TRANSMISSION OF PLANT DISEASES BY INSECTS

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Plant diseases appear as necrotic areas, usually spots of various shapes and sizes on leaves, shoots, and fruit; as cankers on stems; as blights, wilts, and necrosis of shoots, branches and entire plants; as discolorations, malformations, galls, and root rots, etc. Regardless of their appearance, plant diseases interfere with one or more of the physiological functions of the plant (absorption and translocation of water and nutrients from the soil, photosynthesis, etc.), and thereby reduce the ability of the plant to grow and produce the product for which it is cultivated. Plant diseases are generally caused by microscopic organisms such as fungi, bacteria, nematodes, protozoa, and parasitic green algae, that penetrate, infect, and feed off one or more types of host plants; submicroscopic organisms such as viruses and viroids that enter, infect, spread systemically and affect the growth of their host plants; parasitic higher plants which range from about an inch to several feet in size and penetrate and feed off their host plants. Plant diseases are also caused by abiotic, environmental factors such as nutrient deficiencies, extremes in temperature and soil moisture, etc. that affect the normal growth and survival of affected plants.

Of the aforementioned causes of disease, many of the microscopic organisms and of the viruses are transmitted by insects either accidentally (several fungi and bacteria) or by a specific insect vector on which the pathogenic organism (some fungi, some bacteria, some nematodes, all protozoa causing disease in plants, and many viruses) depends on for transmission from one plant to another, and on which some pathogens depend on for survival (Fig. 1).

The importance of insect transmission of plant diseases has generally been overlooked and greatly underestimated. Many plant diseases in the field or in harvested plant produce become much more serious and damaging in the presence of specific or non-specific insect vectors that spread the pathogen to new hosts. Many insects facilitate the entry of a pathogen into its host through the wounds the insects make on aboveground or belowground plant organs. In some cases, insects help the survival of the pathogen by allowing it to overseason in the body of the insect. Finally, in many cases, insects make possible the existence of a plant disease by obtaining, carrying, and delivering into host plants pathogens that, in the absence of the insect, would have been unable to spread, and thereby unable to cause disease. It is offered as a guess that 30-40% of the damage and losses caused by plant diseases is due to the direct or indirect effects of transmission and facilitation of pathogens by insects.

Insects and related organisms, such as mites, are frequently involved in the transmission of plant pathogens from one plant organ, or one plant, to another on which then the pathogens cause disease. Equally important is that

insects can and do transmit pathogens among plants from one field to another, in many cases even when the fields are several to many miles apart. Almost all types of pathogens, that is, fungi, bacteria, viruses, nematodes, and protozoa, can be transmitted by insects. Insects transmit pathogens, such as many fungi and bacteria, mostly externally on their legs, mouthparts, and bodies. Almost all plant pathogenic viruses, all phytoplasmas, xylem- and phloem-inhabiting fungi and bacteria, some protozoa, and some nematodes are also transmitted by insects, and they are usually carried by the insect internally. The insects that transmit fungi and bacteria externally on their bodies and legs belong to many orders of insects. On the contrary, the insects that transmit the other pathogens listed above internally are very specialized and specific for the pathogen they transmit and belong to a certain species or genus of insects (Fig. 2).

Insects transmit pathogens in three main ways. 1) Many insects transmit bacteria and fungal spores passively by feeding in or walking through an infected plant area that has on its surface plant pathogenic bacteria or fungal spores as a result of the infection. The bacteria and spores are often sticky, cling to the insect as it moves about, and are carried by it to other plants or parts of the same plant where they may start a new infection. 2) Some insects transmit certain bacteria, fungi, and viruses by feeding on infected plant tissues and carrying the pathogen on their mouthparts as they visit and feed on other plants or plant parts. 3) Several insects transmit specific viruses, phytoplasmas, protozoa, nematodes, and xylem- and phloem-inhabiting bacteria by ingesting (sucking) the pathogen with the plant sap they eat. Subsequently, the pathogen circulates through the body of the insect until, with or without further multiplication in the insect, the pathogen reaches the salivary glands and the mouthparts of the insect through which it is injected into the next plant on which the insect feeds (Fig. 3).

Role of insects in bacterial diseases of plants

In most plant diseases caused by plant pathogenic bacteria (especially in those that cause spots, cankers, blights, galls, or soft rots, bacteria), which are produced within or between plant cells, escape to the surface of their host plants as droplets or masses of sticky exudates (ooze). The bacteria exudates are released through cracks or wounds in the infected area, or through natural openings such as stomata, nectarthodes, hydathodes, and sometimes through lenticells, present in the infected area. Such bacteria are then likely to stick on the legs and bodies of all sorts of insects, such as flies, aphids, ants, beetles, whiteflies, etc., that land on the plant and come in contact with the bacterial exudates. Many of these insects are actually attracted by the sugars contained in the bacterial exudate and feed on it, thereby further smearing their body and mouthparts with the bacteria-containing exudate. When such bacteria-smeared insects move to other parts of the plant or to other susceptible host plants, they carry on their body numerous bacteria. If the insects happen to land on a fresh wound or on an open natural opening, and there is enough moisture on the plant surface, the bacteria may multiply, move into the plant, and begin a new infection. The same happens if the

insects happen to create a fresh wound on the plant.

The type of insect transmission of bacteria is probably quite common and widespread among bacterial diseases of plants, but it is passive and haphazard, depending a great deal on the availability of wounds or moisture on the plant surface. In any case, there are few data on how frequently such transmission occurs, and many conclusions about it are the result of conjecture. A further point that has been made is that insects which, whether above or below ground, wound the host plant organs (roots, shoots, fruit, etc.) by feeding or by ovipositing in them, increase the probability of transmission of plant pathogenic bacteria. This occurs because such insects place the bacteria, with their mouthparts or the ovipositor, in or around wounded plant cells, where they are surrounded by a suspension of nutrients (plant cell sap) in the absence of active host defenses and where they can multiply rapidly and subsequently infect adjacent healthy tissues.

Numerous plant diseases could be listed among those in which bacteria are spread by insects passively as described above, for example, the bacterial bean blights, fire blight of apple and pear, citrus canker, cotton boll rot, crown gal, bacterial spot and canker of stone fruits, etc. In several bacterial diseases, however, the causal bacterium has developed a special symbiotic relationship with one or a few specific types of insects and depends a great deal on these insects for its spread from infected to healthy host plants. Some of the better known bacterium - insect associations are described briefly below.

Bacterial soft rots

Bacterial soft rots cause tremendous losses worldwide, particularly in the warmer climates and the tropics. They are caused primarily by the bacterium *Erwinia carotovora pv. carotovora*, to some extent by *Pseudomonas fluorescens* and *Ps. chrysanthemi*, and, occasionally, by species of *Bacillus* and *Clostridium*. The last two genera of bacteria cause rotting of potatoes and of cut fleshy leaves in storage while *Pseudomonas fluorescens* and *Ps. chrysanthemi* cause soft rots of many fleshy fruits and fleshy vegetables. The species *E. c. pv. carotovora* causes the vast majority of soft rots on fleshy plant organs of any type (leave, blossoms, fruit, stems, or roots), especially in storage and under cover or in plastic bags. Affected fleshy fruits are, for example, strawberries and other berries, cantaloupes, peaches, pears, etc.; vegetables, for example, tomatoes, potatoes, spinach, celery, onions, cabbage, etc.; and ornamentals, for example, cyclamen, iris, lily, etc. Nearly all fleshy vegetables are subject to bacterial soft rots. The soft rot bacteria enter the plant organ through a wound, sometimes in the field but more commonly during storage, and there they multiply rapidly, secrete enzymes that separate the cells from each other and macerate the plant cell walls, which causes the tissues to become soft and to rot. In many cases, these bacteria are accompanied in the rotting tissues by other saprophytic bacteria that further degrade the softened plant tissue and cause it to give off a foul odor. In all cases, rotting tissues become soft and watery, and slimy masses of bacteria ooze out from cracks in the tissues.

The soft rotting bacteria survive in infected fleshy organs in storage and

in the field, in plant debris, in infected roots and other plant parts of their hosts, in ponds and streams from where irrigation water is obtained, and to some extent in the soil and in the pupae of several insects. The seedcorn maggot, *Delia platura* (Meigen) (Diptera: Anthomyiidae), was shown to play an important role in the dissemination and development of bacterial soft rot in potatoes both in storage and in the field. The soft rot bacteria are usually introduced into a potato field on infected or contaminated seed pieces but they can also live in all stages of the insect, including the pupae, and there they may survive cold or dry weather conditions. The insect larvae become contaminated with the bacteria as they feed in, or crawl about on, infected seed pieces; they also carry the bacteria to healthy plants and there they deposit them into wounds they create. Even when the plants or storage organs are resistant to soft rot bacteria and can normally stop the advance of the bacteria by developing a barrier of cork layers, the maggots destroy the cork layers as fast as they are formed and the soft rot continues to spread. Some other related flies, for example, the bean seed maggot *Delia florilega* (Zetterstedt), *Drosophila busckii* Coquillett (Diptera: Drosophilidae), and probably others, seem to have analogous relationship to the soft rot of potato and other fleshy organs. It has also been shown that several other flies have similar relationships with soft rot bacteria and the host plants on which they prefer to feed. Such relationships, for example, exist between the cabbage maggot, *Delia radicium* (Linnaeus) and soft rot in the Brassicaceae; the onion maggot, *Delia antiqua* (Meigen), the onion black fly, *Tritoxa flexa* (Weidman) (Diptera: Otitidae), the seedcorn maggot, and the onion bulb fly, *Eumerus strigatus* (Fallen) (Diptera: Syrphidae) and the soft rot of onion; and the iris borer, *Macronoctua onusta* (Grote) (Lepidoptera: Noctuidae) and soft rot of iris.

The exact relationship between soft rot in each host and each specific insect found to possibly be involved in the transmission of soft rot bacteria from one organ or plant to another is not clear. There is little doubt, however, that insect transmission of soft rot bacteria does occur, that insects help introduce the bacteria into wounds they open, and that the presence of insects in soft-rotting tissues inhibits the defense reaction of the plants against the bacteria. The insects also, by carrying the soft rot bacteria internally in their bodies, help the bacteria survive adverse environmental conditions. On the other hand, the bacteria seem to help their insect vectors by preparing for them a more nutritive substrate through partial maceration of the host plant tissues.

Bacterial wilts of plants

In several bacterial diseases of plants, the bacteria enter the xylem conductive system of the plant and there they move, multiply, and clog up the vessels. The clogging of the xylem vessels is further increased by substances released from cell walls by bacterial enzymes and interferes with the translocation of water through the stems to the shoots of the plant. As a result of insufficient water, the leaves and shoots loose turgor, wilt, and eventually turn brown and die. In some bacterial wilts, the bacteria destroy and dissolve parts of the xylem walls and move into the adjacent tissues where

they form pockets full of bacteria from which the bacteria ooze out onto the plant surface through cracks or natural openings. In other bacterial wilts, the bacteria remain confined in the xylem and do not reach the plant surface until the plant is killed by the disease.

The wilt-causing bacteria overwinter in plant debris in the soil, in the seed, in vegetative propagative material, and in some cases, in their insect vector. They enter plants through wounds, and they spread from plant to plant through the soil, through tools and direct handling of plants, or through insect vectors. The most important bacterial wilts in which insects play a significant role in the transmission of the bacteria from plant to plant are described briefly below.

Bacterial wilt of cucurbits - Bacterial wilt of cucurbits has been reported from most developed countries but it probably occurs throughout the world. It affects many species of cucurbits, including cucumber, muskmelon, squash, and pumpkin. Watermelon is resistant or immune to bacterial wilt. Diseased plants develop a sudden wilting of their foliage and vines and eventually die. Diseased squash fruit develops a slimy rot in storage. Losses from bacterial wilt vary from an occasional wilted plant to destruction of 75 to 95% of the crop (Fig. 4).

Bacterial wilt of cucurbits is caused by the bacterium *Erwinia tracheiphila*. The bacterium survives in infected plant debris for a few weeks but it survives over winter in the intestines of its two insect vectors, the striped cucumber beetle (*Acalymma vittatum* [Fabricius]) and the spotted cucumber beetle (*Diabrotica undecimpunctata* Mannerheim [Coleoptera: Chrysomelidae]). The bacterium depends on these two vectors for its transmission to and inoculation of new plants. In the spring, striped cucumber beetles and, to a lesser extent, spotted cucumber beetles, that carry bacteria, feed and cause wounds on the leaves of cucurbit plants. The insects deposit bacteria in the wounds through their feces and the bacteria enter the wounded xylem vessels in which they multiply rapidly and through which they move to all parts of the plant. In the xylem, the bacteria excrete polysaccharides, secrete enzymes that break down some of the cell wall substances, and induce xylem parenchyma cells to produce tyloses in the xylem. All of them together form gels or gums that clog the vessels, especially at their end walls, thereby reducing the upward flow of water in the xylem by up to 80% and causing the leaves and vines to wilt. Beetles feeding on infected cucurbit plants pick up bacteria on their mouthparts and when they feed onto healthy plants they deposit the bacteria in the new wounds they have made. Thus, the bacteria start a new infection. Each contaminated beetle can infect several healthy plants after one feeding on an infected plant. It appears that a relatively small percentage of beetles become carriers of the bacteria through the winter. Spotted cucumber beetles transmit the wilt bacteria rather late in the season, therefore they are considered less important vectors of this disease than the striped cucumber beetles.

Bacterial wilt of corn - This disease is also known as Stewart's wilt of corn. It is caused by the bacterium *Pantoea* (formerly *Erwinia*) *stewartii*. It occurs in North and Central America and also in Europe and China. It is more severe in the northern states. The

bacterium invades the vascular tissues but it also spreads into other tissues. When sweet corn plants are affected at the seedling stage they may wilt rapidly and may die, or they develop pale green wavy streaks on the leaves, become stunted, wilt, and may also die. If infected plants survive, they often tassel prematurely, the tassels become bleached and may die, and produce deformed ears. Bacteria also enter the stalk pith, which they macerate in places near the soil line and form cavities. From there the bacteria invade all vascular tissues and spread throughout the plant. Field corn is more resistant to early infection but becomes more severely infected later in the season. Some hybrids are susceptible and their symptoms parallel those of sweet corn. Later infections, after tasseling, produce irregular streaks on the leaves that originate at feeding points of the corn flea beetle, *Chaetocnema pulicaria* Melsheimer. The corn wilt bacteria are also transmitted by the toothed flea beetle (*Chaetocnema denticulata* Illiger), the spotted cucumber beetle (*Diabrotica undecimpunctata howarti* Barber), and by the larvae of the seed corn maggot (*Delia platura* Meigen), wheat wireworm (*Agriotes mancus* Say), and the May beetle (*Phyllophaga* sp.). It appears, however, that overwintering and spread of the bacteria in the field is carried out primarily by the corn flea beetle.

These beetles cause direct damage to corn leaves and seedlings but their main damage comes from harboring and transmitting the bacteria from plant to plant. The beetles pick up the bacteria when they feed on infected corn plants. The bacteria survive in the digestive tract of the insect as long as the latter lives. The insects are also the main place where the bacteria overwinter. The corn flea beetles overwinter as adults in the upper 2-3 cm of soil in grass sod. They are rather sensitive to low temperatures, however. In mild winters, when the sums of mean temperatures for December, January, and February are above 3 to 4°C, large numbers of beetles survive. When the soil warms up to about 17 to 20°C, they begin to feed on corn seedlings, which they infect with bacteria. Following mild winters, bacterial wilt of corn is spread rapidly by corn flea beetles, and corn losses can be quite severe. During cold winters that average temperatures below 0°C, many of the beetles do not survive and the incidence and spread of bacterial wilt of corn the following spring and summer are quite limited.

Southern bacterial wilt of solanaceous and other crops - This vascular wilt is caused by the bacterium *Ralstonia solanacearum*. It occurs in the warmer regions around the world and is particularly severe in the tropics. It is known by different names in different hosts, for example, southern wilt or brown rot in potato and tomato, Granville wilt in tobacco, and Moko disease in banana. Insects, primarily bees (*Trigona corvine* Cockerell, Hymenoptera: Apidae), wasps (*Polybia* spp., Hymenoptera: Vespidae), and flies (*Drosophila* spp., Diptera: Drosophilidae) have been implicated as vectors. Because these and other insects visit infected stem wounds and natural abscission sites oozing out bacteria, they are considered as playing a role in the transmission of the bacteria to natural infection courts and in providing wounds for bacterial entry, but their importance as vectors has not been documented.

Fire blight of pears, apples and other rosaceous plants - The disease

is caused by the bacterium *Erwinia amylovora*. Fire blight occurs in North America, Europe and countries surrounding the Mediterranean Sea, and in New Zealand. It continues, however, to spread into new countries. Fire blight is the most devastating diseases affecting rosaceous plants. The symptoms consist of infected blossoms and young shoots becoming discolored and water-soaked, then being killed rapidly and appearing brown to black as though scorched by fire. The disease spreads rapidly into larger twigs and branches, which it also kills, and parts of or entire trees may be killed. At the base of twig or branch infections, cankers develop at the margins of which the bacteria overwinter. Fruit also become infected and ooze droplets of bacteria. The bacteria kill and macerate the contents of primarily parenchyma cells on flowers and in the bark of young shoots and twigs, but as they destroy these cells they move on mass in the bark. The bacteria also enter the phloem and xylem vessels through which they may move over relatively short distances.

The fire blight bacteria overwinter at the margins of cankers of twigs and branches. In the spring, the bacteria around cankers multiply and their byproducts absorb water and build up internal pressure. This results in droplets of liquid containing masses of fire blight bacteria oozing out of the cankers. The bacteria in the ooze are disseminated by splashing rain and also by flying and crawling insects, several of which are attracted to the bacterial ooze, and their legs, bodies, and mouthparts become smeared with bacteria. More than 200 species belonging to many insect groups, including aphids, leafhoppers, psyllids, beetles, flies, and ants, have been shown to visit oozing cankers and healthy blossoms, although bees and wasps seem not to visit oozing cankers routinely. Insects smeared with bacteria oozing out at cankers carry the bacteria to young shoots where they deposit them in existing wounds or in fresh wounds they make upon feeding, or in the nectar of the flowers. Once the fire blight bacteria are transmitted to blossoms by rain or insects, they enter the flower tissues through nectarthodes or wounds, multiply rapidly in them, and ooze out of them and commingle with the nectar in the flower. The same kinds of insects apparently can transmit fire blight bacteria from infected to healthy flowers but flower to flower transmission of fire blight bacteria is carried out so much more efficiently by pollinating insects, namely bees, that the contribution of other insects to that type of transmission seems to be relatively insignificant. As honeybees, wild bees, bumblebees, wasps, and other insects visit pear, apple, and other flowers infected with fire blight bacteria, their mouthparts, legs, and other body parts become smeared with the bacteria in the nectar. The insects then carry the bacteria and deposit them in the nectar of healthy flowers they visit and there the bacteria start new infections. The bacteria, however, do not survive on or in the insects for more than a few days and do not appear to overwinter in association with the insects.

Olive knot - Olive knot is caused by the bacterium *Pseudomonas savastanoi*. It occurs in the Mediterranean region, in California, and probably the other parts of the world where olive trees grow. The disease occurs as rough galls of varying sizes developing on leaves, branches, roots,

on leaf and fruit petioles, and on wounds in tree branches and trunks. Sometimes the galls are so numerous on twigs that the twigs decline and may die back. The galls are the result of growth regulators being produced by the bacteria, which grow and multiply in the intercellular spaces of the outer cells of the galls. In California, the bacteria are spread by running and splashing rain water that carries the bacteria to existing wounds, pruning wounds, and leaf scars. In other parts of the world, however, such as the Mediterranean region, the olive knot bacteria are also spread by the olive fly or olive fruit fly, *Bactrocera* (formerly *Dacus*) *oleae* (Gmelin) (Diptera: Tephritidae), which is the most destructive pest of olive in its own right.

The bacterium and the olive fly have developed a close symbiotic relationship that contributes to the transmission of the olive knot bacteria from tree to tree. The bacteria are carried by all stages (larvae, pupae, and adults) of the olive fly. The adult olive flies, and related fly species, have specialized structures along their digestive tract that are filled with bacteria. There is even a connection of the digestive tract with the oviduct that insures contamination of the eggs before oviposition. Transmission of the bacteria by the insect takes place during feeding and oviposition into olive tissues. The bacteria actually penetrate the egg through the micropyle, thereby ensuring that when the larvae hatch they are contaminated with the bacteria. It appears that while the olive fly plays a significant role in the transmission of the olive knot bacteria, the bacteria contribute to the insect by hydrolyzing proteins and making available to the insect certain amino acids needed by the insect for survival of the larvae and for development of adults.

Insect transmission of xylem-inhabiting bacteria

Quite a few important bacterial diseases of plants, primarily trees, are caused by the fastidious bacterium *Xylella fastidiosa*. These bacteria inhabit the xylem of their host plants and are rather difficult to isolate and to grow on the usual culture media. The diseases they cause differ from the vascular wilts caused by conventional bacteria in that instead of wilt they cause infected plants to decline, some of their twigs to die back, and in some cases the whole plant to die. The xylem-inhabiting fastidious bacteria are transmitted in nature only by xylem-feeding insects, such as sharpshooter leafhoppers (Cicadellidae: Cicadellinae) and spittlebugs (Cercopidae). *Xylella* bacteria seem to be distributed in tropical and semitropical areas worldwide. Among the most important diseases caused by *Xylella* are Pierce's disease of grape and citrus variegated chlorosis. Other diseases caused by xylem-inhabiting bacteria include phony peach, plum leaf scald, almond leaf scorch, bacterial leaf scorch of coffee, oak leaf scorch, and leaf scorch diseases of oleander, pear, maple, mulberry, elm, sycamore, and miscellaneous ornamentals, as well as the alfalfa dwarf disease (Fig. 5).

Pierce's disease of grape - Pierce's disease is a devastating disease of European-type grapevines (*Vitis vinifera*). It is caused by the xylem-inhabiting bacterium *Xylella fastidiosa*. It occurs in the Southern United States and in California, in Central America, and parts of northwestern South America. The presence of Pierce's

disease in an area precludes the production of European-type grapes in that area but some muscadine grapes and hybrids of European grapes with American wild grapes are tolerant or resistant to Pierce's disease. The Pierce's disease bacterium moves and multiplies in the water-conducting (xylem) vessels of shoots and leaves, some of which become filled with bacteria and reduce the flow of water through them. Leaves beyond such blocked vessels become stressed from lack of sufficient water and develop yellowing and then drying or scorching along their margins. During the summer, the scorching continues to expand towards the center of the leaf, while some or the entire grape clusters begin to wilt and dry up. Scorched leaves fall off leaving their petioles still attached to the vine, while the vines mature unevenly and have patches of brown (mature) and green bark. In the following season(s), affected grapevines show delayed growth and stunting. The leaves and vines repeat the symptoms of the first year and both, the top of the plant as well as the root system, decline and die back.

The bacterium that causes Pierce's disease of grape is *Xylella fastidiosa*. The bacterium apparently consists of various host specific strains. The strain that causes Pierce's disease of grape also causes alfalfa dwarf disease and almond leaf scorch. Apparently related but different strains of the bacterium cause citrus variegation chlorosis, the other related leaf scorch diseases of fruit and forest trees and of ornamental trees and shrubs. The identity and taxonomy, as well as the host range and vector preference of the possible strains of *Xylella fastidiosa*, are unknown. In all cases, the bacterium is transmitted from plant to plant through vegetative propagation, such as cuttings, budding, and grafting, and by one or more of several closely related insects. The known vectors of *Xylella fastidiosa* are sharpshooter leafhoppers (family Cicadellidae, subfamily Cicadellinae) or spittlebugs (family Cercopidae). It is possible that other or all sucking insects that feed on xylem sap, for example, the cicadas (family Cicadidae), are also vectors of *Xylella fastidiosa*. In California, there are at least four important sharpshooter leafhopper vectors of *Xylella*: The blue-green (*Graphocephala atropunctata*), the green (*Draeculacephala minerva*), the red-headed (*Carneocephala fulgida*), and the glassy-winged (*Homalodisca coagulata*) sharpshooters. The vectors may be different in other parts of the world. All vector insects acquire the bacteria when they feed on infected plants. Ingested bacteria seem to adhere to the walls of the foregut of the insect and when the insect moves to and feeds on the next healthy plant, the insect transmits the bacteria into the xylem vessels of that plant where they multiply and cause a new infection. Once a vector acquires bacteria from a diseased plant, it remains infective indefinitely. When, however, infective insects shed their external skeleton by molting, they loose the bacteria and must feed on a diseased plant again before they can transmit the bacteria to healthy plants.

Insect transmission of phloem-inhabiting bacteria

At least four plant diseases are caused by bacteria that inhabit only the phloem of their host plants. These diseases include the destructive citrus greening disease, the severe papaya

bunchy top disease, the cucurbit yellow vine disease, and the infrequent clover club leaf disease. The bacteria causing these diseases have not yet been grown on nutrient media and so far many of their properties remain unknown. All of them, however, are transmitted from plant to plant only by specific insect vectors. The citrus greening bacterium is transmitted by a psyllid, while the papaya bunchy top disease bacterium and the clover club leaf bacterium are transmitted by leafhoppers, and the cucurbit yellow vine disease bacterium is transmitted by the squash bug. In the psyllid and leafhopper vectors, the bacterium also multiplies in and is passed from the mother insect to its offspring through the eggs (transovarial transmission). It is not known what happens to the bacteria transmitted by the squash bug.

Citrus greening disease - Citrus greening is a very destructive disease of all types of citrus. It occurs in most citrus producing areas of Asia, including the Arabian Peninsula, and in Africa. The disease is caused by the bacterium *Liberobacter asiaticum* in Asia, and *L. africanum* in Africa. Both bacteria are limited to the phloem of the host plants, and have yet to be cultured. The disease first appears as a chlorosis and leaf mottling on one shoot or branch, which it has given it the name “huanglongbing”, or “yellow shoot”, in Chinese. Later on, entire trees become chlorotic as though they are suffering from zinc deficiency, their twigs die back, and the trees decline rapidly and become non-productive. Fruit on diseased trees is small, lopsided, and does not color uniformly as it ripens but large parts of it remain green even when mature, thereby the “greening” name of the disease. Diseased fruit is also quite bitter.

Citrus greening is spread by vegetative propagation with buds and grafts, and by at least two citrus psyllids: *Diaphorina citri* Kuwayama, which is the principal vector of the more severe and more destructive Asian form of the citrus greening bacterium that occurs at higher temperatures (30 to 35ºC), commonly found at lower elevations; and *Trioza erytreae* Del Guercio, which is the principal vector of the milder, less severe, lower temperature (27°C) African form of the bacterium, which is normally found at higher elevations. Both vectors, however, can transmit both forms of the bacterium. Asian psyllids acquire the bacterium within 30 minutes of feeding while African psyllids require 24 hours. The bacterium apparently multiplies in the vector and can be transmitted within 8 to 12 days from acquisition.

Infected plants and vectors have been introduced into several citrus-producing countries but in most cases it was eradicated before it could become established. The vector of the greening bacterium *Diaphorina citri* was introduced in Brazil in the early 1980s and in Florida in 1998 but, so far, the causal bacteria apparently have not been introduced and no trees have been found in either place to be infected with citrus greening.

Bunchy top of papaya disease - Bunchy top is a devastating disease of papaya. It occurs in most or all islands of the Caribbean Basin and, probably, also in Central America and in the northern part of South America. Young leaves of infected plants show mottling, then chlorosis and marginal necrosis, and become rigid. Internodes become progressively shorter, further apical

growth stops, and the plant develops a "bunchy" top. Older leaves may fall off, any fruits that are set are bitter, and the entire plant may die.

Bunchy top of papaya is caused by a rickettsia-like phloem-limited bacterium that moves and multiplies in the phloem elements of the plant. The bunchy top bacteria are transmitted from diseased to healthy papaya plants by the leafhoppers *Empoasca papayae* Oman and *E. stevensi* Young. Symptoms appear 30-45 days after inoculation.

Cucurbit yellow vine disease - Yellow vine disease affects watermelon, melon, squash, and pumpkin. It was first reported in the Texas-Oklahoma area and has since been found in Massachusetts, New York, and Tennessee. Affected plants show vines with yellow leaves, the phloem of leaves and vines becomes discolored, and the leaves and vines collapse and die. The disease has been severe in the Texas and Oklahoma areas where it annually destroys thousands of acres of cucurbits costing millions of dollars.

Cucurbit yellow vine disease is caused by a phloem-limited bacterium that has been placed in the species *Serratia marcescens* and its properties are still being characterized. The bacterium is most probably transmitted by insect vectors. The squash bug, *Anasa tristis*, is considered to be a vector of this bacterium, but its involvement in transmitting this bacterium has been questioned.

Insect transmission of plant diseases caused by mollicutes

Mollicutes are prokaryotes (bacteria) that lack cell walls. In nature, plant pathogenic mollicutes are limited to the phloem of their host plants. Plant pathogenic mollicutes are generally classified as belonging to the genus *Phytoplasma*. Most phytoplasmas have an irregular spherical to elongated shape and have been obtained and maintained on complex nutrient media, although they do not readily grow or multiply on them. A few plant pathogenic mollicutes typically have spiral morphology and belong to the genus *Spiroplasma*. Spiroplasmas grow and multiply readily on specialized nutrient media. Plant diseases caused by mollicutes appear as yellowing of leaves, proliferation of shoots (witches' brooms) and of roots, stunting of shoots and whole plants, greening of flowers, abortion of flowers and fruit, dieback of twigs, and decline and death of trees. Numerous important diseases of annual crops are caused by mollicutes, mostly phytoplasmas, for example, aster yellows of vegetables and ornamentals, tomato big bud (stolbur), corn stunt, etc. Phytoplasmas cause even more, and more severe, diseases on trees, including X-disease of peach, peach yellows, apple proliferation, elm yellows, pear decline, and lethal yellowing of coconut palms. Spiroplasmas also cause severe diseases, for example, corn stunt, and citrus stubborn disease.

All mollicutes, that is, phytoplasmas and spiroplasmas, are spread from plant to plant through vegetative propagation and, in nature, these pathogens depend for their transmission on phloem-feeding, sap-sucking insects, mainly leafhoppers, planthoppers, and psyllids. These insects can acquire the pathogen after feeding on appropriate infected plants for several hours or days, or if they are artificially injected with extracts from infected plants or insects. More insects become vectors when they feed on

young leaves and stems of infected plants than on older ones. The insect vector cannot transmit the pathogen immediately after feeding on the infected plant but it begins to transmit it after an incubation period of 10 to 45 days, depending on the temperature. The quickest transmission (10 days) occurs at about 30°C, while the slowest (45 days) takes place at about 10°C.

The reason for the incubation period is that the acquired phytoplasmas or spiroplasmas must first multiply in the intestinal cells of the insect vector and then move through the insect by passing into the hemolymph, then infect internal organs and the brain, and finally reach and multiply in the salivary glands. When the concentration of the pathogen in the salivary glands reaches a certain level, the insect begins to transmit the pathogen to new plants and continues to transmit it with more or less the same efficiency for the rest of its life. Insect vectors are not generally affected adversely by the phytoplasmas or spiroplasmas multiplying in their cells, but in some cases they show severe pathological symptoms. Phytoplasmas and spiroplasmas can be acquired as readily or better by nymphs than by adult leafhoppers, etc., and they survive through subsequent molts of the insect. The pathogens, however, are not passed from the adults to the eggs and to the next generation. For this reason, young insects of any stage must feed on infected plants in order to become infective vectors.

Some of the most important plant diseases caused by mollicutes and their insect vectors are described briefly below.

Aster yellows - Aster yellows is caused by phytoplasmas and occurs worldwide. It affects numerous annual crops, mostly vegetables and ornamentals, for example, tomato, lettuce, carrot, onion, potato, chrysanthemum, aster, and many others, on which it causes severe symptoms and serious losses, in some crops amounting to 10-25% of the crop and occasionally up to 80-90% of the crop. Plants infected with aster yellows develop general chlorosis (yellowing) and dwarfing of the whole plant, abnormal production of shoots and, sometimes, roots, sterility of flowers, malformation of organs, and a general reduction in the quantity and quality of yield. The aster yellows phytoplasma is transmitted by several leafhoppers, one of which is the aster leafhopper *Macrosteles fascifrons*. The various leafhopper vectors have a wide host range, as does the aster yellows phytoplasma. The phytoplasmas survive in perennial ornamental, vegetable, and weed plants. The vector leafhopper acquires the phytoplasmas while feeding by inserting its stylet into the phloem of infected plants and withdrawing the phytoplasmas with the plant sap. After an incubation period, when the insect feeds on healthy plants it injects the phytoplasmas through the stylet into the phloem of the healthy plants where they establish a new infection and multiply. The phytoplasmas move out of the leaf and spread throughout the plant causing the symptoms characteristic of the host plant.

Tomato big bud - The disease occurs in many parts of the world but except for a few areas, it is of little economic importance. It affects most Solanaceous vegetables and lettuce. The symptoms include small, distorted, yellowish green leaves and production of numerous thickened, stiff, and erect

apical stems that have short internodes. The flower buds are excessively big, green, and abnormal looking, and fail to set fruit. Fruit present when infection takes place becomes deformed.

Tomato big bud is caused by a phytoplasma that is transmitted by several leafhoppers, the main one of which is the common brown leafhopper *Orosius argentatus*. The insect feeds and breeds on infected weed hosts and when they become undesirable the insects move into tomato or other crops bringing with them the big bud phytoplasmas.

Apple proliferation - It is the most important insect transmitted disease of apple in most of Europe. It may also occur in South Asia and South Africa. Depending on prevalence in an orchard, apple proliferation may cause economic losses of 10-80% due to reduction in fruit size, total yield, and vigor of trees. The most conspicuous symptoms of apple proliferation are the production of witches' brooms or of leaf rosettes, and of enlarged stipules at the base of leaves. Affected trees leaf out earlier in the spring but flowering is delayed. The leaves, fruit, and entire trees are smaller, and fruit color and taste are also poor. Proliferating shoots are often infected with powdery mildew.

Apple proliferation is caused by a phytoplasma that also infects other wild and ornamental apple species, and possibly pear and apricot. The phytoplasma is spread in nature by several leafhoppers, including *Philaenus spumarius*, *Aphrophora alni*, *Lepyronia coleoptrata*, *Artianus interstitialis*, and *Fieberiella florii*. The leafhopper vectors acquire the phytoplasmas when they feed on the phloem elements of young leaves and shoots of infected apple trees and, after an incubation period, transmit the phytoplasmas into healthy apple trees. The time between inoculation and appearance of symptoms varies with the size of the inoculated tree. Young nursery trees may develop symptoms within a year while large established trees may do so two or three years after inoculation.

Pear decline - It is a serious disease of pear resulting in significant crop losses and also in stunting and death of affected pear trees grown on certain rootstocks. The disease, which is caused by a phytoplasma, occurs in North America and in Europe, and probably in many other parts of the world where pears are grown. The symptoms of pear decline may develop as a quick decline, that is, sudden wilt and death of a tree within a few days or weeks, with or without first showing reddening of leaves, or a slow decline. Quick decline usually develops on trees propagated on certain hypersensitive rootstocks in which a brown necrotic line develops at the graft union of the tree. Slow decline also occurs on trees grafted on the same or other rootstocks, and appears as a progressive weakening of the tree of varying severity. Slow declining trees have reduced or no terminal growth, have few, small, leathery, light green leaves whose margins are slightly rolled up and may be yellow or red in the autumn. Such trees may or may not die a few years after infection. Some infected pear trees, however, show primarily a reddening of the leaves in late summer or early autumn, and mild reduction in vigor.

The pear decline phytoplasma is transmitted from tree to tree by grafting and by the pear psylla (*Psylla pyricola* Forster) and in Europe, probably by *P. pyri* and *P. pyrisuga*. Pear psylla

acquires the phytoplasma after feeding on infected trees for a few hours and remains infective for several weeks. Young trees inoculated with phytoplasma by the insect show symptoms the same or the next year, while older trees may take longer. The phytoplasma is sensitive to low temperatures and dies out in the above-ground parts of the tree but survives in the tree roots. In the spring, the phytoplasma recolonizes the stem, branches and shoots and from the latter it can be acquired and transmitted again by the insect vectors.

Lethal yellowing of coconut palms - Lethal yellowing is a blight that kills coconut and some other palm trees within 3 to 6 months from the time the trees show the first symptoms. It occurs in Florida, Texas, Mexico, most Caribbean islands, in West Africa, and elsewhere. The disease appeared for the first time in Florida in 1971 and killed 15,000 coconut palm trees the first two years, 40,000 by the third year, and by the fourth year (1975), 75% of the coconut palms in the Miami area were dead or dying from lethal yellowing. The disease appears as a premature drop of coconuts followed by blackening and death of the male flowers. Subsequently, first the lower and then the other leaves turn yellow and then brown and die, as does the vegetative bud, and the entire top of the tree falls off leaving the palm trunk looking like a telephone pole.

Lethal yellowing is caused by a phytoplasma that lives and multiplies in the phloem elements of the plant. The main means of spread of lethal yellowing from tree to tree is through the planthopper *Myndus crudus*, although other vectors are also possible. As with the other phytoplasma diseases, the vector acquires the phytoplasma while sucking juice off the phloem of palm leaf veins, the phytoplasma multiplies in the vector during an incubation period, and the insect then transmits the phytoplasma when it visits and feeds on leaf veins of healthy palm trees.

Corn stunt - Corn stunt causes severe losses where it occurs although losses vary from year to year. It occurs in the southern United States, Central America, and northern South America. Symptoms consist of yellow streaks in young leaves followed by yellowing and reddening of leaves, shortening of internodes, stunting of the whole plant, and sterile tassels and ears.

Corn stunt is caused by the spiroplasma *Spiroplasma kunkelii*. The spiroplasma invades phloem cells from where it is acquired by its leafhopper vectors *Dalbulus maidis*, *Dalbulus eliminatus*, and other leafhoppers after feeding on infected plants for several days. The vectors transmit the spiroplasma after an incubation period of 2 to 3 weeks, during which the spiroplasma moves and multiplies in the insect.

Citrus stubborn disease - It is present and severe in hot and dry areas such as the Mediterranean countries, the southwestern United States, Brazil, Australia, and elsewhere. It is one of the most serious diseases of sweet orange and grapefruit. It is hard to diagnose but reduces yield, quality, and marketability of fruit dramatically. Infected trees grow upright but are stunted. There is less fruit and it is smaller, lopsided, green, and sour, bitter, and unpleasant.

Citrus stubborn disease is caused by the spiroplasma *Spiroplasma citri*, which is found in the phloem of affected orange trees. It is transmitted by budding and grafting and, in nature,

by several leafhoppers such as *Circulifer tenellus*, *Scaphytopius nitrides*, and *Neoaliturus haemoceps*.
Role of insects on fungal diseases of plants

As with bacteria, many insects are involved in the transmission of numerous plant pathogenic fungi from diseased to healthy plants. Insects are also involved in plant diseases by breaking the epidermis and other protective tissues of plants with their mouthparts or with their ovipositor and thereby allowing the fungus to enter. Most of the insect transmissions of fungi are accidental, that is, they occur because the insects happen to become externally or internally contaminated with the fungus or its spores when they visit infected plants and then carry the spores with them to the plants or plant parts they visit next. In some cases, insect transmission of a fungus occurs as the insect visits blossoms during pollination, in others it occurs while wounding plants during oviposition, and in other and most frequent cases, transmission occurs while wounding the plant during feeding. In relatively few cases, the insect and the fungus it transmits develop a symbiotic relationship in which each benefit from its association with the other.

Root-infecting fungi - It should be pointed out that there are innumerable cases for which there is circumstantial evidence that insects are apparently involved in the transmission of many plant pathogenic fungi and in the development of disease by them, but this has not been proven experimentally. In this category belong, for example, root infections by fungi such as *Pythium*, *Fusarium*, and *Sclerotium*, being facilitated by billbugs such as *Calendra parvula* and *Anacentrus deplanatus*, by the Hessian fly *Phytophaga destructor*, and by the southern and northern corn root worms *Diabrotica undecimpunctata howardii* and *Diabrotica longicornis*, respectively. In the black stain root disease of pines, hemlock, and Douglas fir is caused by the fungus *Leptographium wageneri*, the teliomorph of which is *Ophiostoma wageneri*, and is transmitted by the root-feeding bark beetle *Hilastes nigrinus* and two root and crown weevils, *Steremnius carinatus* and *Pissodes fasciatus*.

Stalk or stem-infecting fungi - Many fungi infecting stalks or stems, for example *Gibberella*, *Fusarium*, and *Diplodia* in corn, are apparently aided by various insects, for example, the widespread European corn borer, *Pyrausta nubilalis*.

Trunk and branch canker-causing fungi - Many fungi, such as species of *Neofabrea*, *Nectria*, *Leucostoma* (*Cytospora*), *Ceratocystis*, and *Leptosphaeria*, causing tree cankers, are apparently also often associated with and assisted by insects in the initiation and development of the cankers. The insects involved vary with the particular host and fungus. For example, the fungus *Neofabrea perennans* (*Gloeosporium perennans*), the cause of the perennial canker of apple, is transmitted by the wooly aphid *Eriosoma lanigerum*. The woolly aphids feed on the bark at the base of the trunk where they cause the formation of galls within which they multiply. In early spring the galls burst, the aphids come out and the fungus attacks the injured tissue and from it advances into healthy tissue and produces a canker. In the summer, the apple tree produces callus tissue and seals off the fungus and the spread of the canker stops. The aphids,

however, grow into the callus tissue and form a new gall, and the process is repeated.

The spittlebug *Aphrophora saratogensis* seems to be involved in the *Nectria* canker of pines, the nitidulid beetle *Carpophilus freemani* and the drosophilid fly *Chymomyza procnemoides* in the *Ceratocystis* cankers of stone fruit trees, while the tree cricket *Oecanthus niveus* and the raspberry midge *Thomasianna theobaldi* are involved in the tree cricket canker of apple and the midge canker of raspberry, respectively. Many more such insect-pathogen associations could be mentioned.

In the beech bark canker, caused by the fungus *Nectria coccinea* var. *faginata*, the fungus is transmitted to some extent by the scale insect *Cryptococcus fagi* but the main effect of the insect is in weakening the tree and reducing its defenses to the fungus. Thus, after beech trees have been heavily infested by scale insects for about three to five years, the fungus invades and kills the bark and the tree forms a canker that may girdle the tree partially or completely and may kill it.

In the birch constriction disease, the lower parts of shoots become constricted at the point where the apical birch woodwasp (*Pseudoxiphydria betulae* Ensl.) feeds on the shoots. The leaves above the constriction wither and die but cling to the twigs past the autumn. Almost all (92%) of the constrictions are also infected with the anthracnose fungus *Melanconium bicolor*.

A similar case in which twig canker initiation and development are facilitated by insects is the cacao dieback disease in which the fungi *Calonectria* (*Fusarium*) *rigidiuscula* and/or *Botryodiplodia theobromae* enter the twigs through wounds created by the feeding of the capsid insects *Sahlbergella singularis* and *Distantiella theobroma*. In isolated infections the tree defenses take over, isolate the fungus, and its further spread stops. In trees massively infested with the insect, however, the fungus develops unchecked in the insect-infested tissues and causes a chronic dieback of twigs. Control of the insects also halts the invasion by the fungus and the tree recovers.

In mango malformation disease, presumably caused by the fungus *Fusarium moniliforme*, the fungus is transmitted by the eriophyid mite *Aceria mangifera*, while other fungi seem to be carried in the digestive tract of certain termites.

Sooty molds - These are black-colored fungi that grow on the surfaces of mostly leaves of plants, especially in the tropics and subtropics. Sooty mold fungi do not penetrate and infect plants but cause disease by blocking the light from reaching the leaves. Sooty mold fungi do not parasitize plants but feed off the honeydew excreted by insects such as whiteflies, scales, mealybugs, aphids, etc. The sooty mold fungi are disseminated through their spores being blown about by wind. However, they are also spread by the honeydew-producing insects and, also, by several other types of insects such as flies, wasps, bees, and ladybug beetles, all of which seek honeydew as a source of food and in the process become smeared with fungus spores which they carry about.

Wood rots - Rotting of wood is carried out primarily by wood-rotting basidiomycete fungi. The shelf or conk-shaped fruiting bodies of many of these fungi are visited routinely by many types

of insects and it is believed that many of these insects act as vectors of the wood-rotting fungi. Insects and mites have also been implicated in the spread of some pine rust diseases, while at least three common scolytid beetles have been shown to be involved in the transmission of the scleroderis canker of pine and spruce.

Wood-stain diseases - Wood stain or wood discoloration diseases occur in conifer trees and felled timber. They are caused by the so-called blue-stain fungi, of which the most common are species of *Ceratocystis* and *Ophiostoma*. The blue-stain fungi are associated with several species of bark beetles, such as *Dendroctonus ponderosae*, *Ips pini*, etc., which serve as vectors of the fungi and provide them with wounds for penetration. On the other hand, the fungi reduce the water content of the tree and otherwise improve the microenvironment for the developing brood of insects. Such a fungus-insect relationship is described as true mutualistic symbiosis. In other blue-stain diseases, like the ones caused by the fungi *Trachosporium tingens* and *T. t.* var. *macrosporum*, the fungi are constantly associated with their bark-beetle vectors *Myelophilus* (*Blastophagus*) *minor* and *Ips acuminatus*, respectively, and are found regularly in the breeding places of the insects in pine stems. Such fungal-insect associations are known as symbiotic ambrosia cultures.

In the Southern United States, attacks of short-leaf pines by beetles like *Dendroctonus frontalis* are quickly followed by heavy fungus infection soon after the beetles tunnel through the bark and outer wood. Several fungi, including *Ceratocystis pini*, *Saccharomyces pini*, *Dacryomyces* sp. and *Monilia* sp. can be isolated from the infected wood and are carried by the same insects both externally and internally. A similarly complex association seems to occur in spruce attacked by *Dendroctonus engelmani*, followed by the fungi *Leptographium* sp., *Endoconidiophora* sp., or *Ophiostoma* sp. infecting the wood and causing a gray stain in the sapwood of the infected trees.

Vascular wilts

Several vascular wilts affect trees and some of them cause extensive death of trees, because the fungus responsible for the disease is transmitted from diseased to healthy trees by specific insect vectors. The spores produced by the causal fungi are sticky and are produced primarily inside the tree, therefore, they can be spread by no other means but only by certain insects closely associated with the disease. These vascular wilts include: 1) persimmon wilt, a devastating disease caused by the fungus *Cephalosporium diospyri* which enters through all kinds of wounds but is also transmitted by the powder-post beetle *Xylobiops basilaris* and the twig girdler beetle *Oncider cingulatus*, and 2) mango wilts, one caused by the fungus *Diplodia recifensis* and transmitted by the beetle *Xyleborus affinis*, and another caused by the fungus *Ceratocystis fimbriata* and transmitted by the scolytid beetle *Hypocryphalus mangiferae*. The other two vascular wilts are oak wilt and the Dutch elm disease and will be discussed in some detail below.

Oak wilt - It is caused by the fungus *Ceratocystis fimbriata* and is one of the most important diseases of forest trees. The fungus enters the xylem vessels of trees through fresh wounds to which it is carried by air or insects, and

through natural root grafts. Tree parts beyond the point of infection wilt, turn brown and die while newly infected wood shows dark streaks. The fungus is spread to healthy trees by nitidulid beetles such as *Carpophilus lugubris*, *Colopterus niger*, *Cryptarcha ample*, and several species of *Glischrochilus*. These fungi breed in the mycelial mats of the fungus between the bark and wood and carry the fungus both externally on their bodies and internally through their digestive tract. In addition to the nitidulid beetles, several scolytid beetles, such as *Monarthrum fasciatum* and *Pseudopityophthorus minutissimus*, the brentid beetle *Arrhenodes minuta*, the buprestid *Agrillus bilineatus*, the flat-headed borer *Chrysobothrys femorata*, and others, have been shown to carry the spores of the fungus, both externally and internally, when they emerge from the tunnels in diseased trees in which they breed and overwinter and to carry them to susceptible trees in the spring. Transmission of the oak wilt fungus by insects not only spreads the fungus and the disease to new trees and into new areas, it also increases the ability of the fungus to produce new variants and new races more virulent than the existing ones. This is accomplished by the insects bringing together in the same tree the compatible self-sterile mating types which results in the production of perithecia containing the sexual spores ascospores. The latter express any new characteristics brought together during the formation of the spores, some of the characteristics possibly being increased virulence.

Dutch elm disease - It is caused by the fungus *Ophiostoma ulmi* and the most recent variant *Ophiostoma novo-ulmi*, which is replacing the earlier species. The disease was first described in the Netherlands in 1921, found in Ohio and New York in the 1930s, and has since spread throughout the United States and much of the rest of the world. It kills elm tree twigs, branches and whole trees by clogging their xylem vessels and blocking movement of water from the roots to these parts. Dutch elm disease has been particularly devastating in the United States where the native elm tree *Ulmus americana* is extremely susceptible to the pathogen. The disease has killed almost all trees in its path, especially elm trees planted along streets and parks. Elm trees in forests have also been killed but many of them have escaped infection so far (Fig. 6).

The fungus causing Dutch elm disease is spread from diseased to healthy trees by the European elm bark beetle *Scolytus multistriatus* and the native elm bark beetle *Hylurgopinus rufipes*, and by natural root grafts. The fungus overwinters in the bark of dying or dead elm trees and logs as mycelium and spores. The elm bark beetles lay their eggs in galleries they make in the intersurface between bark and wood of weakened or dead elm trees. If the tree is already infected with the Dutch elm disease or if the insects carry with them spores of the fungus, the fungus grows and produces new spores in the tunnels. After the eggs hatch, the larvae make tunnels perpendicular to that made by the adult female and pupate. The adults emerge, carrying thousands of fungal spores on their body. The emerging adults prefer to feed on young twigs of trees and the crotch of small branches. As the beetles burrow into the bark and wood for sap, the spores they carry on their body are deposited in the wounded moist tissues of the tree. There the spores germinate and grow into the

injured bark and wood and the fungus reaches the xylem vessels of the tree in which it grows producing mycelium and spores. The latter are carried upward by the sap stream where they can start new infections. Shoots beyond the infected areas turn brown, wilt, and die, and as their number increases the tree shows more browning and more wilted branches. Eventually large parts of or entire trees wilt and die, while the fungus continues to grow and spread in the dead tree. Such trees are then visited by adult female beetles that lay their eggs in them and the cycle is repeated.

In Dutch elm disease there is a clear dependency of each organism, the fungus and the insect, on the other. Probably more than 99% of the elm tree infections are caused by the fungus being carried to the elm trees by the elm bark beetles. On the other hand, the elm bark beetles depend on the fungus for causing many elm trees to weaken and die, thereby becoming available as breeding grounds for the two species of elm bark beetles that transmit the fungus. The interdependence of the two organisms has provided the most effective means of managing the Dutch elm disease by burning or debarking dead elm trees and logs, thereby denying the insects the breeding ground they need and, through the reduced number of insects produced, reducing the number of elm trees to which they spread the disease.

Foliar diseases

Many foliar diseases are probably spread by various insects visiting and moving about on leaf surfaces that are exhibiting infections by spore-producing fungi. Spores or the spore containers of many such fungi are sticky or have appendages that cling to the legs or other body parts of such insects and are carried by them to other plants or plant parts they visit next. A few examples of foliar diseases in which insect transmission of the fungal pathogen has been shown to occur are described briefly below.

Powdery mildews - These are diseases that affect most annual and perennial plants. They are characterized by white superficial mycelial growth and sporulation by a small group of fungi that cause symptoms on leaves, shoots, blossoms and fruit of their host plants. Powdery mildews serve as food for many mycophagous fungi and produce large numbers of loosely attached spores. Such spores become attached to, and are disseminated by, insects with which they come in contact. Examples include the feeding of thrips on and the transmission of spores of the fungi *Sphaerotheca panosa* and *Uncinula necator* that cause powdery mildew on rose and grape, respectively. Although these fungi are disseminated readily by wind, it is likely that transmission is aided by insects.

Rust diseases - Most rust diseases produce several types of superficial spores on their host plants that, like those of the powdery mildews, are easily disseminated by air currents but are also visited, eaten, and transported by a wide variety of insects. Furthermore, many rust fungi produce spermatia and receptive hyphae in the same spermagonium but they are self-sterile. Many insects, when visiting such spermagonia become smeared with sticky spermatia. When the insects visit successive spermagonia, they transfer to the receptive hyphae spermatia from the opposite, compatible type. These spermatia can fertilize the receptive

hypha which then produces dikaryotic mycelium and spores that contain two nuclei. These dikaryotic spores have entirely different properties. For example, they can infect an entirely different host plant from the plant on which they were produced. The involvement of insects in rust diseases is, therefore, important in both the dissemination of the spores to new hosts and, more importantly, in the fertilization of the fungus and, thereby, in increasing the potential of the fungus to produce more new and possibly more virulent races.

Some other examples of foliar diseases in which insects have been shown to play a role in their transmission include: red pine needle blight caused by the fungus *Pullularia pullulans* and transmitted and aided in penetration of pine needles by a cecidomyiid midge; cucurbit anthracnose, caused by the fungus *Colletotrichum lagenarium* and transmitted and aided in penetration by the spotted cucumber beetle *Diabrotica undecimpunctata*; oil palm leaf spot, caused by the fungus *Pestalotiopsis palmarum* and transmitted and aided in penetration by the oviposition punctures of a tinged of the genus *Gargaphia*.

Diseases of buds and blossoms

Insects often overwinter in buds of plants and many also visit blossoms to feed on the nectar they produce. Buds also often contain mycelium and spores of plant pathogenic fungi, and blossoms are often the first plant organ such fungi attack in the spring. Examples of bud infections in which insects have been implicated to play a role include: bud-blast disease of rhododendron, caused by the fungus *Pycnostyamus azalea* and aided by the leafhopper *Graphocephala coccinea*; bud rot disease of carnations caused by the fungus *Fusarium poae* and aided by the mite *Siteroptes graminum*.

Several diseases of blossoms have been associated with insect vectors. In most cases, transmission of the fungus by the insect is related to the activities of the insect during pollination. Some of the better-known examples include:

Anther smut of carnations - This is caused by the fungus *Ustilago violacea*. In this disease, the pollen is replaced by the teliospores of the fungus but the petals remain unaffected and continue to attract insects. The visiting insects become smeared with the smut spores, which they transfer to previously healthy flowers.

Blossom blight of red clover - This is caused by the fungus *Botrytis anthophila* and is transmitted primarily by pollinating bees.

Ergot of cereals and grasses - This is caused by the fungus *Claviceps purpurea*, which develops in the flowers and produces spores that are contained in a sweet and sticky substance. That substance is attractive to many insects, particularly flies and beetles. The insects feed on the spores and also become smeared with them externally and carry them, externally and through their feces, to healthy flowers. Although primary infections by the ergot fungus are primarily from ascospores produced by sclerotia overwintering on the ground and carried via air currents, insect transmission of conidia is important for secondary transmission of the disease and for transmission over long distances. Some beetles, however, feed on ergot sclerotia on the ground and may carry mycelium and ascospores on their bodies to healthy plants and

through them may cause primary infections.

Anthracnose disease of *Musa balsamiana* - This is caused by the fungus *Gloeosporium musarum* and is transmitted by the hymenopterans *Polybia occidentalis*, *Synoeca surinama*, and *Trigona* sp. The fungus infects the floral parts of the plant but these are dropped while still producing conidia and sweet exudate. The insects are attracted to the exudate on the fallen flowers and there they become smeared wit conidia, which they subsequently carry to healthy flowers, which the spores infect.

Flower spot of azalea - This is caused by the fungus *Ovulina azaleae* and is transmitted by several species of bees, thrips, and ants. The insects carry spores on their bodies and drop them off on healthy flowers they visit which, in addition, may injure directly and facilitate penetration and infection by the fungus.

Diseases of fruit and seeds in the field

Fruits and seeds are the source of food and the breeding grounds of many insects. Insects puncture fruit and seeds to obtain food and to lay their eggs in them. Although insects often cause direct damage to fruits and seeds that make them unsalable, the damage increases manifold when the insects also carry to the fruits and seeds fungi that infect and cause these organs to rot or to develop other symptoms. Numerous examples of fruit-insect-pathogen interactions could be cited, although in many cases no hard data of such interactions exist. Some of the better-studied cases are described briefly below.

Rots of fleshy fruits

Fig rots - Several kinds of fungi attack figs and cause rotting of fruits in the field. In most such cases, certain types of insects play a more or less important role in the transmission and introduction of the fungus into the fig.

Endosepsis of figs - This is caused by the fungus *Fusarium moniliforme*, and results in the entire fruit content turning into a pulp. The fungus is transmitted from fruit to fruit by the fig wasp *Blastophaga psenes*, which also plays a crucial role in the pollination of figs. Fig trees, being dioecious, have male trees that produce staminate flowers around the opening and gall flowers in the cavity, and female trees that produce only pistilate flowers. The fig wasp lays its eggs in the ovules of the gall flowers of male plants, which are thereby stimulated to grow. The eggs hatch and the larvae parasitize the galls until they pupate. The adults emerge from the pupae and the females are fertilized while still in the male fig. When they come out of the fig, the females brush against the staminate flowers that surround the opening and become smeared with pollen. The female wasps carry the pollen to male and female flowers they subsequently visit for oviposition. In female flowers, however, because of the length of styles, oviposition fails but pollination is nevertheless successful and the fruit develop into edible figs. If, however, as the female wasp visits some infected figs it becomes smeared with spores of the fungus, it transmits the spores to male and female figs it visits, and the fungus then causes endosepsis of the female figs.

Souring of figs - This is caused by yeast fungi that cause fermentation, and appears as discoloration and

wateriness of the fig contents which then exude from the fig opening. Such figs shrivel, dry, and cling to the tree. The fermenting yeasts are transmitted to figs externally and internally on the bodies of the two most common visitors of figs still in the tree, the sap beetle *Carpophilus hemipterus* and the fruit fly *Drosophila melanogaster*.

Fig smuts and molds - These are caused by the black mycelium and spores of *Aspergillus niger* and the variously colored growths of other fungi. These fungi are carried into green figs on the bodies of predatory mites and, to a lesser extent, by thrips.

Brown rot of stone and pome fruits - This is caused by the three related fungi *Monilinia fructicola*, *M. fructigena*, and *M. laxa* and affects all the stone fruits and, to a lesser extent, the pome fruits. The fungi are aided in their penetration of the fruit by the feeding and oviposition wounds made by the insects plum curculio (*Conotrachelus nenuphar*) and the oriental fruit moth (*Grapholitha molesta*), and the feeding wounds of the dried fruit beetle (*Carpophilus hemipterus*) and two nitidulid beetles (*Carpophilus mutilatus* and *Haptonchus luteolus*). The insects also become smeared with spores of the brown rot fungi which they carry on their bodies and deposit at the wounds they make on the fruits they visit. In pome fruits, the fungus is facilitated in penetrating the fruit by the feeding holes made by the earwig *Forficula auricularia* at the beginning of ripening of the fruit, at which time they are susceptible to brown rot.

Gray mold of grapes - This is caused by the fungus *Botrytis cinerea*. The fungus spores are generally spread by *air* currents. Penetration of the grapes and shoots, however, seem to be increased by the wounds made on them by the larvae of the lepidopterans *Argyrotaenia pulchellana* and *Lobesia botrana*.

Black pod of cacao - This is caused by the fungus *Phytophthora palmivora* and results in devastating losses of yield. Insects of several different families play a role in the transmission of this disease. At least ten species of ants, especially *Crematogaster striatula* and to a lesser extent *Camponotus acvapimensis* and *Pheidole megacephala*, appear to spread the fungus vertically within the tree, especially during the rainy season, when they carry spore-containing soil particles up the cacao tree for nesting purposes. Certain coleoptera, such as the nitidulid beetle *Brachypeplus pilosellus*, and certain dipterans, such as the fly *Chaetonarius latifemur*, colonize black pods in the field and may carry the fungus internally or externally on their bodies to healthy pods. Because of their large numbers on cocoa trees, their habit of visiting wounded pods, and their proven efficiency to transmit the fungus, these insects are considered the main vectors of the fungus locally and over long distance.

Boll rots of cotton - These are caused by several fungi including *Fusarium moniliforme*, *Alternaria tenuis*, *Aspergillus flavus*, and *Rhizopus nigricans*. Various insects are apparently involved in the transmission of these fungi and they seem to use different mechanisms of transmission. Thus, in boll rot due to *Fusarium* and *Alternaria*, the fungi penetrate cotton bolls through feeding and oviposition wounds made by the boll weevil (*Anthonomus grandis*), the cotton bollworm (*Heliothis zeae*), and the

tarnished plant bug, *Lygus lineolaris*, or they are brought to and penetrate through the nectarines by nectar feeding flies such as *Drosophila* and cabbage looper, *Trichoplusia ni*. In boll rots caused by *Aspergillus flavus* and other aflatoxin-producing species, the fungus is primarily wind disseminated but is also carried internally and externally by insects, such as the lygus bug *Lygus hesperus* and the stink bug *Chlorochroa sayi*, that frequently visit cotton bolls. The latter fungus, however, seems to depend for entrance on the presence of large wounds like the large exit holes made by the mature larvae of the pink bollworm, *Pectinophora gossypiella*. On the other hand, boll rots by *Rhizopus stolonifer* occur when wounds made by the bollworm *Earias insulana* and by the pink bollworm are available. In the lint rot of cotton, caused by the fungus *Nigrospora oryzae*, the fungus is transmitted very efficiently by the mite *Siteroptes reniformis*. In Stigmatomycosis or internal boll disease, caused by the fungus *Nematospora gossypol*, the cotton fibers are stained in the absence of external symptoms. This disease is associated with the feeding of several species of plant bugs primarily of the genus *Dysdercus*, often referred to as cotton stainers. The insects carry the fungus spores externally on the mouthparts and internally in their deep stylet pouches and introduce it via their proboscis through the wall of young cotton bolls.

Coffee bean rot - This is caused by the related fungi *Nematospora corylii and N. gossypii*, which cause berries to turn black and subsequently to rot. The fungi are introduced into the berries through the feeding wounds made by the insects *Antestia lineaticolis and A. faceta*. The insects feed on small and large berries and if they carry the fungus the latter causes infection of the bean. The number of infected berries is proportional to the number of insects, approximately 300 insects per tree resulting in infection of all the berries on the tree.

Molds and decays of grains and legumes

Numerous decays and molds affect the various grains and legumes while still in the field and their frequency and severity increase as the number of insects infesting the crops, and feeding on the seeds, increases. In corn, for example, seed rots can be caused by species of the fungi *Fusarium*, *Gibberella*, *Diplodia*, *Cephalosporium*, *Nigrospora*, *Physalospora*, *Cladosporium*, *Penicillium*, *Aspergillus*, *Rhizopus*, *Trichoderma*, and others. The insects most commonly involved in transmitting and facilitating infection of corn kernels by these fungi are the corn earworm, *Heliothis zea*, and the European corn borer, *Pyrausta nubilalis*, but other borers and other insects also play important roles as vectors and, most importantly, as facilitators of infection by these fungi by creating wounds that allow the fungus to enter the seed. In seed infections by *Aspergillus* and by *Fusarium* there is the added adverse effect of production of debilitating mycotoxins. Similar, although less studied situations have been reported for rice infections by fungi, e.g., *Nematospora corylii*, transmitted and facilitated by wounds made by the rice stinkbug *Oebalus pugnax*; wheat and corn infections by *Nigrospora* sp. and *Fusarium poae,* transmitted by large numbers of *Peliculopsis* mites feeding on and transporting spores of the fungus in their

abdominal sacs; and in various legume infections by the fungi *Nematospora*, *Cladosporium*, *Aureobasidium*, etc. transmitted and facilitated in their penetration and infection of the seeds by the stinkbugs *Acrosternum hilare* and *Thyanta custator*, the lygaeid *Spilostethus pandurus*, by thrips, aphids, and other insects.

Molds and decays of harvested fruits and seeds

Generally little is known definitively about the roles of specific insects on the transmission and facilitation of rots of specific fruits and vegetables, and of molds and decays of seeds of specific grains, legumes, or nuts by specific fungi. It is generally accepted, however, that postharvest infections of plant products are greatly increased in numbers and in severity if insects are also present in the same or adjacent containers. There is agreement that insects moving about among stored fruits, seeds, etc., transport externally and internally on their bodies spores of fungi infecting such fruits and seeds and deposit such spores on the next fruit or seed they feed on. There is also agreement that by creating feeding or oviposition wounds on harvested fruit and seeds, the insects create openings through which the fungi can penetrate and release sap and additional nutrients. The fungi then can grow and build momentum to eventually infect and rot the entire fruit or seed.

The fungi that cause most rots of fleshy fruits and vegetable after harvest include *Penicillium*, *Fusarium*, *Botrytis*, *Rhizopus*, *Alternaria*, *Sclerotinia*, *Monilinia*, and *Colletotrichum*, while the molds and decays of grains and legumes involve primarily *Aspergillus*, *Fusarium*, and *Penicillium*. The insects involved in transmission and facilitation of infection of fleshy organs after harvest include larvae and adults of various Lepidoptera such as the oriental fruit moth, *Grapholitha molesta*, Diptera such as the apple maggot, *Rhagoletis pomonella*, the Mediterranean fruit fly, *Ceratitis capitata*, the house fly, and others. The insects involved in the transmission and facilitation of infection by fungi causing molds and decays of grains and legumes are the larvae and adults of various Coleoptera such as the rice weevil *Sitophilus oryzae*, the granary weevil *Sitophilus granarius*, and the confused grain beetle, *Tribolium confusum*, and also Lepidoptera such as the Angoumois grain moth, *Sitotroga cerealella*, the European corn borer, *Pyrausta nubilalis*, the ear cornworm, *Heliothis zea*, and other insects.

Insect transmission of plant pathogenic nematodes

Two very serious plant diseases caused by nematodes of the genus *Bursaphelenchus* are transmitted by insects. In both diseases there is a symbiotic relationship between the fungal pathogen and the insect vector.

Pine wilt - This is a lethal disease of many species of pines and other conifers. It is caused by the nematode *Bursaphelenchus xylophilus*, known as the pinewood nematode. The nematode is about 800 μm long by 22 μm in diameter and it develops and multiplies rapidly, each female laying about 80 eggs and completing a life cycle in as short as 4 days. The nematode produces the four juvenile stages and the adults. The juvenile stages develop in the resin canals of infected pine trees, feeding at first on plant cells and later on fungi that invade the dying or dead tree. Later, the

nematode produces special fourth-stage dispersal juveniles that are adapted to survive in the respiratory system of the cerambycid beetles *Monochamus carolinensis* and *M. alternatus* by which they are transmitted to healthy pine trees.

The pinewood nematode overwinters in the wood of infected dead trees, which also contain larvae of the beetle vectors of the nematode. Early in the spring, the larvae dig small cavities in the wood in which they pupate. As the adult beetles emerge from the pupae later in the spring, large numbers of fourth-stage juvenile nematodes enter the beetles and almost fill the tracheae of the respiratory system of each insect with about 15,000 to 20,000 juveniles. These nematode-carrying adult beetles emerge and fly to young branch tips of healthy pine trees where they feed for several weeks. As the beetles strip the bark and reach the cambium, the nematode juveniles emerge from the insect and enter the pine tree through the wound. The juveniles in the tree then undergo the final molt and produce adult nematodes. The latter migrate to the resin canals, feed on their cells and cause their death, and then they move in the xylem and in the cortex where thy reproduce quickly and build enormous populations of nematodes and kill twigs, branches and entire trees.

After the adult *Monochamus* beetles, the vector of the pine wilt nematode, have fed on young twigs for about a month, they are ready to breed and look for stressed and dead pine trees, including trees showing symptoms or dying from infection by the pinewood nematode. The female beetles deposit their eggs under the bark of such trees where the first two instars develop and feed. The third instar penetrates the wood where it undergoes the next molt and produces the fourth instar, which overwinters there. In early spring, the fourth instar digs a cavity in the wood where it pupates and to which numerous third-stage nematode juveniles are attracted and congregate. The juvenile nematodes undergo the next molt and produce the fourth-stage dispersal juveniles, which by the thousands infect the tracheae of the adult insects as soon as they emerge from the pupae and are carried by them to healthy pine trees, thus completing the cycle.

Red ring of coconut palms - This disease kills coconut palm trees from Mexico to Brazil and in the Caribbean islands. It is caused by the nematode *Bursaphelenchus cocophilus*, which is transmitted from palm to palm by the American palm weevil, *Rhynchophorus palmarum*, the sugarcane weevil, *Metamasius* sp., and probably other weevils. The nematodes infect, discolor, and kill the palm tissues in a ring 3 to 5 cm wide about 5 cm inside the stem periphery over the length of the stem.

The nematode pathogen lays its eggs and produces all its juvenile stages and the adults inside infected palm trees, completing a life cycle in about 9 to 10 days. Female weevil vectors are attracted to red ring-diseased trees but they also lay eggs on healthy or wounded palm trees. If the female carries red ring nematodes, it deposits them in its feeding wounds at bases of leaves or at internodes. The nematodes then enter the palm tissues and undergo repeated life cycles and spread intercellularly in the parenchyma cells of the petioles, stem, and roots, where the cells break down and form a flaky, orange to red discolored tissue with

cavities. Red ring nematodes do not invade xylem and phloem tissues but cause tyloses to develop in xylem vessels within the red ring that block the upward movement of water and nutrients. In the meantime, the weevil larvae of the insect vector feed on the red ring tissue and swallow several hundred thousand nematode third-stage juveniles. Of these, however, only a few hundred of the nematodes survive and pass through the molt, internally or externally, to the next stage weevil larvae and to the adult weevil. As weevil females emerge from rotted palms, a small percentage of them carry with them third-stage juveniles of the nematodes. Nematode populations increase rapidly at first but later they decline and about 3 to 5 months after infection there are hardly any red ring nematodes or their eggs left in decomposed stem tissue of infected, dead palm trees. The nematodes, however, survive in newly infected palm trees and, briefly, in their insect vector.

Insect transmission of plant pathogenic protozoa

Three plant diseases: phloem necrosis of coffee, heartrot of coconut palms, and sudden wilt of oil palms, are caused by flagellate protozoa of the genus *Phytomonas*. In all three diseases, protozoa invade the phloem elements of infected plants and multiply in them, reaching populations of varying densities. Some of the sieve tubes become plugged by protozoa. Generally, the more severe the symptoms of infected plants, the higher the populations of protozoa in their phloem. The pathogen is transmitted from infected to healthy plants occasionally through natural root grafts, and primarily by stink bugs (Pentatomidae) such as the genera *Lincus* and *Oclenus*, and possibly others.

Insect transmission of plant pathogenic viruses

Plant viruses cause many and severe diseases of plants, their number and importance being second only to fungal diseases of plants. Most viruses infect their host plants systemically, that is, the virus multiplies internally throughout the plant. Almost all viruses enter and multiply in phloem and in parenchyma cells. Viruses do not produce spores, nor do they come to the surface of the plant. All plant viruses are transmitted to new plants that are propagated from infected plants vegetatively (that is, by grafting or budding, by cuttings, by bulbs, corms, roots, tubers, etc.), and many can be transmitted artificially by mechanical inoculation, that is, by rubbing sap from infected plants onto leaves of healthy plants. Some plant viruses can be transmitted from diseased to healthy plants by pollen or seed produced by infected plants, some by the parasitic higher plant dodder when it is infecting both virus-infected and healthy plants, and some plant viruses are transmitted from plant to plant by certain plant pathogenic fungi, nematodes, or certain mites. More than half of the plant viruses, numbering more than 400, are transmitted from diseased to healthy plants by insects.

The number of insect groups that are vectors of plant viruses is relatively small. The most important vector groups, with the number of vector species and viruses transmitted, are listed below. Hemiptera, which includes the aphids (Aphididae, 192 species, 275 viruses), leafhoppers (Cicadellidae, 49

species, 31 viruses), the planthoppers (Fulgoroidea, 28 species, 24 viruses), the whiteflies (Aleurodidae, 3 species, 43 viruses), the mealybugs (Pseudococcidae, 19 species, 10 viruses), and some treehoppers (Membracidae, 1 species, 1 virus), contain by far the largest number and the most important insect vectors of plant viruses, but the true bugs (Hemiptera, 4 species), the thrips (Thysanoptera, 10 species, 11 viruses) and the beetles (Coleoptera, 60 species, 42 viruses) also are implicated. Grasshoppers (Orthoptera, 27 species) seem to occasionally carry and transmit a few viruses. Unquestionably, the most important virus vectors are the aphids, leafhoppers, whiteflies, and thrips. These and the other groups of Hemiptera have piercing and sucking mouthparts, although several thrips have rasping, sucking ones. Beetles and grasshoppers have chewing mouthparts, but many beetles are quite effective vectors of certain viruses. Generally, viruses transmitted by one type of vector are not transmitted by any other type of vector.

Aphids and aphid-transmitted viruses

Aphids have evolved as the most successful exploiters of plants as a food source, particularly in the temperate regions. Many species of aphids alternate between a primary and a secondary host, although there are many variations of aphid life cycles depending on the aphid species and on climate. Some aphids overwinter as parthenogenetic viviparous forms while others go through their life cycle on one host species or on several related species. On the other hand, there are several aphid species, such as *Myzus persicae*, that have as many as 50 primary and alternate species of host plants.

Aphids have mouthparts that consist of two pairs of flexible stylets held within a groove of the labium. During feeding, the stylets are extended from the labium and, through a drop of gelling saliva, the stylets rapidly penetrate the epidermis. Penetration may stop at the epidermis or it may continue into the middle layers of leaf cells with a sheath of saliva forming around the stylets. The stylets move between the cells until they reach and enter a phloem sieve tube from which the aphids obtain their food. Individual aphids vary in their ability to transmit the virus to individual plants. Infection of a plant with a virus often makes the plant more attractive for aphids to grow on and to reproduce. Both acquisition and transmission of virus by aphids are affected by temperature, humidity and light.

Virus-vector relationships

Insect vectors that have sucking mouthparts carry plant viruses on their stylets, and such viruses are known as stylet-borne, externally borne, or non-circulative, because they do not pass to the vector's interior. The remaining viruses are taken up internally within the vector and are called internally borne persistent circulative or persistent propagative viruses.

Stylet-borne non-persistent transmission - Most externally borne viruses can be transmitted in the typical stylet-borne non-persistent manner. In such a transmission the virus is assisted in its transmission by a specific configuration of its coat protein or by a non-structural virus-encoded protein. The insect acquires the virus from the plant by feeding on it for only seconds

or, at most, minutes. The insect can transmit the virus immediately after the acquisition feeding, that is, without any incubation period required for transmission. The insect retains the virus and is usually able to transmit it for only a few minutes after it acquired it. Most of the nearly 300 known aphid-borne plant viruses are stylet-borne non-persistent. Some of the most important groups of plant viruses, such as those in the genera *Potyvirus*, *Cucumovirus*, *Alfamovirus*, and the *Caulimovirus* transmitted by *Myzus persicae* are stylet-borne non-persistent viruses. In the few seconds in which aphids acquire the virus, the aphid stylet usually penetrates only the epidermal cell. Actually, deeper penetration of the stylet into leaf tissues reduces the ability of aphids to transmit the virus. Aphids vary greatly in their ability to transmit viruses, each particular virus being transmitted by one or a few species of aphids. Sometimes, certain virus strains are transmitted by distinct aphid species. Also, even individual aphids in a population vary in their ability to transmit the same virus, some of them being incapable of transmitting the virus.

All non-persistently transmitted viruses have simple structures of elongated or isometric particles with the nucleic acid encapsidated by one or more kinds of coat proteins. In some viruses, the coat protein interacts directly with the binding site of virus retention in the aphid. In other viruses, the virus encodes a non-structural protein which interacts with the aphid-virus retention binding site and forms a bridge between the virus and the aphid stylet. However viruses are bound to the aphid stylet, there must also be a mechanism for release of the virus when the aphid feeds on the next plant. It appears that saliva alone may carry out this function.

Semi-persistent viruses - Some externally borne non-persistent viruses are known as semi-persistent because they reach but do not seem to go past the foregut of the vector; the vector must feed on an infected plant (acquisition period) for several minutes or hours before it can transmit the virus; and the vector can then retain (retention time) and transmit the virus to healthy plants for several hours. Semi-persistent viruses are also assisted in their transmission by a transmission helper protein or coat protein configuration. The best known semi-persistent viruses are caulimoviruses, which occur in most cell types, and the closteroviruses beet yellows virus and curly top virus, which are found primarily in phloem cells. In several of the semi-persistent viruses, a helper component seems to be involved in their transmission. In cauliflower mosaic virus, the helper component consists of two non-capsid proteins, one of which is associated with the virus particles and the other has two binding domains that interact strongly with microtubules. In some cases, certain viruses can be transmitted only in the presence of a second virus which acts as the helper virus.

Persistent viruses - Internally borne viruses are either persistent circulative or persistent propagative. Persistent circulative viruses are acquired from the plant by the vector after an acquisition feeding period of several hours to several days, and then they are retained by the insect vector and can be transmitted by it for several days or weeks. Persistent circulative viruses require a latent period of several hours to several days beyond the acquisition time before they can be

transmitted by the insect vector, they reach the hemolymph of the vector, and pass through the various stages of the insect, but not through the ovaries to the egg. Persistent propagative viruses are acquired by the insect after a feeding period of several hours to several days, are retained by the vector for several weeks to several months, they multiply in the vector, they have a latent period of a few to several weeks, and can pass through the various stages of the insect, including transovarial passage to the egg. Persistent viruses are generally transmitted by one or a few species of aphids and cause symptoms characterized by leaf yellowing and leaf rolling.

Persistent circulative viruses - These include primarily the luteoviruses, such as barley yellow dwarf virus, and the nanoviruses, such as banana bunchy top virus. The luteoviruses are acquired after a feeding period as short as 5 minutes but it usually takes 12 hours. After an incubation period of an additional 12 hours, the vector can transmit the virus within a 10 to 30 minute inoculation feeding and can continue transmitting it for several days or a few weeks. In the vector, the virus particles seem to associate only with the hind gut of the aphid, entering its cells by endocytosis into coated pits and vesicles and accumulating in tubular vesicles and lysosomes. Virus particles are then released into the hemolymph by fusion of the vesicles with the plasmodesmata and enter the salivary glands of the aphid via invaginations with two plasma membranes on the hemocoel side of the salivary gland accessory cells. It appears that persistent circulative viruses do not require a non-capsid protein for helper component but they require a protein produced via a read-through of the coat protein stop code if they are to advance beyond the hemocoel. Some persistent circulative viruses also require a helper virus to be present for them to be transmitted by their aphid vector.

Persistent propagative viruses - Propagative viruses are transmitted primarily by leafhoppers and planthoppers but several members of the Rhabdoviridae multiply in and are transmitted by their aphid vector. These bacilliform viruses replicate in the nucleus and the cytoplasm of cells in the brain, salivary glands, ovaries, and muscle of the insect vector. The virus goes through the egg to about 1% of the nymphs. Infection of aphids with rhabdoviruses results in increased mortality of the aphids.

Leafhoppers and planthoppers, and transmission of plant viruses

Leafhoppers lay eggs that hatch to nymphs which pass through several molts before becoming adults. Some of them overwinter as eggs, some as adults, and some as immature forms. They all feed by sucking sap from phloem elements of plants. Their feeding behavior is similar to that of aphids in that the mouthparts, surrounded by the salivary sheath, penetrate the phloem of host plants.

Virus-vector relationships

All hopper-transmitted viruses are persistent circulative or persistent propagative, and are transmitted by only one or by a few closely related species of the hopper vectors. Only two of the 60 sub-families of leafhoppers (Cicadellidae) contain species that are vectors of viruses: the Agalliinae feed on herbaceous dicotyledonous hosts and the Deltocephalinae that feed on

monocots. Of the 20 planthopper families (Fulgoroidea), only one, the Delphacidae, have species that are vectors of viruses all of which infect monocotyledonous plants and many of them cause severe diseases on cereal crops such as rice, wheat, and corn.

Semi-persistent transmission - Two viruses, maize chlorotic dwarf virus (MCDV) and rice tungro spherical virus (RTSV), are acquired by their vectors (*Graminella nigrifrons* and *Nephotettix virescens*, respectively) from their hosts within about 15 minutes and are retained by their vectors for one to a few days. MCDV particles have been seen in the foregut and a few other tissues but not beyond. Hoppers egest material from the foregut once in a while during feeding and it is thought that transmission occurs during this ingestion-egestion process.

Persistent transmission - This involves the internal movement of the virus obtained from the plant to the salivary glands of the insect vector. Some of these viruses are circulative while others are propagative.

Circulative viruses - Only two genera of geminiviruses (*Mastrevirus* and *Curtovirus*) are transmitted by leafhoppers in the persistent circular manner. The viruses are acquired by the vector after feeding for a few seconds to an hour. There is a latent period of about a day, presumably for the virus to reach the salivary glands. The internal movement of these viruses is determined by the viral coat protein and by receptor-mediated endocytosis.

Propagative viruses - There are four families and genera of plant viruses that replicate within the cells of their insect vectors as well as the cells of their host plants. Two of these families, Rhabdoviridae and Reoviridae, contain viruses that infect animals, and their virus members that infect plants have been considered as animal viruses that infect plants. The propagative viruses have a latent period of about two weeks. During this period the virus replicates and invades most tissues of the insect vector. When the virus reaches the salivary glands of the vector, the latter can transmit the virus to new plants and can continue to transmit it for the rest of their life. Only a small percentage of the hoppers feeding on infected plants become vectors and of these only about 1% pass the virus through their eggs to the next generation. Various capsid proteins seem to be necessary for passage of viruses through the organs of the vector and are required for transmission.

The two genera that have propagative viruses are *Tenuivirus*, members of which are transmitted by delphacid planthoppers, and *Marafivirus*, which is vectored by the leafhopper *Dalbulus maydis*. These viruses have an acquisition feeding period of 15 minutes to 4 hours, a latent period of 4-31 days, inoculation periods as short as 30 seconds, and can transmit the virus for as long as they live. Almost all of these viruses are transmitted transovarially to the egg.

Whitefly transmission of plant viruses

Whiteflies transmit the viruses in the genus *Begomovirus* of the family Geminiviridae, and all the viruses in the genus *Crinivirus* and some in the genus *Closterovirus* of the family Closteroviridae. Whitefly adults are winged but only the first instar among the larvae is mobile. Whiteflies produce many generations in a year and reach high populations. Only a few species of

whiteflies transmit viruses, mostly in the tropics and subtropics, but the viruses they transmit cause very severe diseases. Begomoviruses are transmitted by *Bemisia tabaci* whiteflies, while the criniviruses and the whitefly-transmitted closteroviruses are vectored by the whiteflies *Trialeuroides vaporariorum*, *T. abutilonea*, *B. tabaci*, and the type B of *B. tabaci* (also referred to as *B. argentifolii*). Whitefly mouthparts and feeding behavior resemble those of aphids.

Begomoviruses are bipartite geminiviruses and are transmitted by whiteflies in the persistent circulative manner. A helper factor coded by the virus seems to be involved in the transmission. The whitefly-transmitted monopartite closteroviruses and the bipartite criniviruses reach only the foregut of the vector and are transmitted in the semi-persistent manner. These viruses are retained in the vector for about 3-9 days. Two capsid proteins help the virus in its transmission by the vector.

Thrips transmission of plant viruses

About 10 species of thrips of the family Thripidae are the vectors of about a dozen viruses belonging to four genera (*Carmovirus*, *Ilarvirus*, *Sobemovirus*, and *Tospovirus*) of four families. Thrips are polyphagous insects that have many hosts. Some of the vector species reproduce mainly parthenogenetically. The larvae are rather inactive but the adults have wings and are very active. Thrips adults feed by sucking the contents of subepidermal cells. Adults live up to 3 weeks and there may be as many as 20 generations per year. The tospoviruses are transmitted in the persistent propagative manner, while the viruses of the other genera are transmitted in the pollen carried by the thrips vectors and by mechanical damage during feeding of the vector.

By far the most important thrips-transmitted viruses are the tospoviruses, which include the widespread and severe tomato spotted wilt virus and the impatience necrotic spot virus. In tospoviruses, only the larvae but not the adults can acquire the virus, and their ability to acquire it decreases with age. Larvae sometimes acquire the virus after feeding on a diseased plant for as little as 5 minutes, but usually they must feed for more than an hour both in acquiring and in inoculating the virus. There is a latent period of 3-4 days before the larvae can transmit the virus. The virus is passed from the larvae to the adults which can transmit it, although erratically, for as long as they live. These viruses appear to multiply in the vector but are not passed through the egg. Several structural proteins of the virus seam to be associated with the acquisition, passage through, and inoculation of the virus by its larval and adult insect vector.

Mealybug and other bug transmission of plant viruses

Mealybugs are important as virus vectors primarily on some perennial plants in the tropics and subtropics. They move slowly on plants and therefore are not as efficient virus vectors as those discussed previously. They move from plant to plant, mostly as crawling nymphs, through leaves of adjacent plants being in contact with each other; by ants tending the mealybugs and moving them from one plant to the other; and occasionally by wind.

Mealybugs feed on the phloem and they are vectors of the badnaviruses, such as the cacao swollen shoot virus (CSSV), several closteroviruses, such as grapevine leafroll associated viruses and the pineapple mealybug wilt associated virus, and the trichoviruses, such as grape viruses A and B. Mealybugs acquire the viruses after feeding on diseased plants for only a few, about 20, minutes and retain the virus for a few, up to 24 hours, so the transmission resembles the non-persistent or semi-persistent mechanism of transmission by aphids.

Other bugs that transmit plant viruses include the mirid bugs, which transmit some sobemoviruses in manners that have characteristics of non-persistent, semi-persistent, and beetle-like transmission, and the piesmatid bugs, which transmit beet leaf curl virus in a persistent propagative manner.

Virus transmission by insects that have biting/chewing mouthparts

Although there are a few vectors in the orders Orthoptera and Dermaptera, there are more than 60 vector species in the order Coleoptera (beetles), 30 of them in the family Chrysomelidae. Most beetle vectors tend to eat plant cells between the leaf veins and regurgitate during feeding, thereby bathing their mouthparts with sap and virus. Virus transmission by beetles, however, is specific between each virus and its vector. Beetle-transmitted viruses belong to the genera *Tymovirus*, *Comovirus*, *Bromovirus*, and *Sobemovirus*. Most of these viruses are small (25 to 30 nm in diameter), stable, reach high concentrations, and are easily transmitted by sap. These viruses can also be translocated through the xylem of the plant. Beetles can acquire and can transmit the virus after feeding for a few seconds and they can retain the virus from 1 to 10 days.

Virus transmission by mites

Several members of the mite family Eriophyidae transmit viruses of the genus *Rymovirus* which cause many serious diseases in grain crops. Two mite species of the family Tetranychidae transmit two plant viruses, one of them transmitting the peach mosaic virus. All mites in these families feed by piercing plant cells and sucking their contents.

Eriophyid mites are small (0.2 mm long), move little by themselves and, instead, they are spread by wind. They have two nymphal instars followed by a resting pseudopupa. They complete a life cycle within two weeks. Mites can acquire virus from infected host plants within 15 minutes from the start of feeding and can transmit it to healthy plant within a similar duration. Mites acquire the virus as nymphs but not as adults. They carry the virus through molts and remain infective for 6 to 9 days.

Tetranychid mites are larger (0.8 mm long). Pre-adult mites readily acquire the virus and they, as well as the adults, transmit the virus efficiently.

Virus transmission by pollinating insects

Honey bees and other pollinating insects seem to play a role in distributing virus-infected pollen from infected plants to healthy ones. It appears, however, that no special mechanisms or involvement of the insect are present in such virus transmission.

Summary

Insects play various roles in the transmission of plant pathogens, and in the initiation and development of disease in plants. In some diseases, the insects incidentally carry pathogens on their bodies or in their feces and deposit them on healthy plants where they cause disease, without developing any special relationships with the pathogens. In several cases, the insects weaken the plants on which they feed and make them much more susceptible to attack by pathogens. In other cases, the pathogens depend on the insects to carry them to healthy plants and to deposit them on fresh wounds through which they penetrate and infect the plants. While pathogens seem to be the beneficiaries of these actions, insects also derive advantages by the pathogen making the diseased plant more attractive to the insect for feeding or breeding purposes, and in some cases, by the insect feeding on the pathogen growing in the cavities made by its insect vector. Also, while in most cases the pathogen does not affect its insect vector directly, there are several plant viruses and mollicutes that multiply in the insect vector as well as in their plant host, and such vector insects often show histopathological symptoms, reduced reproduction, and shorter life span. Most of the insect/pathogen associations are highly specific and involve sophisticated molecular mechanisms that regulate the uptake, retention, and transmission of the pathogen by its insect vector.

See also, Plant Viruses and Insects, Management of Insect-Vectored Pathogens of Plants, Transmission of *Xylella Fastidiosa* Bateria by Xylem-Feeding Insects, Vectors of Phytoplasmas.

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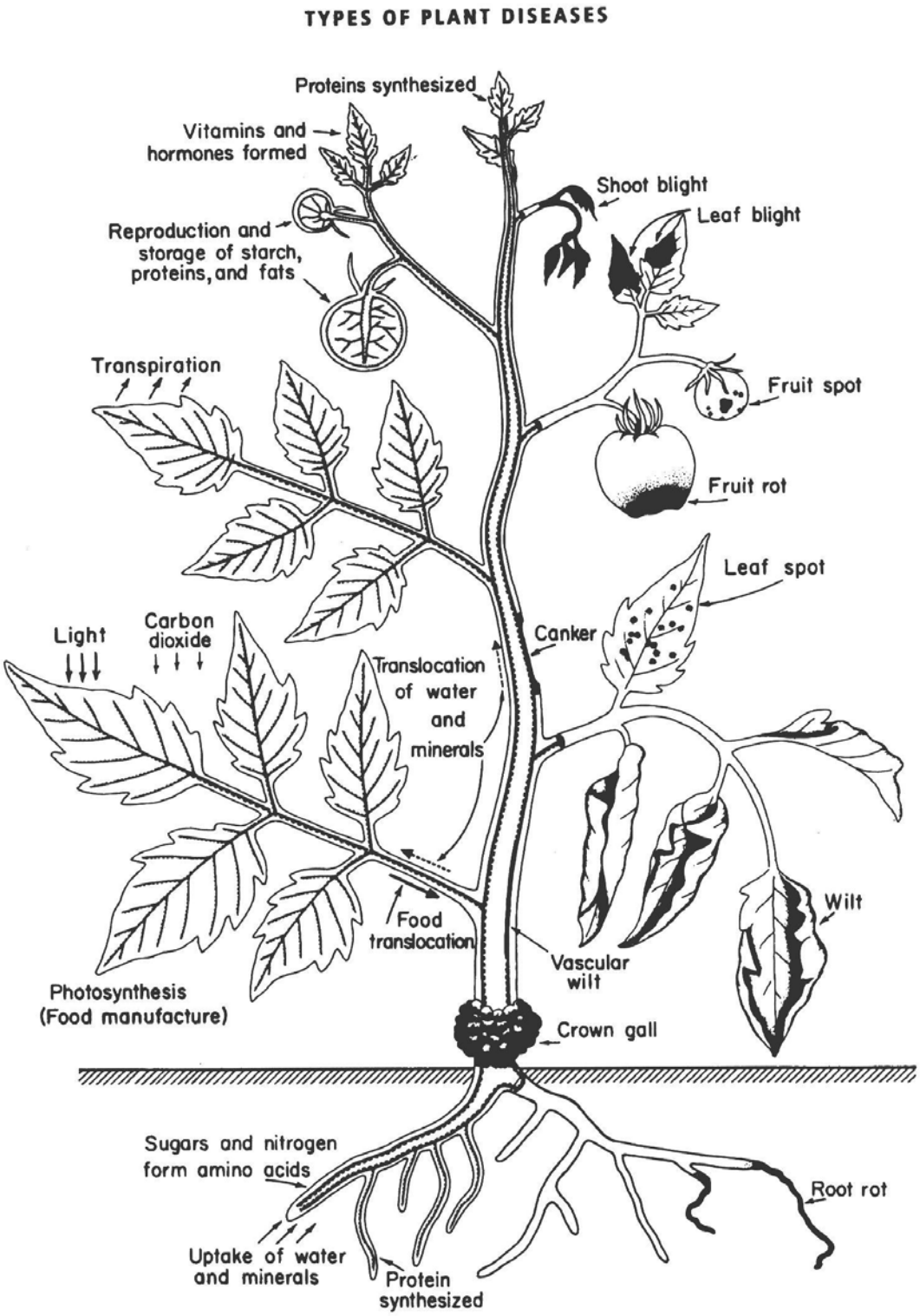

Figure 1. Schematic representation of the basic functions in a plant (left)and the interference with these functions (right) caused by some common types of plant diseases. (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)

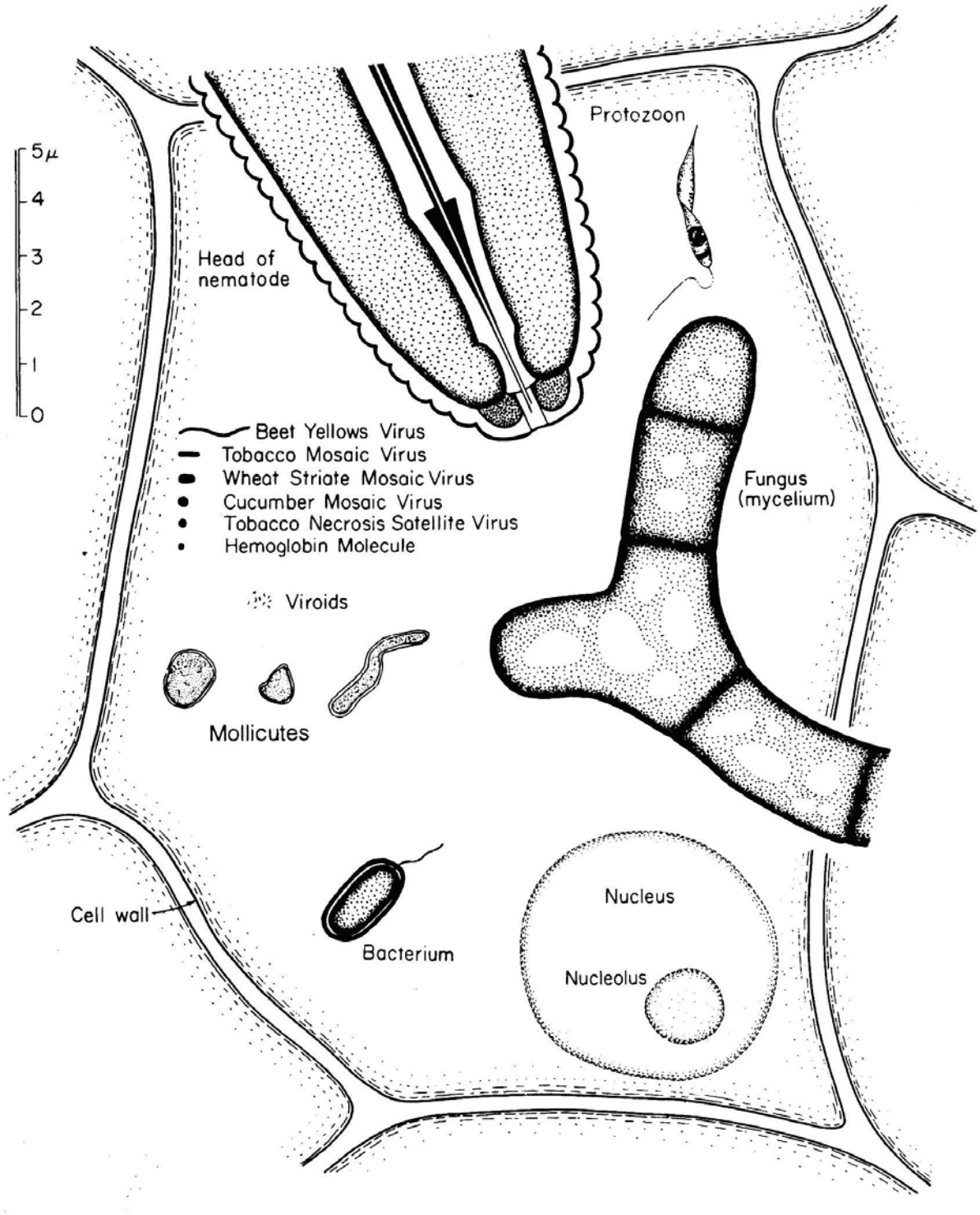

Figure 2. Schematic diagram of the shapes and sizes of certain plant pathogens in relation to a plant cell. (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)

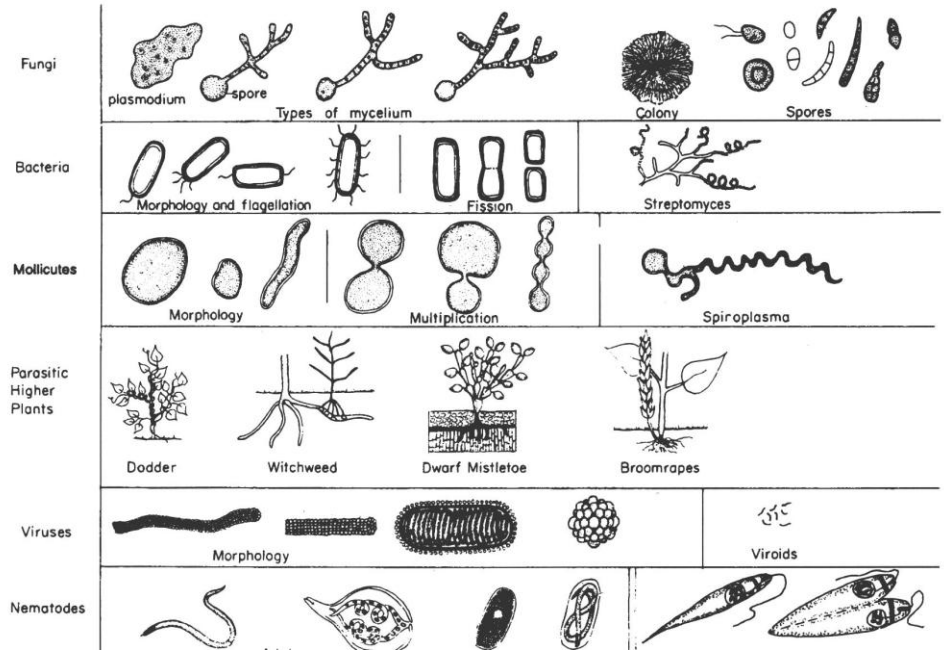

Figure 3. Morphology and multiplication of some of the groups of plant pathogens. (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)

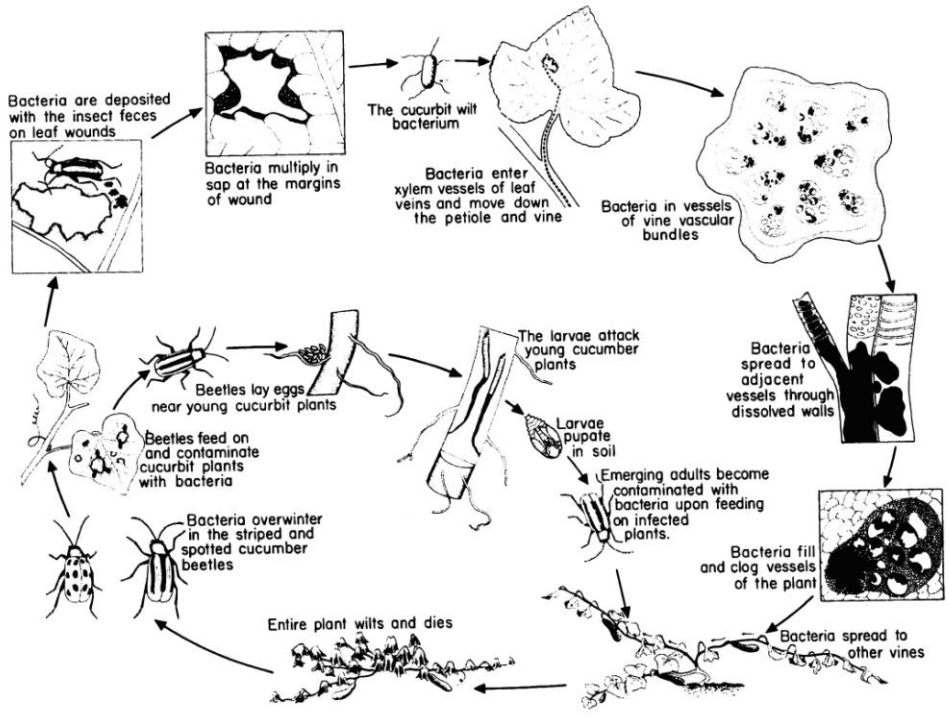

Figure 4. Disease cycle of bacterial wilt of cucurbits caused by *Erwinia tracheiphila* and transmitted by the striped cucumber beetle (*Acalymma vittatum*). (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)

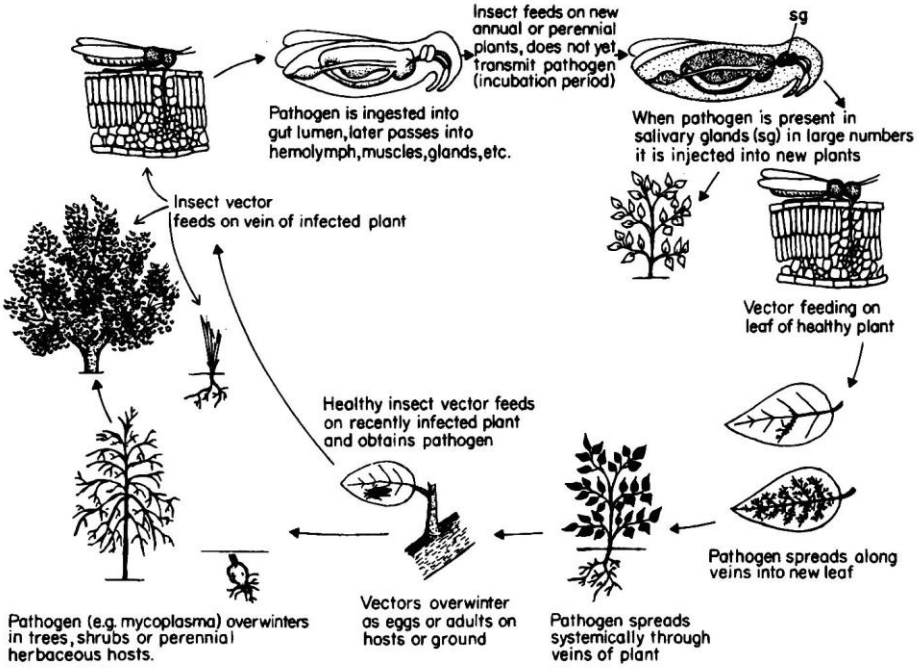

Figure 5. Sequence of events in the overwintering, acquisition, and transmission of plant viruses, mollicutes, and fastidious bacteria by leafhoppers. (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)

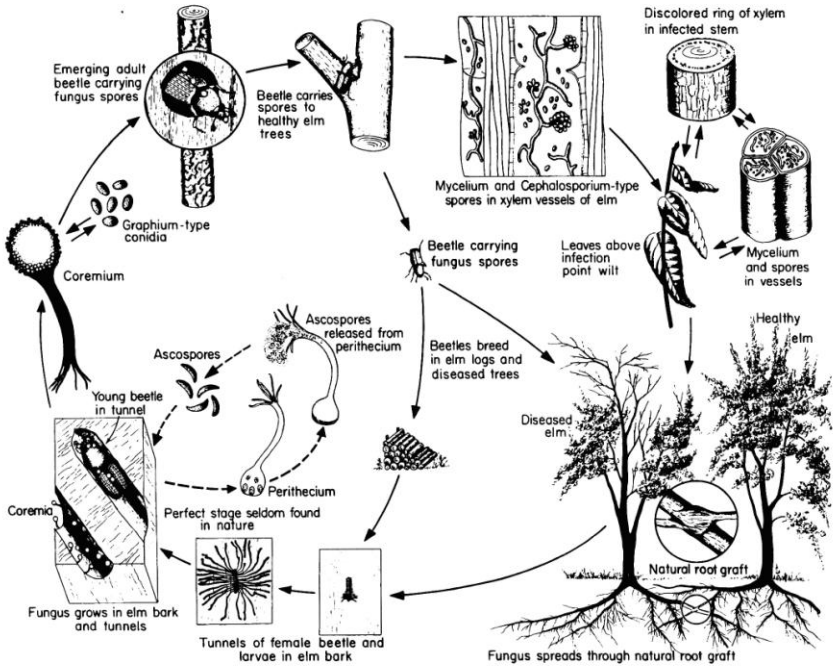

Figure 6. Disease cycle of Dutch elm disease caused by the fungus *Ophiostoma ulmi* and transmitted by the European and the American elm bark beetles. (From Agrios, G.N. 1997. Plant pathology (4th ed.). Academic Press, San Diego, California.)