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Chapter 32

Physiology of Diseased Plants and Plant Response against Pathogen Attack

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Huge losses of the crops are caused by the plant diseases globally. The loss can occur from the time of seed sowing in the field to harvesting and storage. There are many important historical evidences of plant disease epidemics *viz.* Irish Famine due to late blight of potato (Ireland, 1845), Bengal famine due to brown spot of rice (India, 1942) and Coffee rust (Sri Lanka, 1967). Such epidemics had left their huge consequence on the economy of the affected countries.

Concept of Plant Disease

The normal physiological functions of plants are disturbed when they are affected by pathogenic living organisms or by some environmental factors. Initially plants react to the disease causing agents, particularly in the site of infection. Later, the reaction becomes more widespread and histological changes take place. Such changes are expressed as different types of symptoms of the disease which can be visualized macroscopically. As a result of the disease, plant growth in reduced, deformed or even the plant dies.

When a plant is suffering, we call it diseased, *i.e.* it is at 'dis-ease'. Disease is a condition that occurs in consequence of abnormal changes in the form, physiology,

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integrity or behaviour of the plant. According to American Phytopathological Society (*Phytopathology* 30: 361-368, 1940), disease is a deviation from normal functioning of physiological processes of sufficient duration or intensity to cause disturbance or cessation of vital activities. Encyclopedia Britannica (2002) forwarded a simplified definition of plant disease as "A plant is diseased when it is continuously disturbed by some causal agent that results in abnormal physiological process that disrupts the plants normal structure, growth, function or other activities." This interference with one or more plant's essential physiological or biochemical systems elicits characteristic pathological conditions or symptoms.

Effect of Pathogen on the Host Plants

While pathogens infect plants in the course of their obtaining food for them (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.

During the course of pathogenesis, normal activities of the infected host plant undergo malfunction. Consequently, morphological and physiological changes occur, which are enlisted below:

A. Morphological or Structural Changes

Physiological malfunctioning of the host cells causes disturbances in chemical reaction which ultimately lead to some structural changes *viz.*, overgrowth, phyllody, sterile flowers, hairy roots, witches broom, bunchy top, crown gall, root knot, leaf curling, rolling, puckering etc.

B. Physiological Changes

I. Disintegration of the tissues by the enzymes of the pathogen.

II. Negative effect on the growth of the host plant due to growth regulators produced by the pathogen or by the host under the influence of the pathogen.

III. Effect on uptake and translocation of water and nutrients.

IV. Abnormality in respiration of the host tissues due to disturbed permeability of cell membrane and enzyme system associated with respiration.

V. Impairing the phenomenon of photosynthesis due to loss of chlorophyll and destruction of leaf tissue.

VI. Effect on the process of transcription and translation.

VII. Weakening of overall reproduction system of the host.

Effect of Pathogens on Photosynthesis of Host Plants

Plants and pathogens have developed dynamic interactions. Whereas plants

tend to survive through different mechanisms after pathogen attack, the pathogen looks for maximizing feed intake to insure its reproduction and dissemination (Korves and Bergelson, 2003; Berger *et al.*, 2007). The photosynthetic process is the energy source for both the plant and pathogens. The outcome of CO_2 fixation by higher photosynthetic plants is the production of carbohydrates. As a result of photosynthetic CO_2 reduction during the day, starch granules accumulate in the chloroplast while an excess of assimilates are continuously allocated (mostly in the form of sucrose) to sink tissues such as developing leaves, roots, meristems, fruits, and flowers, that are unable to produce sufficient amounts of assimilates by themselves and therefore require their net import via the phloem (Kocal *et al.*, 2008). Sucrose is loaded into the phloem in the minor veins of leaves before export (Zhang and Turgeon, 2009). Thus for the normal growth and development of plants a coordinated sequence of assimilate production, allocation, and utilization is very essential (Kocal *et al.*, 2008).

Plant pathogens like viruses, fungi, oomycetes, and bacteria are known to interfere with the source-sink balance (Biemelt and Sonnewald, 2006; Berger *et al.*, 2007; Seo *et al.*, 2007), and in the case of a successful interaction, pathogens are believed to reprogram a plants' metabolism to their own benefit (Biemelt and Sonnewald, 2006). This comprises the suppression of plant defence responses and the reallocation of photoassimilates to supply the pathogen with sufficient nutrients (Kocal *et al.*, 2008). In accordance with this, the infected leaf is assumed to undergo a source to sink transition or retains its sink character. For example, infection of maize leaves with *Ustilago maydis* prevents establishment of C4 photosynthesis because *U. maydis* induced leaf galls exhibited carbon dioxide response curves, CO_2 compensation points and enzymatic activities that are characteristic of C3 photosynthesis (Horst *et al.*, 2008). An indication for this is provided by a stimulation of cell wall bound invertase (cw-Inv) that mobilizes hexoses at the infection site and a decreased rate of photosynthesis (Kocal *et al.*, 2008).

Pathogen attacks result in the development of symptoms that include leaf and fruit wilt, stem and root rot (Rekah *et al.*, 1999), coverage of leaf surface with pustule, chlorosis and necrosis (Fofana *et al.*, 2007; Kocal *et al.*, 2008), a decreased rate of plant photosynthesis (Kocal *et al.*, 2008), and as a consequence plant death or yield loss (Berger *et al.*, 2007). Pathogens that cause defoliation rob the photosynthetic tissue of plant and decrease the photosynthetic rate by damaging chloroplasts and killing cells. Pathogens affect photosynthesis in varying degrees, depending on the severity of the infection.

Pathogen attacks result in a decreased rate of plant photosynthesis (Kocal *et al.*, 2008), and as a consequence yield loss (Berger *et al.*, 2007). Pathogen infection often leads to plant death, the development of chlorotic and necrotic lesions (Kim *et al.*, 2010) and to a decrease in photosynthetic assimilate production. Using chlorophyll fluorescence imaging, it has been reported that the changes in photosynthesis upon infection are local. A decrease in photosynthesis has also been reported in incompatible interactions (Bonfig *et al.*, 2006). It is suggested that plants switch off photosynthesis and other assimilatory metabolism to initiate respiration and other processes required for defence (Berger *et al.*, 2007). Foliar symptoms were associated with stomatal closure and alteration of the photosynthetic apparatus. Petit *et al.*

(2006) has reported a decrease in CO_2 assimilation, transpiration, a significant increase in intercellular CO_2 concentration, a strong drop in the maximum fluorescence yield and the effective photosystem II quantum yields, and a reduction of total chlorophyll but a stable carotenoid content after of pathogen infection to grapevines plants.

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 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.

Damage to foliage by biotic agents including arthropods, fungi, bacteria and viral pathogens, down regulates the expression of genes involved in photosynthesis. Transcript levels of photosynthesis light reaction, carbon reduction cycle and pigment synthesis genes decreased regardless of the type of biotic attack. Strong convergence in the response of transcription suggests that the universal down regulation of photosynthesis related gene expression is an adaptive response to biotic attack. Slow turnover of many photosynthetic proteins allows plants to invest resources in immediate defence needs without weakening near term losses in photosynthetic capacity (Bilgin *et al.*, 2010).

Effect of Pathogens on Translocation of Water, Nutrients and Photo-assimilates in the Host Plants

Translocation or long distance transport in plants is achieved by a vascular network that connects and is an integral part of all organs. The vasculature comprises two different and separate cellular translocation pathways: xylem and phloem. The principal xylem pathway is the transpiration stream that moves nutrients and water taken up by roots to the shoot. This stream also bears products of root metabolism and solutes that reflect features of the internal and external root environment. Phloem provides the means for redistributing xylem delivered solutes to weakly transpiring organs, but most significantly phloem distributes the carbon assimilated by photosynthesis (principally as Sucrose) to heterotrophic organs like roots, vegetative and reproductive apices, flowers, fruits, and developing seeds. All living plant cells require abundance of water and 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. Plant diseases can infect the plant's vascular system and impair nutrient or water translocation. Pathogens that infect the roots directly affect the ability of plant to absorb water by killing the root system, thus producing secondary symptoms such as wilting and defoliation.

Mineral nutrients are essential for the growth and development of plants and micro-organisms, and are important factors in plant-pathogen interactions. When a pathogen infects a plant, it alters the physiology of plant, particularly with regard to uptake of mineral nutrient, assimilation, translocation, and its utilization.

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. 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.

Some pathogens invade the xylem of roots and stems and produce diseases primarily by interfering with the upward movement of water through the xylem. When a pathogen interferes with the upward movement of inorganic nutrients and water or with the downward movement of organic substances, unhealthy 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. Pathogens may also interfere with immobilize nutrients in the soil or in infected tissues. They may also interfere with translocation or utilization of nutrients, inducing nutrient deficiencies or toxicities. Still other pathogens may themselves utilize nutrients, reducing their availability to the plant and thereby increasing the plant's susceptibility to infection. Soil borne pathogens commonly infect plant roots, reducing the ability of plant to take up water and nutrients. 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. These conditions may lead to secondary infections by other pathogens. Such infections can cause root starvation, wilting, and plant decline or death, even though the pathogen itself may not be toxic.

Plant pathogens may interfere with the movement of photo-assimilates from the leaf cells to the phloem, with their translocation through the phloem elements or with their movement from the phloem into the cells that will utilize them. An infection site becomes a strong metabolic sink, changing the pattern of nutrient translocation within the plant, and causing net influx of nutrients into infected leaves to satisfy the demands of the pathogen.

Effect on Host Plant Transpiration

Transpiration is the process of water movement through a plant and its evaporation from aerial parts especially from leaves but also from stems and flowers. Transpiration occurs through the stomatal apertures and can be thought of as a necessary "cost" associated with the opening of the stomata to allow the diffusion of carbon dioxide gas from the air for photosynthesis. Transpiration also cools plants, changes osmotic pressure of cells, and enables mass flow of mineral nutrients and water from roots to shoots. 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. Higher fungi and oomycetes cause physical disruption to the cuticle and stomata and also cause impairment of stomatal closing

in the dark. Higher fungi and viruses are associated with impairment of stomatal opening in the light (Grimmer *et al.*, 2012). A number of toxins produced by bacteria and higher fungi have been identified that impair stomatal function (Grimmer *et al.*, 2012). At the site of infection 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, it can be resulted into loss of turgor and wilting of leaves. 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 (Agrios, 2005).

Effect of Pathogens on Host Plant Respiration

Respiration refers to the metabolic process by which an organism obtains energy by reacting oxygen with glucose to give water, carbon dioxide and ATP (energy). This is very important process of energy generation for other metabolic processes. The respiration rate of plants invariably increases after infection by fungi, bacteria or viruses. This means that affected tissues use up their reserve carbohydrates faster than healthy tissues would. Thus, the activity or concentration of several enzymes of the respiratory pathways seems to be increased. The higher rate of glucose catabolism causes a measurable increase in the temperature of infected leaves. An early step in the plant's response to infection is an oxidative burst, which is manifested as a rapid increase in oxygen consumption, and the release of reactive oxygen species, such as hydrogen peroxide (H_2O_2) and the superoxide anion (O_2^-). The oxidative burst is involved in a range of disease resistance and wound repair mechanisms link to rapid active defence. The accumulation and oxidation of phenolic compounds, many of which are associated with defence mechanisms in plants, are also greater during increased respiration. In resistant plants, the increase in respiration and glucose catabolism is used to produce defence related metabolites via the pentose phosphate pathway (main source of phenolic compounds). In susceptible plants, the extra energy produced is used by the growing pathogen.

Effect of Pathogens on Permeability of Cell Membranes of Host Plants

The cell membrane, or plasma membrane, is a biological membrane that separates the interior of all cells from the outside environment. The cell membrane is selectively permeable to ions and organic molecules and controls the movement of substances in and out of cells. The basic function of the cell membrane is to protect the cell from its surroundings. Most studies on the earliest stages of the host-parasite interaction conclude that the host membrane is involved in pathogen recognition and signal transduction. Membrane permeability changes rapidly following the exposure of plant cell suspension cultures to fungal and bacterial elicitors, usually leading to a loss of cellular electrolytes *i.e.*, of small water-soluble ions and molecules from the cell, such as K^+ and an uptake of H^+. At the same time, there is often an influx of Ca^{2+}, a key intracellular signal in plants that is involved in the activation of enzymes and gene expression. The experimental blocking of Ca^{2+} transport across membranes in

inoculated bean cells also inhibits gene activation and subsequent defence responses. 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.

This permeability of the cell membrane leads to a pH change in the apoplast, which is essential for a full oxidative burst in some species such as *P. vulgaris* and important for other species such as *Arabidopsis* which is related with defence mechanism of host plants against pathogens (Bolwell *et al.*, 2002; Bindschedler *et al.*, 2006; Davies *et al.*, 2006). The increase of cytosolic Ca^{2+} is also involved in the induction of PR genes and phytoalexin production which are also the important components of defence mechanism of host plants against pathogens (Blume *et al.*, 2000).

Effect of Pathogens on Transcription and Translation Process of Host Plants

Transcription of cellular DNA into messenger RNA and translation of messenger RNA to produce proteins are two of the most basic and precisely controlled processes in the biology of any normal cell. They 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.

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.

In some cases, plant host cells have the ability to recognize and silence double stranded forms of virus transcripts. In fact, viral infection often activates or inactivates transcription or translation of a number of host plant genes in both the susceptible and the resistant reactions (Hajimorad and Hill, 2001).

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.

Effect of Pathogens on Plant Growth

Growth represents increase in size, number and complexity of plant cells and organs. It is the irreversible increase in mass that results from cell division (number) and cell expansion (size). Development is the sum of all the changes (patterned differentiated cells, tissues, organs) that progressively elaborate an organism's body.

Growth and development in general are affected by pathogen infection, as a result of the changes in source-sink patterns in the plant. Many pathogens disturb the hormone balance in plants by either releasing plant hormones themselves, or by triggering an increase or a decrease in synthesis or degradation of hormones in the plant. This can cause a variety of symptoms, such as the formation of adventitious roots, gall development, and epinasty (the down-turning of petioles). Phytopathogen infection leads to changes in secondary metabolism based on the induction of defence programmes as well as to changes in primary metabolism which affect growth and development of the plant. Therefore, pathogen attack causes crop yield losses even in interactions which do not end up with disease or death of the plant (Berger *et al.*, 2007).

Effect of Pathogens on Plant Reproduction

Plant reproduction is the production of new individuals or offspring in plants, which can be accomplished by sexual or asexual means. Sexual reproduction produces offspring by the fusion of gametes, resulting in offspring genetically different from the parent or parents. Asexual reproduction produces new individuals without the fusion of gametes, genetically identical to the parent plants and each other, except when mutations occur. In seed plants, the offspring can be packaged in a protective seed, which is used as an agent of dispersal. Pathogens that attack various organs and tissues of plants weaken and often kill these organs or tissues, thereby weakening the plants. As a result, such plants remain smaller in size, may produce fewer flowers, and may set fewer fruit and seeds; the latter may be of inferior vigour and vitality and, therefore, if planted, they may produce fewer and weaker new plants. In addition to these indirect effects of pathogens on plant reproduction, many pathogens have a direct adverse effect on plant reproduction because they attack and kill the flowers, fruit, or seed directly, or interfere and inhibit their production, or the pathogens interfere directly or indirectly with the propagation of their host plant.

Plants Response to Pathogens Attack

Plants defend themselves against pathogens by various ways which can be divided into different components:

1. Structural characteristics that act as physical barriers and inhibit the pathogen from gaining entrance and spreading through the plant and
2. Biochemical reactions that take place in the cells and tissues of the plant and produce substances that are either toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant. The combination of structural characteristics and biochemical reactions employed in the defence of plants are different host-pathogen systems.

(1) Structural Defences

(a) Pre-existing Structural Defences

This structural characteristic may already be present in plant even before the pathogen comes in contact with the plant.

i. Wax and cuticle that cover the epidermal cells

Waxes on leaf and fruit surfaces forms a water-repellent surface, thereby preventing the formation of a film of water on which pathogens might be deposited and germinate (fungi) or multiply (bacteria).

ii. Tough and thick epidermal cells

These are important factors in the resistance of some plants to certain pathogens by making direct penetration fungal pathogens difficult or impossible.

iii. Nature of natural opening

Many pathogenic fungi and bacteria enter plants only through stomata. The structure of stomata *e.g.* a very narrow entrance and broad, elevated guard cells, may confer resistance to some varieties against certain bacterial pathogens.

iv. Internal structural barriers

The cell walls of the tissue invaded vary in thickness and toughness and may sometimes inhibit the advance of the pathogen.

(b) Induced Structural Defences

These are usually not present in the plants but are produce in response to invading pathogens.

i. Cork layers formation

Infection by fungi or bacteria and even by some viruses and nematodes frequently induces plants to form several layers of cork cells beyond the point of infection, apparently as a result of stimulation of the host cells by substances secreted by the pathogen.

ii. Formation of abscission layers

An abscission layer consists of gap formed between two circular layers of leaf cells surrounding the locus of infection. Upon infection, the middle lamella between these two layers of cells is dissolved throughout the thickness of the leaf, completely cutting off the central area of infection from the rest of the leaf.

iii. Formation of tyloses

Tyloses are overgrowths of the protoplast of adjacent living parenchymatous cells which protrude into xylem vessels through pits. Tyloses form an impenetrable barrier to the movement of water and nutrients.

iv. Deposition of gums

The defensive role of gums stems from the fact that they are deposited quickly in the intercellular spaces and within the cells surrounding the locus of infection, thus forming an impenetrable barrier that completely encloses the pathogen.

Table 32.1: Events Involved in the Coordination of Defence Responses in Plants to Challenge by Pathogens

Time	Event
Minutes	Membrane depolarisation and electrolyte leakage
	Reactive oxygen generation
	Expression of genes involved in phytoalexin biosynthesis
Hours	Oxidative burst
	Membrane lipid peroxidation
	Rise in salicylic acid levels
	Cytoplasmic aggregation, cell collapse and hypersensitive cell death
	Phytoalexin accumulation
	Cell wall reinforcements
Days	Accumulation of pathogenesis-related proteins (PRP)
	Systemic acquired resistance (SAR)

(2) Biochemical Defences

(a) Pre-existing Biochemical Defences

i. Inhibitors released by the plant in its environment

Plants exude a variety of substances through the surface of their aboveground parts as well as through the surface of their roots. Some of these exudates seem to have an inhibitory action against certain pathogens.

ii. Inhibitors present in plant cells before infection

Some plants are resistant to diseases caused by certain pathogens because of one or more inhibitory antimicrobial compounds known as phytoanticipins, which are present in the cell before infection.

iii. Defence through deficiency in nutrients essential for pathogen

Species or varieties of plants that for some reason do not produce one of the substances essential for the survival of an obligate or for development of infection by any parasite, would be resistant to the pathogen that requires it.

(b) Induced Biochemical Defence

i. Inhibitors produced by plants

Many plants produce substances in response to microorganism or to mechanical and chemical injuries *e.g.*, phenolics and phytoalexins.

ii. Defence through production of substances that inhibit effect of enzymes produced by pathogens

The production of substances which inhibit the effect of extra cellular enzymes produced by phytopathogenic organism has been known to contribute to resistance

e.g. production of polyvalent cations such as Ca^{2+} around developing *Rhizoctonia* lesions, restrict further tissue maceration by polygalacturonase produced by invading pathogen.

iii. Defence through detoxification of pathogen toxins

In some plants, resistance to the pathogens is the same as resistance to the toxin as shown by the correlation between toxins production and pathogenicity.

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