drought stressed plants infected with *F.* This suppression might have been caused by an increase in abscisic acid, a plant hormone produced in response to water-deficit conditions, which is thought to inhibit Jasmonic acid-ethylene dependent resistance. Unlike the infected stem tissue, drought stressed roots of uninfected and infected plants had increased phytoalexin concentrations. The leaf area and root mass of plants exposed to double ambient CO<sub>2</sub> levels were significantly higher than ambient CO<sub>2</sub> grown plants. Phytoalexin production of infected stem tissue was unaltered by elevated CO<sub>2</sub> concentrations and there was no induction the plant roots. Leaf photosynthesis decreased with drought stress, and drought stressed plants that had pathogen infection did not recover as well after rehydration. We are continuing to examine the effect of both elevated CO<sub>2</sub> and drought stress on plant phytoalexin production. Regarding infection of corn with European Corn Borer, total quantities of benzoxazinoids and phytoalexins kauralexins were increased as early as 24 hrs after feeding started. With the history of the baseline of sweet corn defenses under normal current climate conditions at this location, we can further investigate modifications of plant defenses that could occur with climate change. Our results indicate that the perception and response to abiotic stress may dictate or alter subsequent biotic stress responses. Effects of spring post-planting flooding on early soybean production systems in Mississippi.

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