

# EFFECTS OF PATHOGENS ON PHYSIOLOGICAL ACTIVITIES OF PLANTS

## Abstract

In plants infected by many vascular pathogens, stomata remain partially closed, chlorophyll is reduced, and photosynthesis stops even before the plant eventually wilts. Most virus, mollicute, and nematode diseases also induce varying degrees of chlorosis and stunting. Pathogens can directly affect plant growth and fitness by modifying resource availability, metabolism and physiology of their host plant. Infected plants are expected to have reduced resources for growth and reproduction as defence is costly and pathogens withdraw nutrients from the host. However, due to a diversity of molecular and physiological plant responses upon pathogen infection. The strength and direction of effects of pathogens on plant growth and fitness vary greatly. In most cases the relationship between the symptoms of the plant and the physiological functions affected is obvious and understandable.

**Keywords:** Physiological, Photosynthesis, Transpiration, Host Plant Respiration, Transcription, Translation, Plant Growth.

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## I. INTRODUCTION

The pathogens infect plants in the course of their obtaining food for themselves, depending on the kind of pathogen and on the plant organ and tissue they infect, pathogens interfere with the different physiological function(s) of the plant and lead to the development of different symptoms. Thus, a pathogen that infects and kills the flowers of a plant interferes with the ability of the plant to produce seed and multiply. A pathogen that infects and kills part or all of the roots of a plant reduces the ability of the plant to absorb water and nutrients and results in its wilting and death. Similarly, a pathogen that infects and kills parts of the leaves or destroys their chlorophyll leads to reduced photosynthesis, growth, and yield of the plant, and so forth. In most cases the relationship between the symptoms of the plant and the physiological functions affected is obvious and understandable. In other cases, however, the relationship of the two is more complex and the explanation is not always straightforward.

## II. EFFECT OF PATHOGENS ON PHOTOSYNTHESIS

**Photosynthesis** is the basic function of green plants: it enables them to transform light energy into chemical energy, which they can utilize in all cell activities. Photosynthesis is the ultimate source of nearly all energy used in all living cells, plant or animal, as all activities of living cells, except photosynthesis, expend the energy provided by photosynthesis. In photosynthesis, carbon dioxide from the atmosphere and water from the soil are brought together in the chloroplasts of the green parts of plants and, in the presence of light, react to form glucose with a concurrent release of oxygen. In view of the fundamental position of photosynthesis in the life of plants, it is apparent that any interference by pathogens with photosynthesis results in a diseased condition in the plant. That pathogens do interfere with photosynthesis is obvious from the chlorosis they cause on many infected plants, from the necrotic lesions or large necrotic areas they produce on green plant parts, and from the reduced growth and amounts of fruits produced by many infected plants. In leaf spot, blight, and other kinds of diseases in which there is destruction of leaf tissue, e.g., in cereal rusts and fungal leaf spots, bacterial leaf spots, viral mosaics and yellowing and stunting diseases or in defoliations, photosynthesis is reduced because the photosynthetic surface of the plant is lessened. Even in other diseases, however, plant pathogens reduce photosynthesis, especially in the late stages of diseases, by affecting the chloroplasts and causing their degeneration. The overall chlorophyll content of leaves in many fungal and bacterial diseases is reduced, but the photosynthetic activity of the remaining chlorophyll seems to remain unaffected. In some fungal and bacterial diseases, photosynthesis is reduced because the toxins, such as tentoxin and tabtoxin, produced by these pathogens inhibit light chlorophyll. Some of the enzymes that are involved directly or indirectly in photosynthesis. In plants infected by many vascular pathogens, stomata remain partially closed, chlorophyll is reduced, and photosynthesis stops even before the plant eventually wilts. Most virus, mollicute and nematode diseases also induce varying degrees of chlorosis and stunting. In the majority of such diseases, the photosynthesis of infected plants is reduced greatly. In advanced stages of disease, the rate of photosynthesis is no more than one-fourth the normal rate.



(A)

(B)



(C)

(D)



(E)

(F)

Pathogens reduce photosynthetic area. (A) Spots on barley leaves caused by the fungus *Rhynchosporium* sp. (B) Nearly complete destruction of pumpkin leaves downy mildew *Pseudoperonospora cubensis*. (C) lesions on stems and leaves of wheat stem rust fungus *Puccinia graminis* f.sp. *tritici* (D) Angular leaf spots on cucumber leaf bacterium *Pseudomonas lacrymans*. (E) Reduced chlorophyll with *cowpea chlorotic mottle virus* or (F) stunting and yellowing of rice plants *rice tungro virus*.

- 1. Pathogens on Translocation of Water and Nutrients in the Host Plant:** All living plant cells require an abundance of water and an adequate amount of organic and inorganic nutrients in order to live and to carry out their physiological functions. Plants absorb water and inorganic (mineral) nutrients from the soil through their root system. These substances are generally translocate upward through the xylem vessels of the stem and into the vascular bundles of the petioles and leaf veins, from which they enter the leaf cells. Minerals and part of the water are utilized by the leaf and other cells for the synthesis of the various plant substances, but most of the water evaporates out of the leaf cells into the intercellular spaces and from there diffuses into the atmosphere through the stomata. However, nearly all organic nutrients of plants are produced in the leaf cells, following photosynthesis, and are translocated downward and distributed to all the living plant cells by passing, for the most part, through the phloem tissues. When a pathogen interferes with the upward movement of inorganic nutrients and water or with the downward movement of organic substances, diseased conditions result in the parts of the plant denied these materials. The diseased parts, in turn, will be unable to carry out their own functions and will deny the rest of the plant their services or their products, thus causing disease of the entire plant. For example, if water movement to the leaves is inhibited, the leaves cannot function properly, photosynthesis is reduced or stopped, and few or no nutrients are available to move to the roots, which in turn become starved and diseased and may die.
- 2. Interference with Upward Translocation of Water and Inorganic Nutrients:** Many plant pathogens interfere in one or more ways with the translocation of water and inorganic nutrients through plants. Some pathogens affect the integrity or function of the roots, causing them to absorb less water; other pathogens, by growing in the xylem vessels or by other means, interfere with the translocation of water through the stem; and, in some diseases, pathogens interfere with the water economy of the plant by causing excessive transpiration through their effects on leaves and stomata.
- 3. Absorption of Water by Roots:** Many pathogens, such as damping-off fungi, root-rotting fungi and bacteria, most nematodes, and some viruses, cause an extensive destruction of the roots before any symptoms appear on the aboveground parts of the plant. Some bacteria and nematodes cause root galls or root knots which interfere with the normal absorption of water and nutrients by the roots. Root injury affects the amount of functioning roots directly and decreases proportionately the amount of water absorbed by the roots. Some vascular parasites, along with their other effects, seem to inhibit root hair production, which reduces water absorption. These and other pathogens also alter the permeability of root cells, an effect that further interferes with the normal absorption of water by roots.



**4. Translocation of Water through the Xylem:** Fungal and bacterial pathogens that cause damping off, stem rots and cankers may reach the xylem vessels in the area of the infection and, if the affected plants are young, may cause their destruction and collapse. Cankers in older plants, particularly older trees may cause some reduction in the translocation of water, but, generally, do not kill plants unless the cankers are big or numerous enough to encircle the plant. In vascular wilts, however reduction in water translocation may vary from little to complete. In many cases, affected vessels may be filled with the bodies of the pathogen) and with substances secreted by the pathogen or by the host in response to the pathogen and may become clogged. Whether destroyed or clogged, the affected vessels cease to function properly and allow little or no water to pass through them. Certain pathogens, such as the crown gall bacterium (*Agrobacterium tumefaciens*), the clubroot protozoan (*Plasmodiophora brassicae*), and the root-knot nematode (*Meloidogyne* sp.), induce gall formation in the stem, roots, or both. The enlarged and proliferating cells near or around the xylem exert pressure on the xylem vessels, which may be crushed and dislocated, thereby becoming less efficient in transporting water. The most typical and complete dysfunction of xylem in translocating water, however, is observed in the vascular wilts caused by the fungi *Ceratocystis*, *Ophiostoma*, *Fusarium*, and *Verticillium* and bacteria such as *Pseudomonas*, *Ralstonia*, and *Erwinia*. These pathogens invade the xylem of roots and stems and produce diseases primarily by interfering with the upward movement of water through the xylem. In many plants infected by these pathogens the water flow through the stem xylem is reduced to a mere 2 to 4% of that flowing through stems of healthy plants. In general, the rate of flow through infected stems seems to be inversely proportional to the number of vessels blocked by the pathogen and by the substances resulting from the infection. Evidently more than one factor is usually responsible for the vascular dysfunction in the wilt diseases. Although the pathogen is the single cause of the disease, some of the factors responsible for the disease syndrome originate directly from the pathogen, whereas others originate from the host in response to the pathogen. The pathogen can reduce the flow of water through its physical presence in the xylem as mycelium, spores, or bacterial cells and by the production of large molecules (polysaccharides) in the vessels. In most host-pathogen combinations, the destruction of xylem vessels by fungi. Results in the collapse and death of the plant, as does the invasion of xylem vessels by fungi or bacteria. In host combinations with the fastidious bacterium *Xylella fastidiosa*, growth, multiplication, and spread of bacteria in xylem vessels are slower and, instead of causing wilting and rapid death of the plant, a scorching of the margins of the leaves and several other symptoms occur, but rarely does the plant die quickly. In all cases, however, in infected hosts the flow of water is reduced through reduction in the size or collapse of vessels due to infection, development of tyloses. in the vessels, release of large molecule compounds in the vessels as a result of cell wall breakdown by pathogenic enzymes and reduced water tension in the vessels due to pathogen-induced alterations in foliar transpiration.



(1)

(2)

Examples of reduction of water absorption by plants. (1) Numerous galls caused by the bacterium *Agrobacterium tumefaciens* on roots of a cherry tree. (2) Root knot galls caused by the nematode *Meloidogyne* sp. on roots



(A)

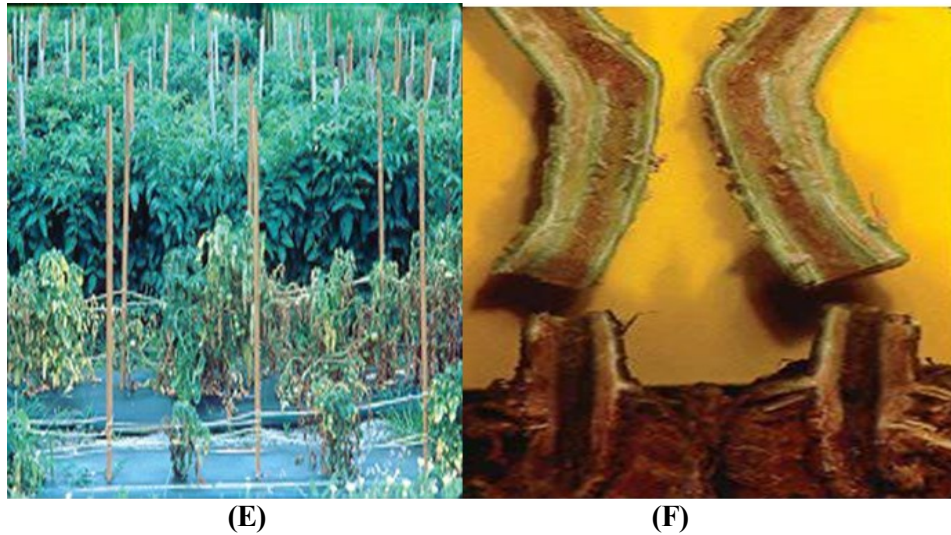
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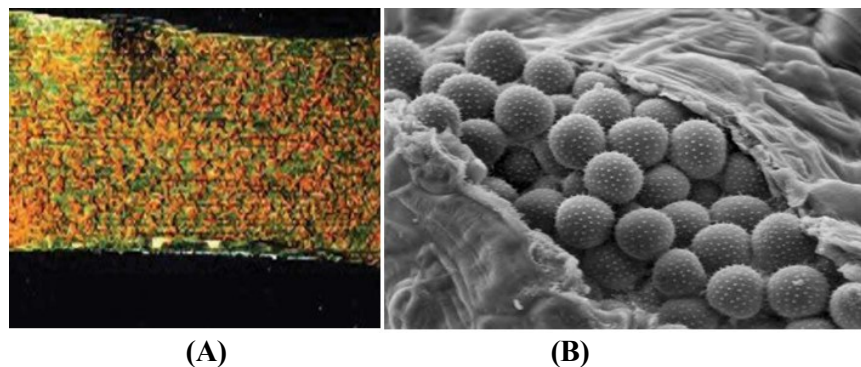
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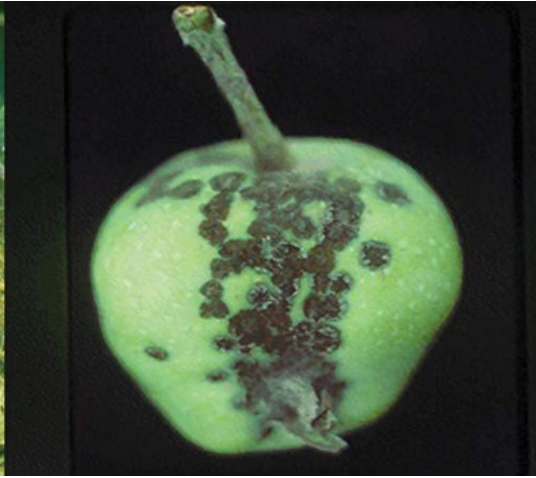
Examples of reduction of upward translocation of water and mineral nutrients (A) the fungus *Phomopsis* sp. (B) Canker on an almond tree caused by the fungus *Ceratocystis fagacearum*. (C) Vascular wilt of tomato caused by the fungus *Fusarium*. (D) Discolored vascular tissues of a tomato stem infected with the same fungus. (E) Wilted tomato plants infected with the vascular bacterium *Ralstonia solanacearum*. (F) Discolored vascular tissues of a tomato plant infected with the same bacterium.

5. **Transpiration:** In plant diseases in which the pathogen infects the leaves, transpiration is usually increased. This is the result of destruction of at least part of the protection afforded the leaf by the cuticle, an increase in the permeability of leaf cells, and the dysfunction of stomata. In diseases such as rusts, in which numerous pustules form and break up the epidermis, in most leaf spots, in which the cuticle, epidermis, and all the other tissues, including xylem, may be destroyed in the infected areas, in the powdery mildews, in which a large proportion of the epidermal cells are invaded by the fungus and in apple scab, in which the fungus grows between the cuticle and the epidermis in all these examples, the destruction of a considerable portion of the cuticle and epidermis results in an uncontrolled loss of water from the affected areas. If water absorption and translocation cannot keep up with the excessive loss of water, loss of turgor and wilting of leaves follow. The suction forces of excessively transpiring leaves are increased abnormally and may lead to collapse or dysfunction of underlying vessels through the production of tyloses and gums.

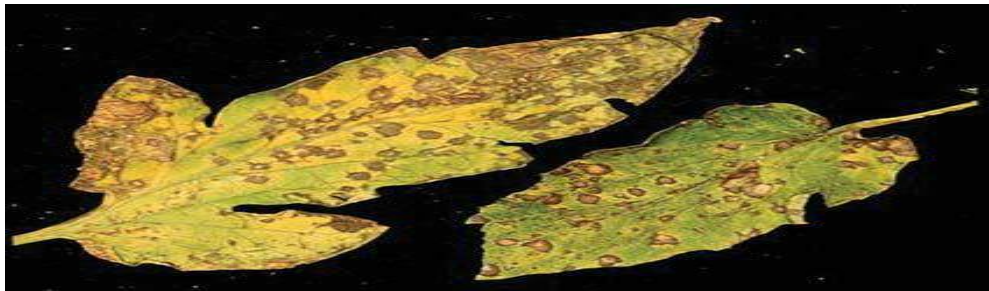




(B)



(D)



(E)

Pathogens cause increased transpiration in infected plants. (A) wheat leaf rust pathogen *Puccinia recondita* (B) Uredospores breaking the epidermis and emerging from the surface of an infected leaf. (C) Grape berries infected with the powdery mildew fungus *Uncinula necator*, and forms haustoria in almost every epidermal cell. (D) The apple scab fungus *Venturia inaequalis* grows between the cuticle and the epidermis, (E) Tomato leaves caused by the fungus *Septoria* sp. and through which excessive transpiration occurs.

- 6. Interference with Translocation of Organic Nutrients through the Phloem:** Organic nutrients produced in leaf cells through photosynthesis move through plasmodesmata into adjoining phloem elements. From there they move down the Phloem sieve tubes and eventually, again through plasmodesmata, into the protoplasm of living non photosynthetic cells, where they are utilized, or into storage organs, where they are stored. Thus, in both cases, the nutrients are removed from “circulation.” Plant pathogens may interfere with the movement of organic nutrients from the leaf cells to the phloem, with their translocation through the phloem elements, or, possibly, with their movement from the phloem into the cells that will utilize them. Obligate fungal parasites, such as rust and mildew fungi, cause an accumulation of photosynthetic products, as well as inorganic nutrients, in the areas invaded by the pathogen. In these diseases, the infected areas are characterized by reduced photosynthesis and increased respiration. However, the synthesis of starch and other compounds, as well as dry weight, is increased temporarily in the infected areas, indicating translocation of organic nutrients from



uninfected areas of the leaves or from healthy leaves toward the infected areas. In stem diseases of woody plants in which cankers develop, the pathogen attacks and remains confined to the bark for a considerable time. During that time the pathogen attacks and may destroy the phloem elements in that area, thereby interfering with the downward translocation of nutrients. In diseases caused by phytoplasmas, as well as in diseases caused by phloem-limited fastidious bacteria, bacteria exist and reproduce in the phloem sieve tubes, thereby interfering with the downward translocation of nutrients. In several plants propagated by grafting a variety scion onto a rootstock, infection of the combination with a virus (e.g., infection of an apple or stone-fruit rootstock with *tomato ringspot virus*) leads to formation of a necrotic plate at the points of contact of the hypersensitive scion variety with the rootstock which leads to the death of the scion. However, infection of a pear scion grafted on an oriental Rootstock with the pear decline phytoplasma, or of a citrus variety propagated on sour rootstock with the citrus tristeza virus, results, in both cases, in the necrosis of a few layers of cells of each rootstock in contact with the tolerant variety. In these cases, the rootstock is the component of the scion/stock combination that is hypersensitive to and becomes killed by the appropriate pathogen.

### III. EFFECT OF PATHOGENS ON HOST PLANT RESPIRATION

Respiration is the process by which cells, through the enzymatically controlled oxidation (burning) of the energy-rich carbohydrates and fatty acids, liberate energy in a form that can be utilized for the performance of various cellular processes. Plant cells carry out respiration in, basically, two steps. The first step involves the degradation of glucose to pyruvate and is carried out, either in the presence or in the absence of oxygen, by enzymes found in the ground cytoplasm of the cells. The production of pyruvate from glucose follows either the **glycolytic pathway**, otherwise known as **glycolysis**, or, to a lesser extent, the **pentose pathway**. The second step, regardless of the pathway, involves the degradation of pyruvate, however produced, to CO<sub>2</sub> and water. This is accomplished by a series of reactions known as the **Krebs cycle**, which is accompanied by the so-called **terminal oxidation** and is carried out in the mitochondria only in the presence of oxygen. Under normal (aerobic) conditions, i.e., in the presence of oxygen, both steps are carried out, and one molecule of glucose yields, as final products, six molecules of CO<sub>2</sub> and six molecules of water, with a concomitant release of energy (678,000 calories). Some of the energy is lost, but almost half is converted to 20–30 reusable high-energy bonds of adenosine triphosphate (ATP). The first step of respiration contributes two ATP molecules per mole of glucose, and the second step contributes the rest. Under anaerobic conditions, however (i.e., in the absence of oxygen), pyruvate cannot be oxidized; instead it undergoes **fermentation** and yields lactic acid or alcohol. Because the main process of energy generation is cut off, for the cell to secure the necessary energy a much greater rate of glucose utilization by glycolysis is required in the absence of oxygen than is in its presence. The energy-storing bonds of ATP are formed by the attachment of a phosphate (PO<sub>4</sub>) group to adenosine diphosphate (ADP) at the expense of energy released from the oxidation of sugars. The coupling of the oxidation of glucose with the addition of phosphate to ADP to produce ATP is called **oxidative phosphorylation**. Any cell activity that requires energy utilizes the energy stored in ATP by simultaneously breaking down ATP to ADP and inorganic phosphate. The presence of ADP and phosphate in the cell, in turn, stimulates the rate of respiration. If, however, ATP is not utilized sufficiently by the cell for some reason, there is little or no regeneration of ADP and respiration is slowed down. The

amount of ADP (and phosphate) in the cell is determined, therefore, by the rate of energy utilization; this rate, in turn, determines the rate of respiration in plant tissues. The energy produced through respiration is utilized by the plant for all types of cellular work, such as accumulation and mobilization of compounds, synthesis of proteins, activation of enzymes, cell growth and division, defense reactions, and a host of other processes. The complexity of respiration, the number of enzymes involved in respiration, its occurrence in every single cell, and its far-reaching effects on the functions and existence of the cell make it easy to understand why the respiration of plant tissues is one of the first functions to be affected when plants are infected by pathogens.

#### **IV. RESPIRATION OF DISEASED PLANTS**

When plants are infected by pathogens, the rate of respiration generally increases. This means that affected tissues use up their reserve carbohydrates faster than healthy tissues would. The increased rate of respiration appears shortly after infection certainly by the time of appearance of visible symptoms —and continues to rise during the multiplication and sporulation of the pathogen. After that, respiration declines to normal levels or to levels even lower than those of healthy plants. Respiration increases more rapidly in infections of resistant varieties, in which large amounts of energy are needed and used for rapid production or mobilization of the defence mechanisms of the cells. In resistant varieties, however, respiration also declines quickly after it reaches its maximum. In susceptible varieties, in which no defence mechanisms can be mobilized quickly against a particular pathogen, respiration increases slowly after inoculation, but continues to rise and remains at a high level for much longer periods. Several changes in the metabolism of the diseased plant accompany the increase in respiration after infection. Thus, the activity or concentration of several enzymes of the respiratory pathways seems to be increased. The accumulation and oxidation of phenolic compounds, many of which are associated with defence mechanisms in plants, are also greater during increased respiration. Increased respiration in diseased plants is also accompanied by an increased activation of the pentose pathway, which is the main source of phenolic compounds. Increased respiration is sometimes accompanied by considerably more fermentation than that observed in healthy plants, probably as a result of an accelerated need for energy in the diseased plant under conditions in which normal aerobic respiration cannot provide sufficient energy. The increased respiration in diseased plants is apparently brought about, at least in part, by the uncoupling of oxidative phosphorylation. In that case, no utilizable energy (ATP) is produced through normal respiration, despite the use of existing ATP and the accumulation of ADP, which stimulates respiration. The energy required by the cell for its vital processes is then produced through other less efficient ways, including the pentose pathway and fermentation. The increased respiration of diseased plants can also be explained as the result of increased metabolism. In many plant diseases, growth is at first stimulated, protoplasmic streaming increases, and materials are synthesized, translocated, and accumulated in the diseased area. The energy required for these activities derives from ATP produced through respiration. The more ATP is utilized, the more ADP is produced and further stimulates respiration. It is also possible that the plant, because of the infection, utilizes ATP energy less efficiently than a healthy plant. Because of the waste of part of the energy, an increase in respiration is induced, and the resulting greater amount of energy enables the plant cells to utilize sufficient energy to carry out their accelerated processes. Although oxidation of glucose via the glycolytic pathway is by far the most common way through which plant cells obtain their energy, part of the energy is produced via the pentose pathway. The latter seems to be an alternate pathway of energy production to which plants resort under conditions of stress. Thus,

the pentose pathway tends to replace the glycolytic pathway as the plants grow older and differentiate and it tends to increase on treatment of the plants with hormones, toxins, wounding, starvation, and so on. Infection of plants with pathogens also tends, in general, to activate the pentose pathway over the level at which it operates in the healthy plant. Because the pentose pathway is not linked directly to ATP production, the increased respiration through this pathway fails to produce as much utilizable energy as the glycolytic pathway and is, therefore, a less efficient source of energy for the functions of the diseased plant. However, the pentose pathway is the main source of phenolic compounds, which play important roles in the defense mechanisms of the plant against infection.

## V. EFFECT OF PATHOGENS ON PERMEABILITY OF CELL MEMBRANES

**Cell membranes** consist of a double layer of lipid molecules in which many kinds of protein molecules are embedded, parts of which usually protrude on one or both sides of the lipid bilayer. Membranes function as permeability barriers that allow passage into a cell only of substances the cell needs and inhibit passage out of the cell of substances needed by the cell. The lipid bilayer is impermeable to most biological molecules. Small water-soluble molecules such as ions (charged atoms or electrolytes), sugars, and amino acids flow through or are pumped through special membrane channels made of proteins. In plant cells, because of the cell wall, only small molecules reach the cell membrane. In animal cells and in artificially prepared plant protoplasts, however, large molecules or particles may also reach the cell membrane and enter the cell by endocytosis, in which a patch of the membrane surrounds and forms a vesicle around the material to be taken in, brings it in, and releases it inside the cell. Disruption or disturbance of the cell membrane by chemical or physical factors alters (usually increases) the permeability of the membrane with a subsequent uncontrollable loss of useful substances, as well as the inability to inhibit the inflow of undesirable substances or excessive amounts of any substances. Changes in cell membrane permeability are often the first detectable responses of cells to infection by pathogens, to most host-specific and several nonspecific toxins, to certain pathogen enzymes, and to certain toxic chemicals, such as air pollutants. The most commonly observed effect of changes in cell membrane permeability is the loss of **electrolytes**, i.e., of small water-soluble ions and molecules from the cell. Electrolyte leakage occurs much sooner and at a greater rate when the host-pathogen interaction is incompatible, and the host remains more resistant than when the host is susceptible and develops extensive symptoms. It is not certain, however, whether the cell membrane is the initial target of pathogen toxins and enzymes and whether the accompanying loss of electrolytes is the initial effect of changes in cell membrane permeability or whether the pathogen products actually affect other organelles or reactions in the cell, in which case cell permeability changes and loss of electrolytes are secondary effects of the initial events. If pathogens do, indeed, affect cell membrane permeability directly, it is likely that they bring this about by stimulating certain membrane bound enzymes, such as ATPase, which are involved in the pumping of  $H^+$  in and  $K^+$  out through the cell membrane, by interfering with processes required for the maintenance and repair of the fluid film making up the membrane, or by degrading the lipid or protein components of the membrane by pathogen produced enzymes.



## VI. EFFECT OF PATHOGENS ON TRANSCRIPTION AND TRANSLATION

Transcription of cellular DNA into messenger RNA and translation of messenger RNA to produce proteins are two of the most basic, general, and precisely controlled processes in the biology of any normal cell. The part(s) of the genome involved and the level and timing of transcription and translation vary with the stage of development and the requirements of each cell. Nevertheless, disturbance of any one of these processes, by pathogens or environmental factors, may cause drastic, unfavourable changes in the structure and function of the affected cells by its effect on the expression of genes.

- 1. Transcription:** Several pathogens, particularly viruses and fungal obligate parasites, such as rusts and powdery mildews, affect the transcription process in infected cells. In some cases, pathogens affect transcription by changing the composition, structure, or function of the chromatin associated with the cell DNA. In some diseases, especially those caused by viruses, the pathogen, through its own enzyme or by modifying the host enzyme (RNA polymerase) that makes RNA, utilizes the host cell nucleotides and machinery to make its own (rather than host) RNA. In several diseases, the activity of ribonucleases (enzymes that break down RNA) is increased, perhaps by formation in infected plants of new kinds of ribonucleases not known to be produced in healthy plants. Finally, in several diseases, infected plants, particularly resistant ones, seem to contain higher levels of RNA than healthy plants, especially in the early stages of infection. It is generally believed that greater RNA levels and, therefore, increased transcription in cells indicate an increased synthesis of substances involved in the defence mechanisms of plant cells.
- 2. Translation:** Infected plant tissues often have increased activity in several enzymes, particularly those associated with the generation of energy (respiration) or with the production or oxidation of various phenolic compounds, some of which may be involved in (defence) reactions to infection. Although a certain amount of some of these enzymes (proteins) may be present in the cell at the time of infection, several are produced *de novo*, necessitating increased levels of transcription and translation activity. Increases in protein synthesis in infected tissues have been observed primarily in hosts resistant to the pathogen and reach their highest levels in the early stages of infection, i.e., in the first few minutes and up to 2–20 hours after inoculation. If resistant tissues are treated before or during infection with inhibitors of protein synthesis, their resistance to the pathogen is reduced. These observations suggest that much of the increased protein synthesis in plants attacked by pathogens reflects the increased production of enzymes and other proteins involved in the defence reactions of plants.

## VII. EFFECT OF PATHOGENS ON PLANT GROWTH

It is easily understood and expected that pathogens that destroy part of the photosynthetic area of plants and cause significantly reduced photosynthetic output often result in smaller growth of these plants and smaller yields. Similarly, pathogens that destroy part of the roots of a plant or clog their xylem or phloem elements, thereby severely interfering with the translocation of water and of inorganic or organic nutrients in these plants, often cause a reduction in size and yields by these plants and, sometimes, their death. In many plant diseases, however, infected tissues or entire plants increase or reduce abnormally in size without a clear-

cut explanation of how these changes are brought about. It is apparent that growth regulators affecting plant cell division and enlargement are involved, but very little is known about the specific compounds and mechanisms involved or the genes that control these events.



(A)

(B)



(B)

(D)



(E)

(F)

Effect of pathogens on plant growth. (A) Leaf curling (B) fruit enlargement by the leaf curl fungus *Taphrina deformans* on peach and plum (C) Leaf malformations caused by the *common bean mosaic virus* on bean (D) stunting caused by the *maize streak virus* on corn (E) Galls caused by the crown gall bacterium *Agrobacterium tumefaciens* and (F) galls along the roots of a plant caused by the root knot nematode *Meloidogyne* sp.

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