# POST GRADUATE DEGREE PROGRAMME
# (CBCS)
# IN
# BOTANY

**SEMESTER -III**

**Course: BOTCOR T309**

**(Plant Pathology & Crop Protection)**

**Self-Learning Material**

**DIRECTORATE OF OPEN AND DISTANCE LEARNING**

**UNIVERSITY OF KALYANI**

**KALYANI – 741 235, WEST BENGAL**

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# Foreword

Satisfying distance learners' needs of verifying kinds and magnitude as well as minimizing distance and to reach the unreached in Open and Distance Learning (ODL) systems has the novelty in it. Nevertheless, this novelty puts challenges to the ODL systems managers, curriculum designers, Self Learning Materials (SLMs) writers, editors, production professionals and may other personnel involved in it. A dedicated team of University of Kalyani under leadership of Hon'ble Vice-Chancellor have puts their best efforts, committed professionalism as a Team for promoting Post Graduate Programmes under distance mode under University of Kalyani. Developing quality printed SLMs for students under DODL within a limited time to cater academic requirements of the Course as per standards set by Distance Education Bureau of the University Grants Commission, New Delhi, India under Open and Distance Mode DEB Regulations, 2020 successfully completed with best efforts.

Utmost care has been taken to develop the SLMs useful to the learners and to avoid errors as far as possible. Further, suggestions from the learners-end will be gracefully admitted and to be appreciated.

During the academic productions of the SLMs, the team received continuously positive stimulations and feedback from Professor (Dr.) Amalendu Bhunia, Hon'ble Vice-Chancellor, University of Kalyani, who kindly accorded directions, encouragements and suggestions, made constructive criticisms to develop it within proper requirements. We gracefully, acknowledge his inspiration and guidance.

Due sincere thanks are being expressed to all the Members of PGBOS (DODL), University of Kalyani, Course Writers- who are serving subject experts serving at University Post Graduate departments and also to the authors and academicians whose academic contributions have been utilized to develop these SLMs. We humbly acknowledge their valuable academic contributions. I would like to convey thanks to all other University dignitaries and personnel who have been involved either in conceptual level or in the operational level of the DODL of University of Kalyani.

For a comprehensive, learners friendly, adaptable text that meets curriculum requirements of the Post Graduate Programme through distance mode.



Kalyani, Nadia, West Bengal

Director
Directorate of Open & Distance Learning
University of Kalyani

**SYLLABUS**
**COURSE – BOTCOR T309**
**Plant Pathology & Crop Protection**

| Course | Group | Details Contents Structure | | Study hour |
|---|---|---|---|---|
| BOTCOR T309 | Plant Pathology & Crop Protection | **Unit 1.** Historical and Developmental Aspects of Plant Pathology. | Historical and developmental aspects of plantpathology | 1 |
| | | **Unit 2.** Inoculum: | Production, liberation and dispersal of inoculum, inoculum potential; factors affecting inoculumpotential. | 1 |
| | | **Unit 3.** Pathogenesis: | Host-pathogen interaction: penetration anddisease development, role of cell-wall degrading enzymes and toxins, recognition mechanism and signal transduction during plant-pathogen interaction. | 1 |
| | | **Unit 4.** DefenseMechanisms of Plants against Infection: | Defense mechanisms of plants against infection: Pre-existing structural and biochemical defense, induced structural and biochemical defense, hypersensitive reaction, phytoalexins, | 1 |
| | | **Unit 5.** Defense Mechanisms of Plants against Infection: | PR proteins, systemic acquired resistance, induced systemic resistance; Gene for gene hypothesis; concept of horizontal and verticalresistance. | 1 |
| | | **Unit 6.** Physiological and MolecularChanges in Diseased Plants. | Physiological (photosynthesis, respiration, translocation of water and nutrients) and molecular (protein and nucleic acid) changes in diseasedplants. | 1 |
| | | **Unit 7.** Predisposition | Predisposition, survival of pathogen in nature and its spread, diseaseepidemics. | 1 |
| | | **Unit 8.** Principles of Plant Disease Control | Principles of plant disease control: exclusion, eradication, protection andtherapy. | 1 |
| | | **Unit 9.** Strategies of Plant Disease Management | Strategies of plant disease management with special emphasis on cultural and biological management; | 1 |
| | | **Unit 10.** Strategies of Plant Disease Management | Plant quarantine; integrated pestmanagement. | 1 |

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|---|---|---|---|---|
| | | **Unit 11.** Fungicides: | Fungicides: types and uses | 1 |
| | | **Unit 12.** Fungicides: | Fungicides: types and uses | 1 |
| | | **Unit 13.** Study of Some Plant Diseases | Fungal diseases: | 1 |
| | | **Unit 14.** Study of Some Plant Diseases | Oomycete and Bacterial diseases: | 1 |
| | | **Unit 15.** Study of Some Plant Diseases | Viral diseases and Disease caused by nematode Disease caused by mycoplasma-like organism | 1 |

**Content**
**COURSE –** BOTCOR T309
**Plant Pathology & Crop Protection**

## Two Year M.Sc Course (CBCS)

# BOTANY

### Course: BOTCOR T309

### (Plant Pathology & Crop Protection)

**DIRECTORATE**

**OF**

**OPEN AND DISTANCE LEARNING**

**UNIVERSITY OF KALYANI**

# Theoreticalcourse

## BOTCOR T309

## Plant Pathology & Crop Protection

**Points:75** **Credits:3**

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**Content Structure**

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**1.** Introduction

**2.** Objectives

**3.** Historical and developmental aspects of plantpathology

**4.** Production, liberation and dispersal of inoculum, inoculum potential; factors affecting inoculumpotential

**5.** Host-pathogen interaction: penetration and disease development, role of cell-wall degrading enzymes and toxins, recognition mechanism and signal transduction during plant-pathogen interaction

**6.** Defense mechanisms of plants against infection: Pre-existing structural and biochemical defense, induced structural and biochemical defense, hypersensitive reaction, phytoalexins, PR proteins, systemic acquired resistance, induced systemic resistance; Gene for gene hypothesis; concept of horizontal and verticalresistance

**7.** Physiological (photosynthesis, respiration, translocation of water and nutrients) and molecular (protein and nucleic acid) changes in diseasedplants.

**8.** Predisposition, survival of pathogen in nature and its spread, diseaseepidemics

**9.** Principles of plant disease control: exclusion, eradication, protection andtherapy

**10.** Strategies of plant disease management with special emphasis on cultural and biological management; plant quarantine; integrated pestmanagement

**11.** Fungicides: types and uses.

**12.** Study of some plant diseases with reference to symptoms, etiology and controlmeasures:

i. Fungal diseases: wart and early blight of potato, downy mildew of grapes, powdery mildew of rose, white rust of crucifers, peach leaf curl, linseed rust, smut and red rot of sugarcane, Tikka

disease of groundnut, Panama disease (*Fusarium* wilt) of banana, blast and sheath blight of paddy.Bacterial diseases: leaf blight of paddy, Moko disease (bacterial wilt) ofbanana

ii. Viral diseases: Tungro viral disease of paddy, mosaic ofpotato

iii. Disease caused by nematode: root knot oftomato

iv. Disease caused by mycoplasma-like organism: little leaf ofbrinjal

**13.** SuggestedReading

**14.** Assignment

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## 1. Introduction:

**Plant pathology** (also **phytopathology**) is the scientific study of diseases in plants caused by pathogens (infectious organisms) and environmental conditions (physiological factors). Organisms that cause infectious disease include fungi, oomycetes, bacteria, viruses, viroids, virus-like organisms, phytoplasmas, protozoa, nematodes and parasitic plants. Not included are ectoparasites like insects, mites, vertebrate, or other pests that affect plant health by consumption of plant tissues. Plant pathology also involves the study of pathogen identification, disease etiology, disease cycles, economic impact, plant disease epidemiology, plant disease resistance, how plant diseases affect humans and animals, pathosystem genetics, and management of plant diseases.

**Crop protection** is the science and practice of managing plant diseases, weeds and other pests (both vertebrate and invertebrate)that damage agricultural crops and forestry. Agricultural crops include field crops (maize, wheat, rice, etc.), vegetable crops (potatoes, cabbages, etc.) and fruits. The crops in field are exposed to many factors. The crop plants may be damaged by insects, birds, rodents, bacteria, etc. Crop protection encompasses:

- Pesticide-based approaches such as herbicides, insecticides and fungicides
- Biological pest control approaches such as cover crops, trap crops and beetle banks
- Barrier-based approaches such as agrotextiles and bird netting
- Animal psychology-based approaches such as bird scarers
- Biotechnology-based approaches such as plant breeding and genetic modification

## 2. Objectives

After completion of the module you will be able to:

 provide an overview of fungi systematics and theories explaining its origin.

 apply this knowledge in understanding the evolutionary significance of fungi and use it as a basis for understanding the evolutionary pathways to other plant groups.

 describe the general characteristics of important groups of algae especially the phytoplankton and explain their ecology, role in environment and in human welfare;

 apply the knowledge and skills acquired to identify various fungi species.

## 3. Historical and developmental aspects of plantpathology:

Plant diseases find mention in the earliest religious literature Rigveda, Atharvanaveda (1500-500 B.C.), the Artha Shashtra of Kautilya (321-186 B.C.). The book "Vraksha Ayurveda" written by Surapal in ancient India contained information on plant diseases. Plant diseases like rust, smut, downy mildew, powdery mildew and blight were mentioned in the Bible. The Greek Philosopher, Theophrastus (370-286 B.C.) was the first to study and write about the diseases of trees, cereals and legumes. In his book **'Enquiry into plants**' Theophrastus has recorded his observations, imaginations and experiences but they were not based on any experiments.

In **1729**, Italian botanist P. A. Micheli proposed that fungi came from spores; father of Mycology. Up to the 18th Century it was believed that microorganisms and their spores formed through spontaneous generation and were the result rather than the cause of disease.

**1775** - Tillet showed that he could increase the number of wheat plants developing covered smut by dusting wheat kernels before planting with smut dust/smut spores.

**1821** - E. M. Fries published Systema Mycologicum for naming of fungi; he was named as Linnaeus of Mycology. 1821 - Robertson of England stated that sulphur is effective against peachmildew.

**Golden Era (1800 – 1900)-** Devastating epidemic, the Late blight (1845) in Ireland dramatized the effect of plant diseases on human suffering besides stimulated interest in their cause and control.

**1845** - Irish Potato famine due to *Phytophthora infestans* caused starvation of million and immigration of 1.5 million people.

**1858** - J. G. Kuhn published first textbook in Plant Pathology – The Diseases of Cultivated Crops, their Causes and theirControl

**1861** -Anton de Bary (Germany) worked out the life cycle of potato late blight and first to prove experimentally *Phytophthora infestans* is the cause of potato late blight. He proved that fungi are causes but not the results of diseases. He is the Father of Modern Plant Pathology.

**1918** - E. J. Butler published book on Fungi and Disease in Plants; he made exhaustive study on Indian fungi and the diseases caused by them. He is called as the Father of Modern Plant Pathology in India; He joined as the first Director of Imperial Bureau of Mycology (Commonwealth Mycological Institute, CMI) now CAB International Mycological Institute in Kew, England in 1920. He began the journal Review of Applied Mycology; with S.G. Jones he wrote, 'Plant Pathology' in 1949

**1948** -B. B. Mundkur started Indian Phytopathological Society with its journal Indian Phytopathology. He has written a book 'Fungi and Plant Diseases' in 1949, which is the second, book in plant pathology in India.

**1951-57** -E. A. Gaümann was one of the first to investigate the physiology of the wilts caused by *Fusarium* spp. He put forth the involvement of toxin (toxin theory) in wilt diseases

**1961** - Ross – Coined the term SAR, resistance that developed in the untreated portion of TMV inoculated plants

**1966** -van Schmeling and Marshall Kulka were the first to find out systemic fungicides (oxathiin compounds – carboxin and oxycarboxin).

**1970** -S. D. Garrett investigated the management of root diseases and he is the pioneer worker in the field of biological control.

**1972** – G. Rangaswami wrote a book on Diseases of Crop Plants in India.

**1985** - Van Loon – Isolated first pathogenesis related proteins (PR protein).

**1980s onward** - Advent of Molecular Plant Pathology – detailed studies on plant-pathogen interaction, host-pathogen recognition, signalling pathways undertaken.

## 4. Production, liberation and dispersal of inoculum, inoculum potential; factors affecting inoculumpotential

In every infectious disease a series of more or lessdistinct events occurs in succession and leads to thedevelopment and perpetuation of the disease and thepathogen. This chain of events is called a **disease cycle**.

A disease cycle sometimes corresponds fairly closely tothe **life cycle** of the pathogen, but it refers primarily tothe appearance, development, and perpetuation of thedisease as a function of the pathogen rather than to thepathogen itself. The disease cycle involves changes inthe plant and its symptoms as well as those in thepathogen and spans periods within a growing seasonand from one growing season to the next. The primaryevents in a disease cycle are inoculation, penetration,establishment of infection, colonization (invasion),growth and reproduction of the pathogen, disseminationof the pathogen, and survival of the pathogen in theabsence of the host, i.e., overwintering or oversummering of the pathogen. In somediseases there may be several **infection cycles** within onedisease cycle.

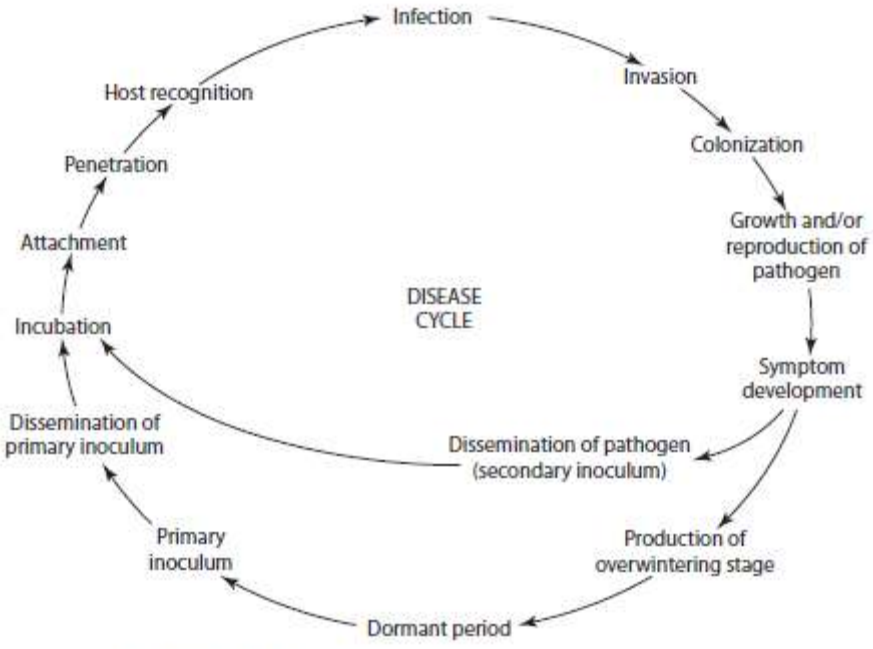



FIGURE 2-2 Stages in development of a disease cycle.

**Inoculation:**

Inoculationis the initial contact of a pathogen with asite of plant where infection is possible. The pathogen(s) that lands on or is otherwise brought into contact withthe plant is called the **inoculum**. The inoculum is anypart of the pathogen that can initiate infection. Thus, infungi the inoculum may be spores (Figs. 2-3A–2-3C),**sclerotia** (i.e., a compact mass of mycelium), or fragmentsof mycelium. In bacteria, mollicutes, protozoa,viruses, and viroids, the inoculum is always whole individualsof bacteria (Fig. 2-3D), mollicutes, protozoa,viruses, and viroids, respectively. In nematodes, theinoculum may be adult nematodes, nematode juveniles,or eggs. In parasitic higher plants, the inoculum may beplant fragments or seeds. The inoculum may consist ofa single individual of a pathogen, e.g., one spore or onemulticellular sclerotium, or of millions of individuals ofa pathogen, e.g., bacteria carried in a drop of water. Oneunit of inoculum of any pathogen is called a **propagule**.

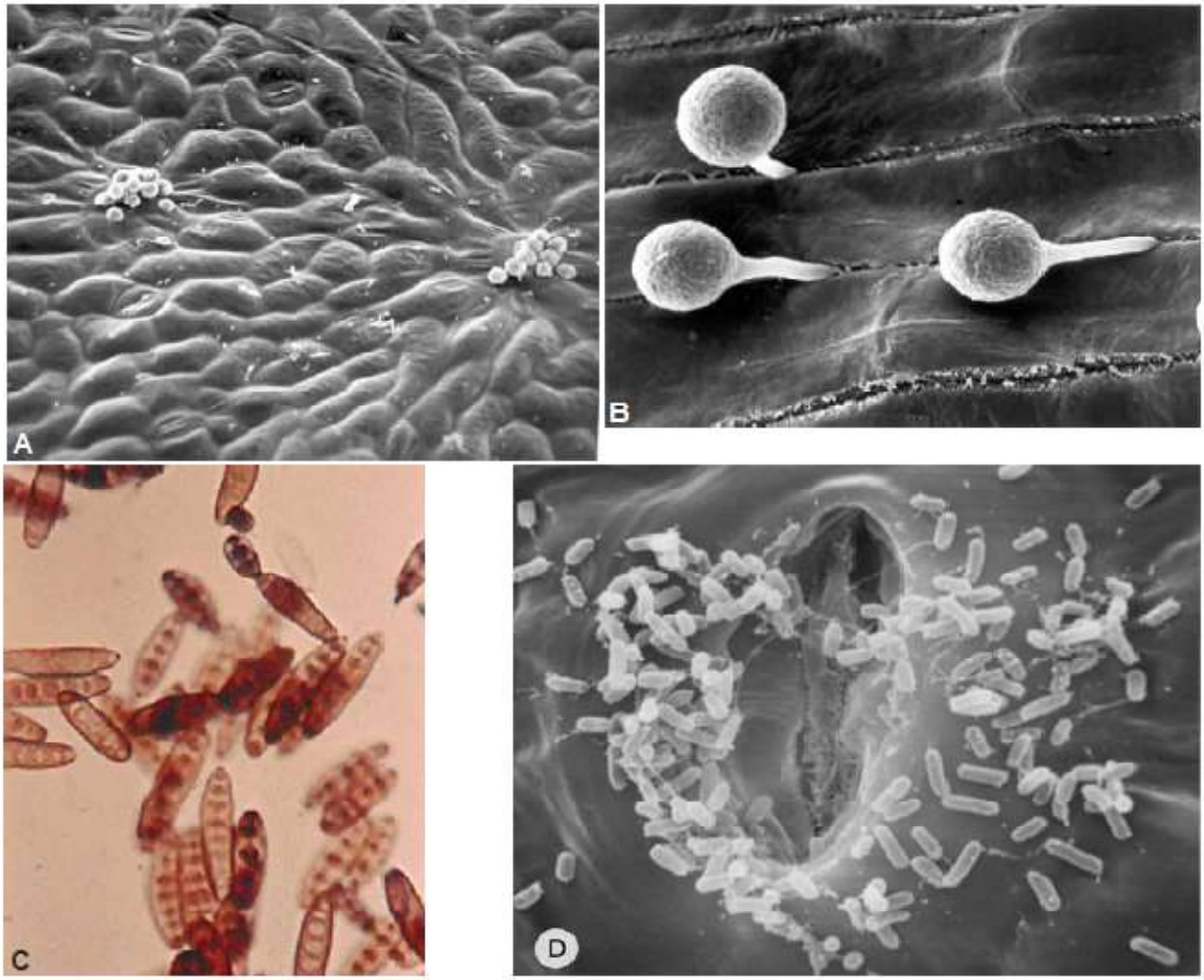



FIGURE 2-3 Types of inoculum and ways in which some pathogens enter a host plant. (A) Two groups of zoospores of the grape downy mildew oomycete have gathered over two leaf stomata. (B) Encysted zoospores of the soybean root rot pathogen *Phytophthora sojae* germinating and penetrating the root. (C) Mitospores (conidia) of a fungus that causes a corn leaf spot disease. (D) Bacteria of *Pseudomonas syringae* that causes bacterial spot and canker of stone fruits are seen in and surrounding a stoma of a cherry leaf. [Photographs courtesy of (A) D. J. Royle, (B) C. W. Mims and K. Enkerli, University of Georgia, and (D) E. L. Mansvelt, Stellenbosch, South Africa.]

**Types of Inoculum**

An inoculum that survives dormant in the winter orsummer and causes the original infections in the springor in the autumn is called a **primary inoculum**, and theinfections it causes are called **primary infections**. Aninoculum produced from primary infections is called a**secondary inoculum** and it, in turn, causes **secondaryinfections**. Generally, the more abundant the primaryinoculum and the closer it is to the crop, the more severethe disease and the losses that result.

**Sources of Inoculum**

In some fungal and bacterial diseases of perennialplants, such as shrubs and trees, the inoculum is produced

on the branches, trunks, or roots of the plants.The inoculum sometimes is present right in the plant debris or soil in the field where the crop is grown; othertimes it comes into the field with the seed, transplants,tubers, or other propagative organs or it may come fromsources outside the field. Outside sources of inoculummay be nearby plants or fields or fields many milesaway. In many plant diseases, especially those of annualcrops, the inoculum survives in perennial weeds or alternatehosts, and every season it is carried from them tothe annual and other plants. Fungi, bacteria, parasitichigher plants, and nematodes either produce their inoculumon the surface of infected plants or their inoculumreaches the plant surface when the infected tissue breaksdown. Viruses, viroids, mollicutes, fastidious bacteria,and protozoa produce their inoculum within the plants;such an inoculum almost never reaches the plant surfacein nature and, therefore, it can be transmitted from oneplant to another almost entirely by some kind of vector,such as an insect.

**Landing or Arrival of Inoculum:**

The inoculum of most pathogens is carried to hostplants passively by wind, water, and insects. An airborneinoculum usually gets out of the air and onto the plantsurface not just by gravity but by being washed out byrain. Only a tiny fraction of the potential inoculum producedactually lands on susceptible host plants; the bulkof the produced inoculum lands on things that cannot become infected. Some types of inoculum in the soil,e.g., zoospores and nematodes, may be attracted tothe host plant by such substances as sugars andamino acids diffusing out of the plant roots. Vectortransmittedpathogens are usually carried to their hostplants with an extremely high efficiency.

**Prepenetration Phenomena**

**Attachment of Pathogen to Host**

Pathogens such as mollicutes, fastidious bacteria, protozoa,and most viruses are placed directly into cells ofplants by their vectors and, in most cases, they are probablyimmediately surrounded by cytoplasm, cytoplasmicmembranes, and cell walls. However, almost all fungi,bacteria, and parasitic higher plants are first broughtinto contact with the external surface of plant organs.

Before they can penetrate and colonize the host, theymust first become attached to the host surface (Figs.2-3–2-6). Attachment takes place through the adhesionof spores, bacteria, and seeds through adhesivematerials that vary significantly in composition andin the environmental factors they need to becomeadhesive. Disruption of adhesion by nontoxic syntheticcompounds results in failure of the spores to infectleaves. In any case, the act of attachment often seems necessaryfor the subsequent transmission of signals for germ tubeextension and production of infection structure. It isnow clear that many proteins of the fungal cell wall, inaddition to their structural role, play an important rolein the adhesion of fungi, as well as in the host-surfaceperception by the fungus.

**Spore Germination and Perception of the Host Surface**

It is not clear what exactly triggers spore germination,but stimulation by the contact with the host surface,hydration and absorption of low molecular weightionic material from the host surface, and availability ofnutrients play a role. Spores also have mechanisms thatprevent their germination until they sense such stimulationsor when there are too many spores in their vicinity.

Once the stimulation for germination has beenreceived by the spore, the latter mobilizes its stored foodreserves, such as lipids, polyoles, and carbohydrates,and directs them toward the rapid synthesis of cell membraneand cell wall toward the germ tube formation andextension (Figs. 2-4 and 2-5). The germ tube is a specializedstructure distinct from the fungal mycelium,often growing for a very short distance before it differentiatesinto an appressorium.

The germ tube is also thestructure and site that perceives the host surface and, ifit does not receive the appropriate external stimuli, thegerm tube remains undifferentiated and, when the nutrientsare exhausted, it stops growing. When appropriatephysical and chemical signals, such as surface hardness,hydrophobicity, surface topography, and plant signals,are present, germ tube extension and differentiation takeplace.

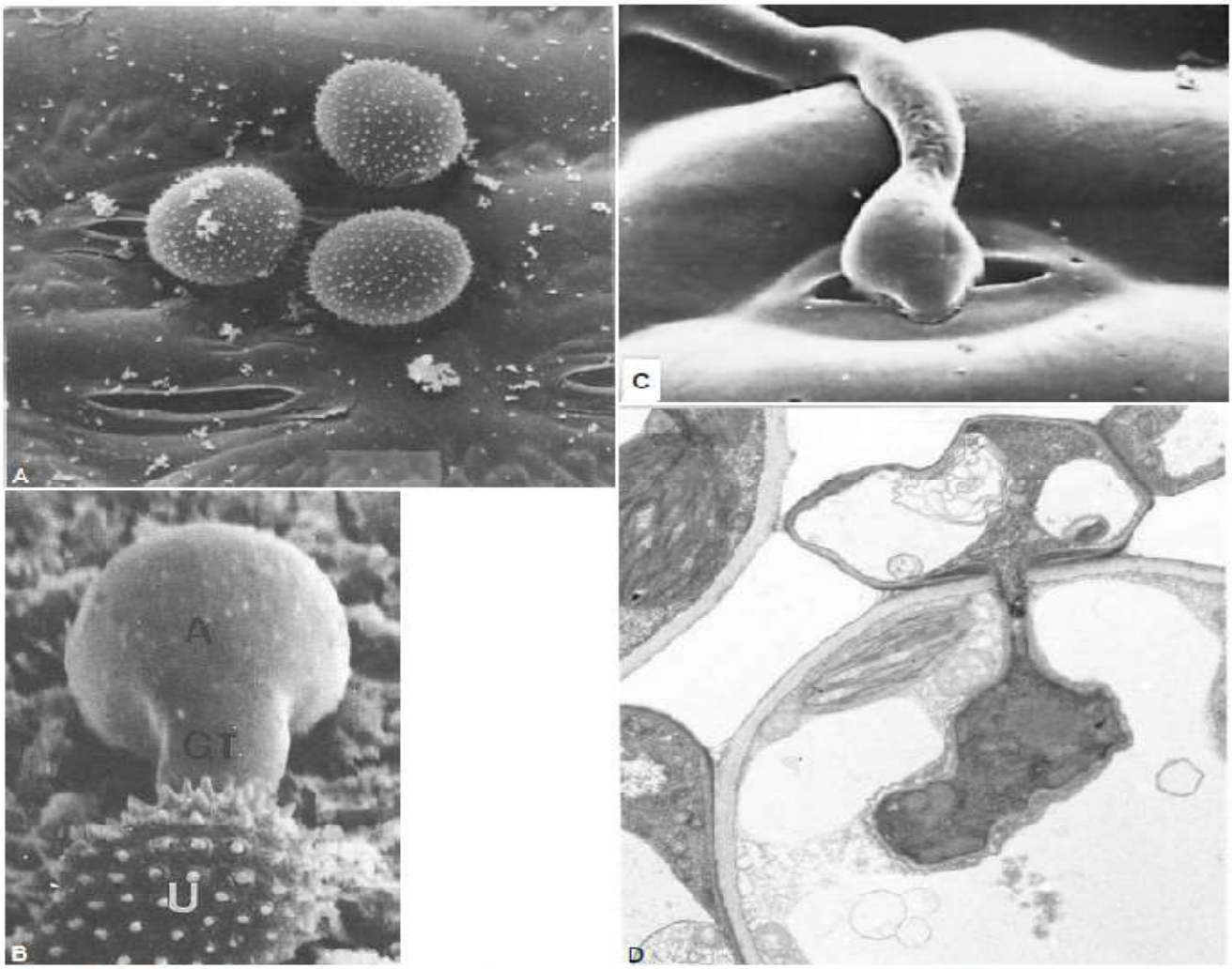



FIGURE 2-4 Methods of germination and penetration by fungi. (A) Uredospores of a rust fungus on a grass leaf next to open stomata. (B) A rust uredospore (U) that has germinated and produced a dome-like appressorium. (C) Uredospore germination, germ tube elongation, and appressorium penetration through a stoma. (D) A haustorium of a rust fungus inside a host cell. (E) A spore of the apple black rot fungus that has germinated directly into mycelium. (F) Two multicellular conidia of *Alternaria* sp. (G) A germinating conidium of *Alternaria* with a germ tube covered with extracellular material. [Photographs courtesy of (A) Plant Pathology Department, University of Florida, (B and C) W. K. Wynn and (D) C. W. Mims, University of Georgia, (E) J. Rytter and J. W. Travis, Pennslyvania State University, (F and G) Mims *et al.* (1997). *Can. J. Bot.* 75, 252–260.]

The perception of signals from plant surfaces bypathogenic fungi (Fig. 2-6) seems to be the result ofsignaling pathways mediated by cyclic adenosinemonophosphate (cAMP) and mitogen-activated proteinkinase (MAPK), which have been implicated in regulatingthe development of infection-related phenomena inmany different fungi. In response to a signal from thehost plant, e.g., the presence of a hydrophobic plantsurface, which transmits a cue for appressorium formation,the fungus perceives the extracellular signal and itstransmission via the plasma membrane and, as a firststep, it accumulates intracellular signaling moleculesand induces a phosphorylation cascade. In some fungi,the receptor of

the signal is a protein in the plasma membraneof the fungal spore. Signaling pathways for infection-related developmentare also achieved through mitogen-activated proteinkinases (**MAPKs**) and their upstream regulatory kinases.

All of these together comprise a functional unit thattransmits input signals from the periphery of the cell tothe cell nucleus to elicit the expression of appropriategenes. A MAP kinase, K1 or P1, regulates appressoriumformation in response to a signal from the plant surfacebut it is also required for invasive growth or viability inits host plant.

After attachment of the propagule to the host surface,as spores and seeds germinate, germ tubes also producemucilaginous materials that allow them to adhere to thecuticular surface of the host, either along their entirelength or only at the tip of the germ tube. In regions ofcontact with the germ tube, the structure of the hostcuticle and cell walls often appears altered, presumablyas a result of degradative enzymes contained in themucilaginous sheath.

**Appressorium Formation and Maturation**

Once appressoria are formed, they adhere tightly to theleaf surface (Figs. 2-4 and 2-9). Subsequently, appressoriasecrete extracellular enzymes, generate physicalforce, or both to bring about penetration of the cuticleby the fungus. Appressoria must be attached to the hostplant surface strongly enough to withstand the invasivephysical force applied by the fungus and to resist thechemical action of the enzymes secreted by the fungus.

Appressoria of some fungi contain lipids, polysaccharides,and proteins. Fungi that produce melaninpigmentedappressoria produce a narrow penetrationhypha from the base of the appressorium and use primarilyphysical force to puncture the plant cuticle withthat hypha. Mobilization of spore-storedproducts to the developing appressorium and glycerolbiosynthesis in it is regulated by the cAMP signalling pathway, whereas the initial movement of lipid andglycogen reserves to the developing appressorium wasalso found to be regulated by the K1 MAP. Thisindicates that the maturation of appressoria and theirspecific biochemical activity are intimately associatedwith genetic control of the initial development ofappressoria.

The production of penetration hyphae by appressoria,or directly from germ tubes, is not well understoodat the genetic level. Production of the penetration pegrequires the localization of actin to the hyphal tip andrapid biosynthesis of the cell wall as the hypha growsthrough the cuticle and the layers of the epidermal cellwalls. Production of penetration hyphae appears to beregulated by a MAP kinase pathway.

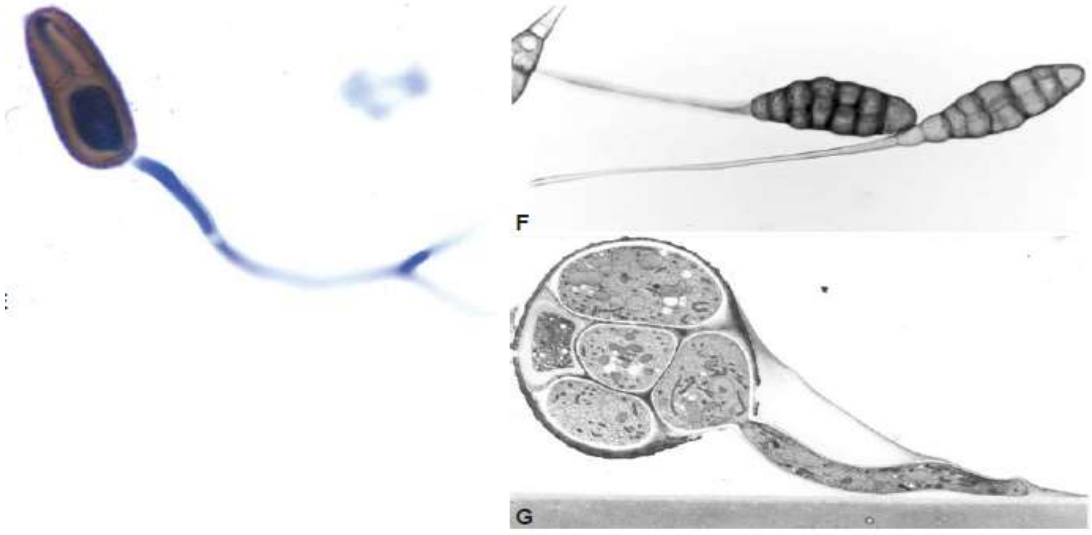



FIGURE 2-4 (*Continued*)

**Recognition between Host and Pathogen**

It is still unclear how pathogens recognize their hostsand vice versa. It is assumed that when a pathogencomes in contact with a host cell, an early event takesplace that triggers a fairly rapid response in each organismthat either allows or impedes further growth of thepathogen and development of disease. The nature ofthe "early event" is not known with certainty in anyhost–parasite combination, but it may be one of manybiochemical substances, structures, and pathways.

These may include specific host signal compounds orstructures, or specific pathogen elicitor molecules, andeither of them may induce specific actions or formationof specific products by the other organism (Fig. 2-6).

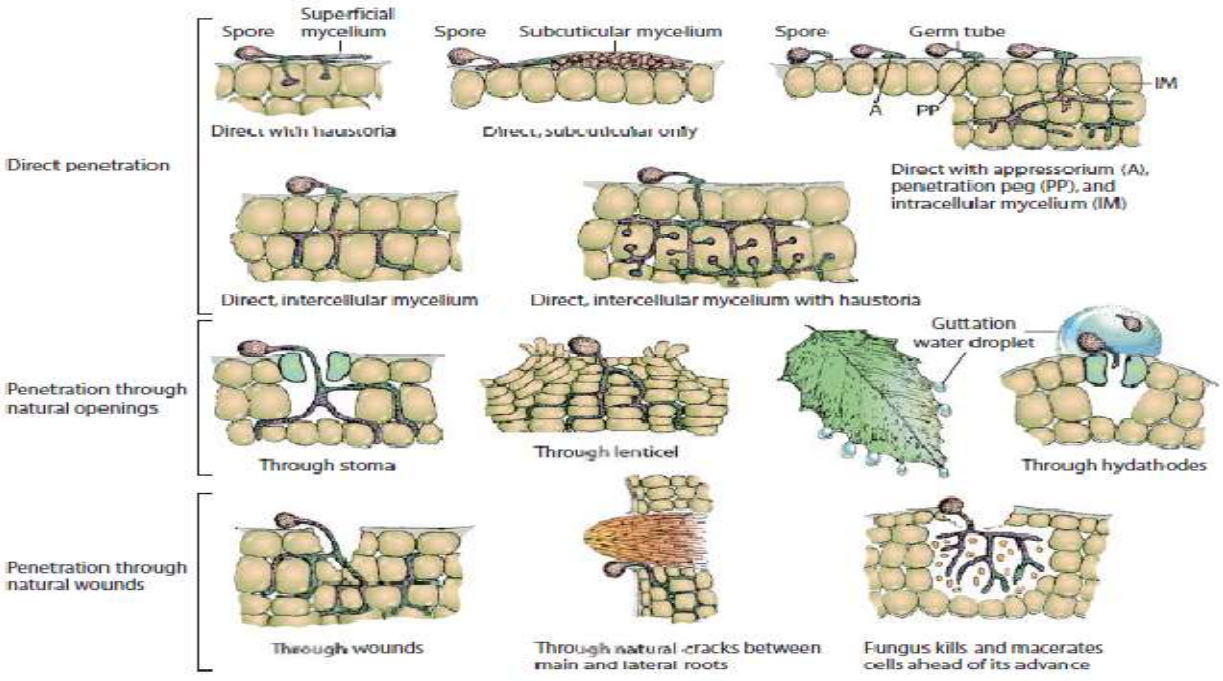



FIGURE 2-5 Methods of penetration and invasion by fungi.

**Germination of Spores and Seeds**

Almost all pathogens in their vegetative state are capableof initiating infection immediately. Fungal spores andseeds of parasitic higher plants, however, must firstgerminate (Figs. 2-4 and 2-5). Spores germinate byproducing a typical mycelium (Figs. 2-4E and 2-4G) thatinfects and grows into host plants or they produce ashort germ tube that produces a specialized infectiousstructure, the haustorium (Figs. 2-4B–2-4D).

In order togerminate, spores require a favourable temperature andalso moisture in the form of rain, dew, or a film of wateron the plant surface or at least high relative humidity.The moist conditions must last long enough for thepathogen to penetrate or else it desiccates and dies. Mostspores can germinate immediately after their maturationand release, but others (so-called resting spores) requirea dormancy period of varying duration before they cangerminate. When a spore germinates it produces a germtube, i.e., the first part of the mycelium,that can penetratethe host plant. Some fungal spores germinateby producing other spores, e.g., sporangia producezoospores and teliospores produce basidiospores. Spore germination is often favoured by nutrients diffusingfrom the plant surface; the more nutrients (sugarsand amino acids) exuded from the plant, the morespores germinate and the faster they germinate. In somecases, spore germination of a certain pathogen is stimulatedonly by exudates of plants susceptible to that particularpathogen. In other cases, spore germination maybe inhibited to a lesser or greater extent by materials released into the surrounding water by the plant, bysubstances contained within the spores themselves,especially when the spores are highly concentrated(“quorum sensing”), and by saprophytic microflorapresent on or near the plant surface.

**Penetration**

Pathogens penetrate plant surfaces by direct penetrationof cell walls, through natural openings, or throughwounds (Figs. 2-3–2-5). Some fungi penetrate tissues inonly one of these ways, others in more than one. Bacteriaenter plants mostly through wounds, less frequentlythrough natural openings, and never directlythrough unbroken cell walls (Fig. 2-5). Viruses, viroids,mollicutes, fastidious bacteria, and protozoa enterthrough wounds made by vectors, although someviruses and viroids may also enter through woundsmade by tools and other means. Parasitic higher plantsenter their hosts by direct penetration. Nematodes enterplants by direct penetration and, sometimes, throughnatural openings (Fig. 2-10).

Penetration does not always lead to infection. Manyorganisms actually penetrate cells of plants that are notsusceptible to these organisms and that do not becomediseased; these organisms cannot proceed beyond thestage of penetration and die without producing disease.

**Infection**

**Infection** is the process by which pathogens establishcontact with susceptible cells or tissues of the host andprocure nutrients from them. Following infection,pathogens grow, multiply, or both within the planttissues and invade and colonize the plant to a lesser orgreater extent. Growth and/or reproduction of thepathogen (colonization) in or on infected tissues areactually two concurrent substages of disease development(Fig. 2-2).

Successful infections result in the appearance ofsymptoms, i.e., discolored, malformed, or necrotic areason the host plant. Some infections, however, remainlatent, i.e., they do not produce symptoms right awaybut at a later time when the environmental conditionsor the stage of maturity of the plant become morefavorable.

All the visible and otherwise detectable changes in theinfected plants make up the **symptoms** of the disease.

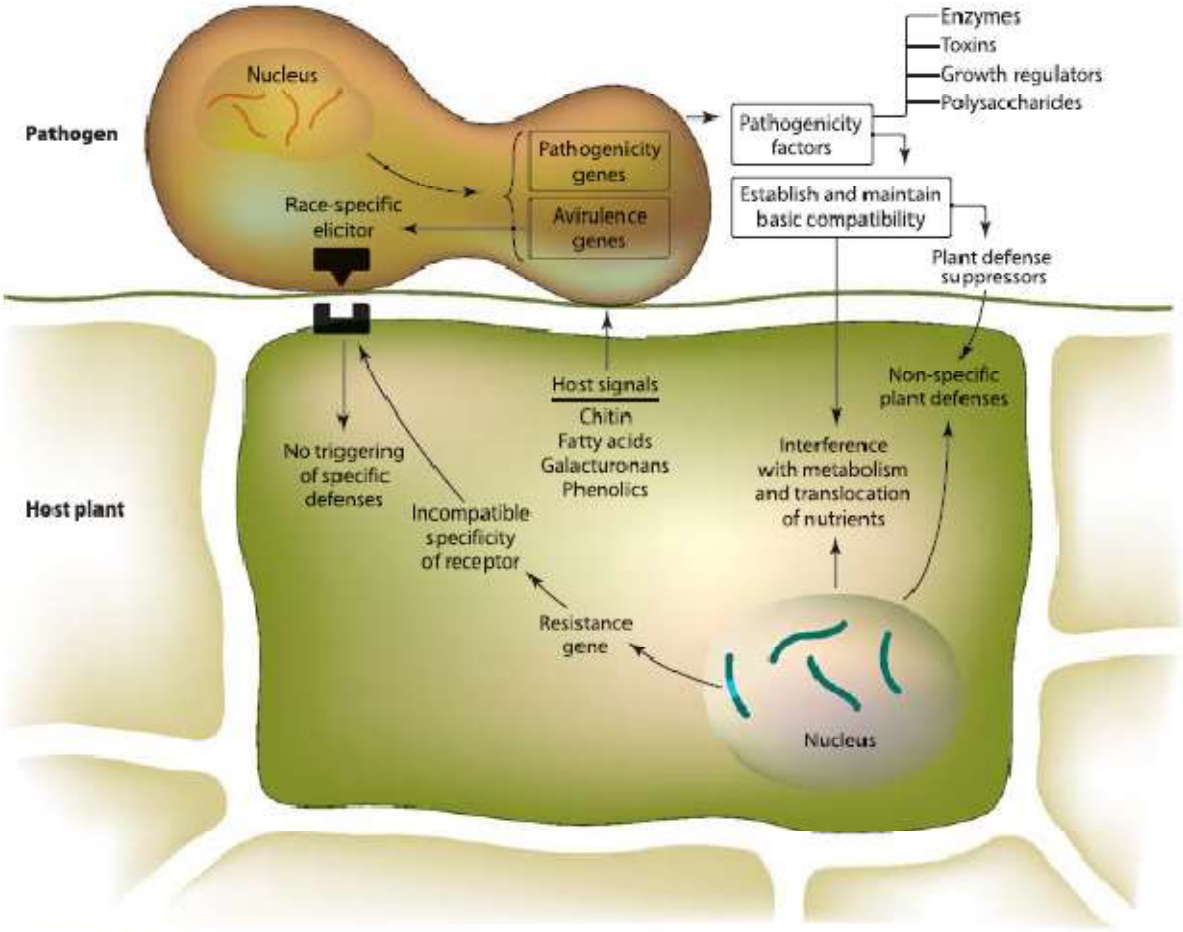



FIGURE 2-6 Establishment of infection in a compatible reaction between a pathogen and its host plant.

Symptoms may change continuously from the momentof their appearance until the entire plant dies or theymay develop up to a point and then remain more or lessunchanged for the rest of the growing season. Symptomsmay appear as soon as 2 to 4 days after inoculation, ashappens in some localized viral diseases of herbaceousplants, or as late as 2 to 3 years after inoculation, as inthe case of some viral, mollicute, and other diseases oftrees. In most plant diseases, however, symptoms appearfrom a few days to a few weeks after inoculation.

**Invasion**

Various pathogens invade hosts in different ways and todifferent extents (Figs. 2-4, 2-5, 2-9, and 2-12). Somefungi, such as those causing apple scab and black spotof rose, produce mycelium that grows only in the areabetween the cuticle and the epidermis (subcuticularcolonization) (Fig. 2-11A); others, such as those causingpowdery mildews, produce mycelium only on thesurface of the plant (Fig. 2-11B) but send haustoria intothe epidermal cells. Most fungi spread into all the tissuesof the plant organs (leaves, stems, and roots) they infect,either by growing directly through the cells as an **intracellularmycelium** or by growing between the cells as an**intercellular mycelium** (Figs. 2-11C and 2-11D). Fungithat cause vascular wilts invade the xylem vessels ofplants (Fig. 2-11E).Bacteria invade tissues intercellulary, although whenparts of the cell walls dissolve, bacteria also grow intracellularly.Bacteria causing vascular wilts, like the vascularwilt fungi, invade the xylem vessels (Fig. 2-11E).

Most nematodes invade tissues intercellularly, but somecan invade intracellularly as well (Fig. 2-12). Manynematodes do not invade cells or tissues at all but feedby piercing epidermal cells with their stylets.

Viruses, viroids, mollicutes, fastidious bacteria, andprotozoa invade tissues by moving from cell to cell intracellularly.Viruses and viroids invade all types of livingplant cells, mollicutes and protozoa invade phloem sievetubes and perhaps a few adjacent phloem parenchymacells, and most fastidious bacteria invade xylem vesselsand a few invade only phloem sieve tubes.

Many infections caused by fungi, bacteria, nematodes,viruses, and parasitic higher plants are local, i.e.,they involve a single cell, a few cells, or a small areaof the plant. These infections may remain localizedthroughout the growing season or they may enlargeslightly or very slowly. Other infections enlarge more orless rapidly and may involve an entire plant organ(flower, fruit, leaf), a large part of the plant (a branch),or the entire plant.

Infections caused by fastidious xylem- or phloeminhabitingbacteria, mollicutes, and protozoa andnatural infections caused by viruses and viroids are **systemic**,i.e., the pathogen, from one initial point in aplant, spreads and invades most or all susceptible cellsand tissues throughout the plant.

**Growth and Reproduction of the Pathogen(Colonization)**

Individual fungi and parasitic higher plants generallyinvade and infect tissues by growing on or into themfrom one initial point of inoculation. Most of thesepathogens, whether inducing a small lesion, a largeinfected area, or a general necrosis of the plant, continueto grow and branch out within the infected host indefinitelyso that the same pathogen individual spreads intomore and more plant tissues until the spread of the infectionis stopped or the plant is dead. In some fungal infections,however, while younger hyphae continue to growinto new healthy tissues, older ones in the alreadyinfected areas die out and disappear so that a diseasedplant may have several points where separate units ofthe mycelium are active. Also, fungi causing vascularwilts often invade plants by producing and releasingspores within the vessels, and as the spores are carried in the sap stream they invade vessels far away from themycelium, germinate there, and produce a mycelium,which invades more vessels.

All other pathogens, namely bacteria, mollicutes,viruses, viroids, nematodes, and protozoa, do notincrease much, if at all, in size with time, as their sizeand shape remain relatively unchanged throughout theirexistence. These pathogens invade and infect new tissueswithin the plant by reproducing at a rapid rate andincreasing their numbers tremendously in the infectedtissues.The rate of reproduction varies considerably amongthe various kinds of pathogens, but in all types, one ora few pathogens can produce tremendous numbers ofindividuals within one growing season. Some fungiproduce spores more or less continuously (Fig. 2-14),whereas others produce them in successive crops. Ineither case, several thousand to several hundreds of thousands of spores may be produced per squarecentimeter of infected tissue. Even small specializedsporophores can produce millions of spores, and thenumber of spores produced per diseased plant is oftenin the billions or trillions (Fig. 2-14). The number ofspores produced in an acre of heavily infected plants,therefore, is generally astronomical, and enough sporesare released to land on every conceivable surface in thefield and the surrounding areas, enough to easily inoculatewith a heavy inoculum every plant in the area.

**Dissemination of the Pathogen**

A few pathogens, such as nematodes, oomycetes, zoosporicfungi, and bacteria, can move short distances ontheir own power and thus can move from one host toanother one very close to it. Fungal hyphae cangrow between tissues in contact and sometimes throughthe soil toward nearby roots for a few too many centimetres.

Both of these means of dissemination, however,are quite limited, especially in the case of zoospores andbacteria.The spores of some fungi are expelled forcibly fromthe sporophore or sporocarp by a squirting or puffingaction that results in the successive or simultaneous dischargeof spores up to a centimeter or so above thesporophore. The seeds of some parasitic plants are alsoexpelled forcibly and may arch over distances of severalmeters.

Almost all dissemination of pathogens responsible forplant disease outbreaks, and even for disease occurrencesof minor economic importance, is carried outpassively by such agents as air and insects (Figs. 2-13–2-15). To a lesser extent, water, certain other animals,and humans may be involved (Fig. 2-15).

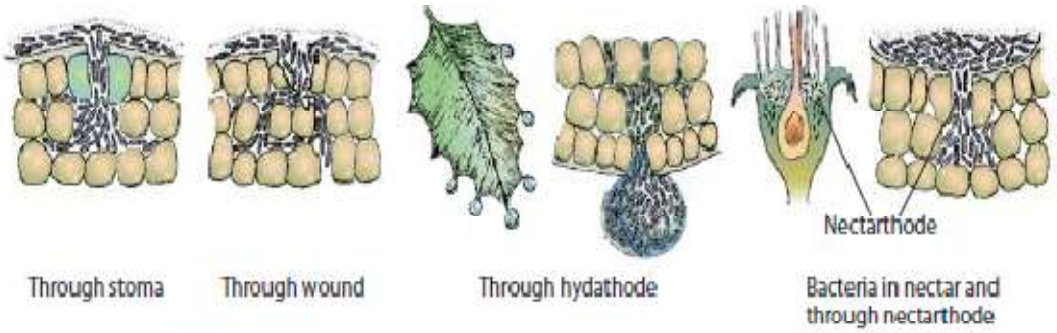



FIGURE 2-7 Methods of penetration and invasion by bacteria.

**Dissemination by Air**

Spores of most oomycetes and most fungi and the seedsof most parasitic plants are disseminated by air currentsthat carry them as inert particles to various distances.

Air currents pick up spores and seeds off the sporophores(Figs. 2-13A–2-13E, 2-14, and 2-16) or whilethey are being expelled forcibly or are falling at maturity.Depending on the air turbulence and velocity, aircurrents may carry the spores upward or horizontally ina way similar to that of particles contained in smoke.

While airborne, some of the spores may touch wet surfacesand get trapped; when air movement stops orwhen it rains, the rest of the spores land or are "**washedout**" from the air and are brought down by the raindrops.

Most of the spores, of course, land on anythingbut a susceptible host plant. Also, the spores of manyfungi are actually too delicate to survive a long tripthrough the air and are therefore successfully disseminatedthrough the air for only a few hundred or a fewthousand meters. The spores of other fungi, however,particularly those of the cereal rusts, are very hardy andoccur commonly at all levels and at high altitudes(several thousand meters) above infected fields. Sporesof these fungi are often carried

over distances of severalkilometers, even hundreds of kilometers, and in favourable weather may cause widespread epidemics. Somefungi can spread into new areas quite rapidly and maycause severe epidemics over large areas, including entirecontinents, within a few years. This happened, forexample, in the airborne pathogens of sugar cane smutin the Americas (Fig. 2-18) and of barley stripe rust inSouth America (Fig. 2-15).

Air dissemination of other pathogens occurs ratherinfrequently and only under special conditions, or indirectly. For example, bacteria causing fire blight of appleand pear produce fine strands of dried bacterial exudatecontaining bacteria, and these strands may be brokenoff and disseminated by wind. Bacteria and nematodespresent in the soil may be blown away along with plantdebris or soil particles in the dust. Wind also helps inthe dissemination of bacteria, fungal spores, and nematodesby blowing away rain splash droplets containingthese pathogens, and wind carries away insects thatmay contain or are smeared with viruses, bacteria,mollicutes, protozoa, or fungal spores. Finally, windcauses adjacent plants or plant parts to rub against oneanother, which may help the spread by contact of bacteria,fungi, some viruses and viroids, and possibly somenematodes.

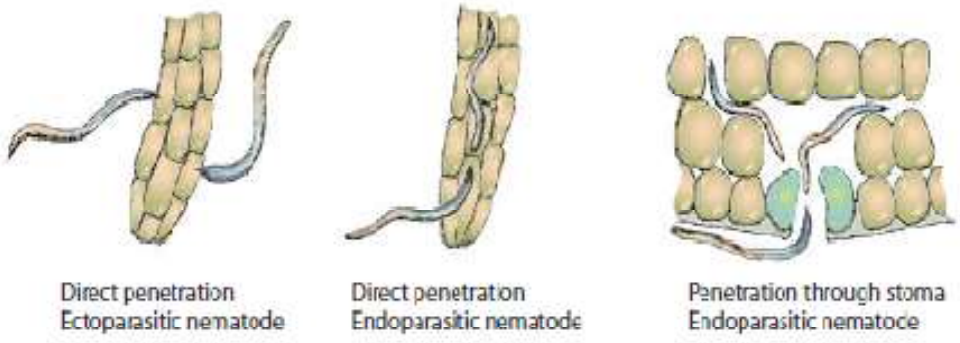



FIGURE 2-10 Methods of penetration and invasion by nematodes.

**Dissemination by Water**

Water is important in disseminating pathogens in threeways. (1) Bacteria, nematodes, and spores and mycelialfragments of fungi present in the soil are disseminatedby rain or irrigation water that moves on the surface orthrough the soil. (2) All bacteria and the spores of manyfungi are exuded in a sticky liquid (Figs. 2-16A, 2-16B,and 2-16D) and depend on rain or (overhead) irrigationwater, which either washes them downward or splashesthem in all directions, for their dissemination (3) Raindropsor drops from overhead irrigation pick up thefungal spores and any bacteria present in the air andwash them downward, where some of them may landon susceptible plants. Although water is less importantthan air in the long-distance transport of pathogens, thewater dissemination of pathogens is more efficient

fornearby infections, as the pathogens land on an alreadywet surface and can move or germinate immediately.

**Dissemination by Insects, Mites, Nematodes, andOther Vectors**

Insects, particularly aphids, leafhoppers, and whiteflies,are by far the most important vectors of viruses, whereasleafhoppers are the main vectors of mollicutes, fastidiousbacteria, and protozoa. Each one of these pathogensis transmitted, internally, by only one or a few speciesof insects during feeding and movement of the insectvectors from plant to plant. Specific insects also transmitcertain fungal, bacterial, and nematode pathogens,such as the fungus causing Dutch elm disease, the bacterialwilt of cucurbits, and the pine wilt nematode. Inall diseases in which the pathogen is carried internallyor externally by one or a few specific vectors, disseminationof the pathogen depends, to a large extent orentirely, on that vector. In many diseases, however, suchas bacterial soft rots, fungal fruit rots, anthracnoses, andergot, insects become smeared with various kinds ofbacteria or sticky fungal spores as they move amongplants. The insects carry these pathogens externallyfrom plant to plant and deposit them on the plantsurface or in the wounds they make on the plants duringfeeding. In such diseases, dissemination of the pathogenis facilitated by but is not dependent on the vector.

Insects may disseminate pathogens over short or longdistances, depending on the kind of insect, theinsect–pathogen association, and the prevailing weatherconditions, particularly wind.A few species of mites and nematodes can transmitinternally several viruses from plant to plant. In addition,mites and nematodes probably carry externallybacteria and sticky fungal spores with which theybecome smeared as they move on infected plantsurfaces.

Almost all animals, small and large, that move amongplants and touch the plants along the way can disseminatepathogens such as fungal spores, bacteria, seeds ofparasitic plants, nematodes, and perhaps some virusesand viroids. Most of these pathogens adhere to the feetor the body of the animals, but some may be carried incontaminated mouthparts.

Finally, some plant pathogens, e.g., the zoospores ofsome fungi and certain parasitic plants, can transmitviruses as they move from one plant to another(zoospores) or as they grow and form a bridge betweentwo plants (dodder).

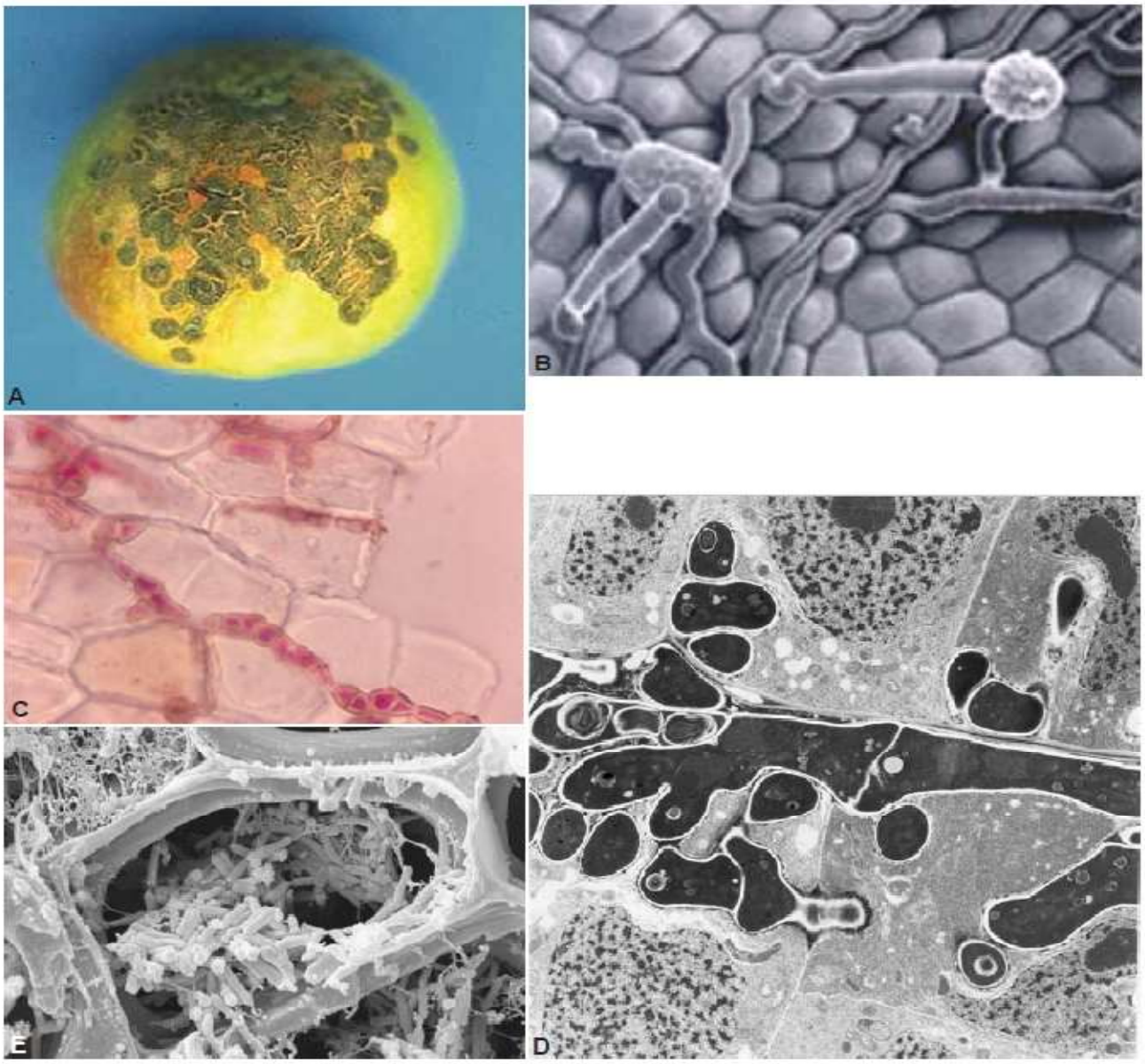



**FIGURE 2-11** Types of invasion of pathogens in infected plants. (A) In apple scab disease, the pathogenic fungus grows only between the cuticle and the epidermal cells of leaves and fruit. (B) In powdery mildews the fungal mycelium grows only on the surface of host plants, but sends haustoria into the epidermal cells. (C) In many diseases the fungal mycelium (stained red here) grows only intercellularly (between the cells). (D) Hyphae of the smut fungus *Ustilago* in an infected leaf. (E) In bacterial vascular diseases, bacteria grow in and may clog the xylem vessels. [Photographs courtesy of (A) University of Oregon, (B) G. Celio, APS, (D) Mims *et al.* (1992). *Intern. J. Plant Sci.* **153**, 289–300, and (E) E. Alves, Federal University of Lavras, Brazil.]

**Dissemination by Pollen, Seed, Transplants, Budwoodand Nursery Stock**

Some viruses are carried in the pollen of plants infectedwith these viruses and, when virus-carrying pollenpollinates a healthy plant, the virus may infect not onlythe seed produced from such pollination, which willthen grow into a virus-infected plant, it may also infectthe plant that was pollinated with the virus-carryingpollen.

Many pathogens are present on or in seeds, transplants,budwood, or nursery stock and are disseminatedby them as the latter are transported to other fields orare sold and transported to other areas near and far. Disseminationof pathogens through seed, transplants, andso on is of great practical importance because it introducesthe pathogen along with the plant at the beginningof the growth season and enables the pathogen tomultiply and be disseminated by all the other means ofspread discussed. It is also important because it bringspathogens into new areas where they may have neverexisted before.

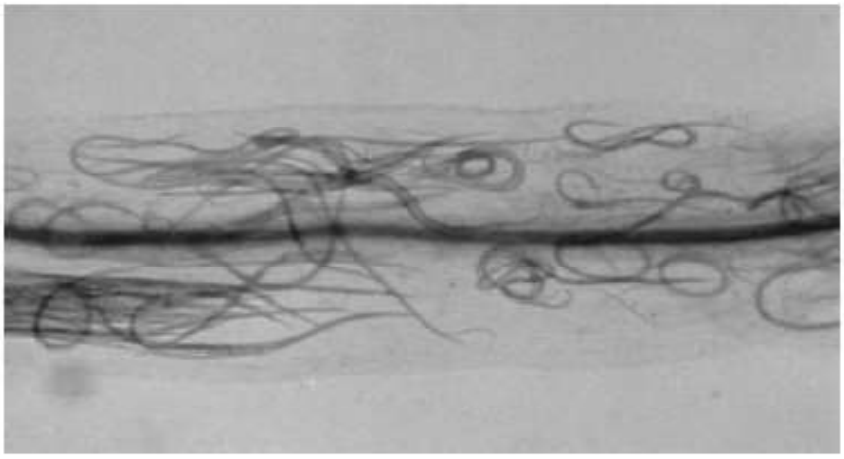


FIGURE 2-12 Alfalfa shoot invaded by plant parasitic nematodes (*Ditylenchus dipsaci*). (Photograph courtesy of J. Santo.)

**Dissemination by Humans**

Human beings disseminate all kinds of pathogens overshort and long distances in a variety of ways. Within afield, humans disseminate some pathogens, such astobacco mosaic virus, through the successive handlingof diseased and healthy plants. Other pathogens are disseminatedthrough tools, such as pruning shears, contaminatedwhen used on diseased plants (e.g., pearinfected with fire blight bacteria), and then carried tohealthy plants. Humans also disseminate pathogens bytransporting contaminated soil on their feet or equipment,using contaminated containers, and using infectedtransplants, seed, nursery stock, and budwood asmentioned previously. Finally, humans disseminatepathogens by importing new varieties into an area thatmay carry pathogens that have gone

undetected, bytraveling throughout the world, and by importing foodor other items that may carry harmful plant pathogens.

Examples of the role of humans as a vector of pathogenscan be seen in the introduction into the United States ofthe fungi causing Dutch elm disease and white pineblister rust and of the citrus canker bacterium, in theintroduction in Europe of the powdery and downymildews of grape, and, more recently, in the rapidspread of sorghum ergot almost throughout the world(Fig. 2-20).

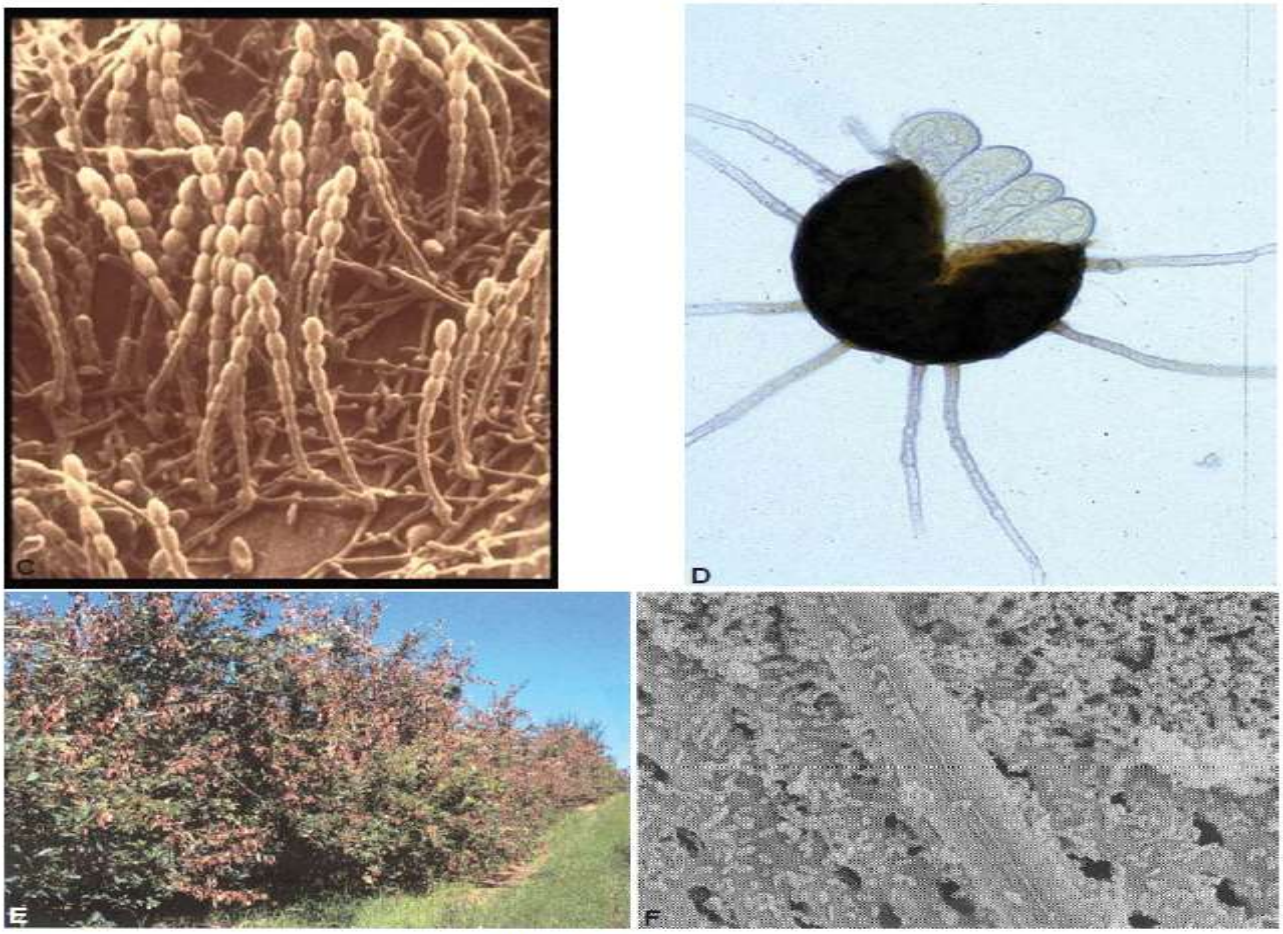



FIGURE 2-13 (*Continued*)

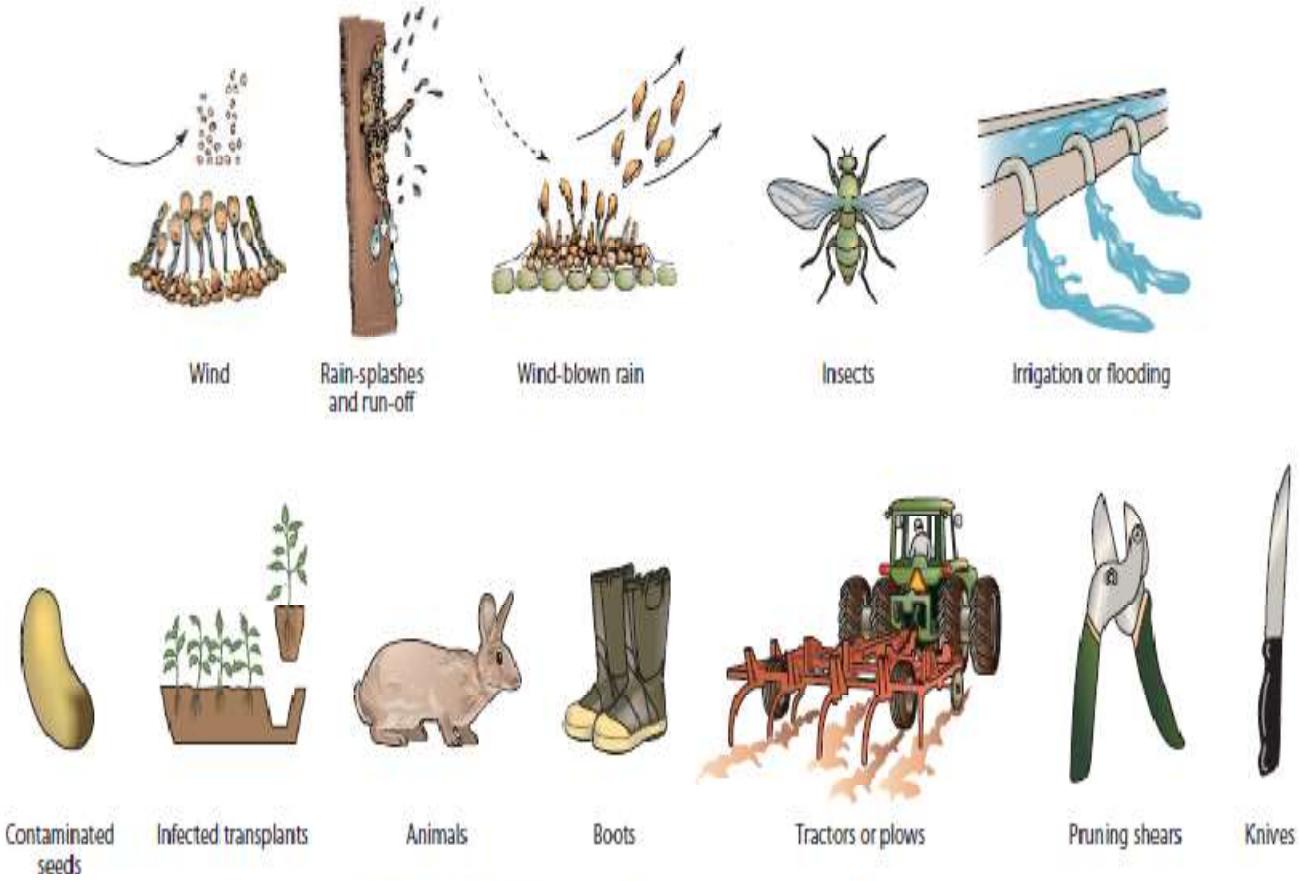



FIGURE 2-15 Means of dissemination of fungi and bacteria.

## 5. Host-pathogen interaction: penetration and disease development, role of cell-wall degrading enzymes and toxins, recognition mechanism and signal transduction during plant-pathogeninteraction

The host-pathogen interaction is defined as how microbes or viruses sustain themselves within host organisms on a molecular, cellular, organism or population level. This term is most commonly used to refer to disease-causing microorganisms although they may not cause illness in all hosts. Because of this, the definition has been expanded to how known pathogens survive within their host, whether they cause disease or not.

On the molecular and cellular level, microbes can infect the host and divide rapidly, causing disease by being there and causing a homeostatic imbalance in the body, or by secreting toxins which cause symptoms to appear. Viruses can also infect the host with virulent DNA, which can affect normal cell processes (transcription, translation, etc.), protein folding, or evading the immune response.

**Penetration and disease development:**

The following points highlight the three main stages of host pathogen relationship. The stages are: **1.** Pre-Penetration Stage **2.** Penetration Stage **3.** Post-Penetration Stage.

**1. Pre-PenetrationStage:**

During pre-penetration stage the pathogen (inoculum) on arrival on the host surface interacts sharply with the surrounding environment and host itself. The environment which is an aggregate of all external conditions including temperature, moisture (relative humidity), light and the competing microorganisms; affects the life and form of the pathogen of the inoculum.

For example, the development and abstraction of conidia are favored by high air temperature and humidity in downy mildew.

Whereas in powdery mildew both the number of spores and their germination are greater in brightsunlight.Incerealrustsuredospore'sgerminateatlowtemperature,buttheinfection

process is delayed at this temperature. Again soil pH plays a very vital role in the growth of bacterial plant pathogen in the rhizosphere (area of soil immediately surrounding the roots).

Whether a pathogen will survive and grow on the host surface also depends on its behavior with the exudates of, the host surface and the microbial population present on it. The exudates of the host surface may encourage or inhibit the growth of the pathogen. The root exudates mainly sugars and amino acids are nutrients for the growth of fungi and bacteria.

But root exudates like hydrocyanic acid, various organic acids and antibiotics are antifungal and antibacterial. For example, spores of *Rhizopus arrhizus* germinate only in presence of proline (amino acid) present in the rhizosphere region; whereas exudates of root of onion varieties inhibit spore germination of *Colletotrichumcircinaus*.

Leaves also exude substances which may go in favor or against the growth of the pathogen. The glands of leaf hairs of gram contain malic acid which is antifungal and arrests the growth of *Uromyces ciceris arietini*.

Protocatechuic acid, an exudate of onion skin is also antifungal. The pathogen has to neutralize these exudates or has to be resistant to them for survival. Besides these, the rhizosphere region contains microbial population which is antagonistic to the growth of the pathogen. As such, the pathogen has to overcome the above barriers during pre-penetration stage before it can survive for host penetration.

**2. Penetration Stage:**

The success of host penetration leading to disease development is a very compli•cated process which is a combined effect of various factors like:

**(i)** The nature and behavior of the pathogen including its multiplicationcapacity,

**(ii)** Favorable physical conditions,and

**(iii)** Hostsusceptibility.

Of all these factors, the factor nature and behavior of the pathogen is the most important one which controls the overall disease development. The nature and behavior of pathogen encompass the inoculum potential of the pathogen. The inoculum potential is again a measure of the biological energy available for the colonization of a host.

It is a function of:

**(i)** Inoculums density which refers to the number of viable propagules per unit area of leaf or stem or per unit volume ofsoil;

**(ii)** The nutrients available to the infectious units that allow them to germinate orgrow;

**(iii)** The environment (temperature range of 15 to 25°C, moisture content 70 per cent, and relative humidity 90 to 95 percent);

**(iv)** The virulence (aggressiveness) or genetic capacity of the pathogen to cause disease;and

**(v)** The susceptibility of thehost.

Besides these, the physiological state of the host may have an effect on the ability of the pathogen to attack it or on the extent to which a pathogen may harm it. The concept that encompasses this phenomenon is termedpredisposition.

**Factors involved in predisposition are:**

**(i)** Age of thehost;

**(ii)** Environmental conditions to which the pathogen has been exposed, i.e., light, humidity, soil environment, andtemperature;

**(iii)** Infection by other pathogens;and

**(iv)** Presence of chemicals, i.e., pesticides,herbicides.

Generally adverse conditions predispose a plant to greater susceptibility to attack by a pathogen. Temperature predisposition phenomenon in nature is extremely variable depending on the natureof host and pathogen. In most bacterial and fungal diseases, free moisture is necessary for pathogen development.

Most known cases of predisposition usually result in increased susceptibility of plants that were genetically resistant. A few cases are known in which a predisposing treatment has greatly increased resistance of plants that were genetically susceptible.

Certain fungal pathogens exhibit specificity in the part of host tissue infection. For example, some of them may remain restricted in the cortical tissue only through•out the entire period of attack and cause damage to it. Again others remain restricted in the vascular tissue only. Whereas, still others do not attack plant unless its heart wood is developed and the pathogen remains confined there causing damage.

Host penetration takes place:

**(i)** Through naturalopenings,

**(ii)** Throughwounds,

**(iii)** By direct penetration of surface cells causing tissue disintegration,and

**(iv)** Through specific parts ororgans.

Both bacteria and viruses enter the host tissue mainly through wounds. Whereas, the fungal pathogens gain entrance in the host through natural openings, wounds and by direct penetration through cuticle and outer wall of the surface cells, or root hairs, or through specific parts or organs of the host.

**(i) Entry through naturalopenings:**

Both bacteria and fungi gain entrance into the host through natural openings such as: stomata, lenticels, hydathodes, nectaries, leaf scars, stigma, etc. This is a process in which the pathogens have an easy access to the host, except in cases where sub-stomatal hairs may cause resistance against the host entry.

**(ii) Entry throughwounds:**

Wounds caused due to natural calamities (storm, fire, etc.); during field operations; by insects,by accidental breaking of parts or otherwise; offer easy passages of pathogens in the host. But so far as viruses are concerned, the host entry is only throughwounds.

**(iii) Entry by direct penetration of surface cells:**

The entry of pathogen by direct penetration of the outer wall of the host surface cells is rather a difficult process for which the pathogen usually requires high moisture or free water supply. It is even more difficult in leaves with waxy covering on their surface which allows water to rim off freely.

Fungal pathogens penetrate into host either by boring through the outer wall of the surface cell or penetration is effected by pressure and sometimes due to chemical softening or solubilizing of the barrier caused by the solvent action of enzymes secreted by the infectingorgan.

After the hypha made contact with a suitable host surface, some growth in close contact with it takes place. This is followed by the development of an aspersorium or increase in diameter of hypha serving as adherent area from which develops penetration tube.

The penetration tube penetrates through the cuticle at a point softened by enzymatic action and followed by mechanical pressure. In some cases germ lube produced by spore germination passes down between the racial wall of the adjoining cells without actually entering the cells.

This papilla is penetrated by the penetration hypha which enters the host cell and ultimately develops into a haustorium.

Bacteria are mostly weak parasites which cannot employ force to effect pene•tration. Their penetration is effected by chemical action. The plant parasitic nematodes pierce the host surface

with spears or stylet.

The entire process of Direct Penetration, however, depends on:

**(a)** The nature of cell walllayers,

**(b)** The potentialities of the enzyme system of the pathogen,and

**(c)** The potential force the pathogen canexert.

**Role of cell wall degrading enzyme and toxins:**

The main components of the host 'cell wall are pectin, cellulose, hemicellulose, lignin and small quantity of protein. The three main softening or solubilizing enzymes found in fungi are: pectolytic, cellulolytic, and lignolytic. The outer layer of host plant cell may have a layer of wax which is followed by cuticle impregnated with wax.

The cutin gradually decreases with the depth of the epidermis and is replaced by pectin which occurs as a homogeneous layer in some host plant. Subsequently in the secondary wall of the cell pectin is replaced by cellulose. Cellulose layers contain appreciable amounts of protein.

No pathogen secretes enzymes that would degrade wax. Only mechanical entry can enable a pathogen to breach this layer. Cutin layer is penetrated either by pres•sure (mechanically exerting force) or by the action of degrading enzymes: cutinase, cutin esterase, and carboxyl cutin esterase. Cutinase breaks cutin into fatty acid and hydroxyfatty acid.

Pectic substances which form basic material of middle lamella, primary and secondary walls are degraded by pectinolytic enzymes.

The pectinolytic enzymes act against pectin and pectic substances in many steps:

(a) The enzymes pectin methyl esterases (**PME**) hydrolyse the pectic substances into methanol and pectic acid;

(b) The enzymes pectin glycosidases, polygalacturonases (**PG**) and polymethyl galacturo- nases (**PMG**) degrade the pectic acid and methylated chains of pectin. Besides these, wall modifying enzyme (**WME**) modifies the pectic material for subsequentdegradation.

The degradation of pectic substances provides nutrients for many fungal pathogens and due to weakening of the cell wall facilitates inter- and intra-cellular invasion by hyphae. Pectic enzymes

are produced both constitutively and adaptively. High C/N ratio of the substrate favors increased mycelial growth and low enzyme synthesis whereas lower ratios give poorer growth but increased enzymesynthesis.

Cellulose forms the structural framework of cell walls. Cellulolytic enzymes act upon the cellulose and break it to simple compounds and make the way for the easy penetration of the pathogen into the host cell.

According to unienzyme theory, complete degradation of cllulose into glucose units is by a single enzyme: Cellulose Cellulose Cellobiose Cellobiase Glucose. Whereas, the multienzyme theory explains cellulose degradation in a series of steps through two groups ofenzymes.

One group of enzymes loosens the cellulose fibrils of the crystalline area by hydrolytic mechanism. The other group of enzymes penetrates the cellulose lattice and causes hydrolytic cleavage of hydrogen bondage.

The degradation of hemicelluloses by the enzymes hemicellulases produces simpler sugars. In vascular wilt diseases the molecules released by cellulose degradation can cause plugging of the vessels.

The most complex chemical compound in the plant cell wall is lignin which occurs chiefly in the matrix surrounding the cellulose fibrils. It is one of the most structurally complex biopolymers whose degradation is caused by polyphenol oxidases produced by wood-rotting fungi. But fungi cannot utilize the degradation products.

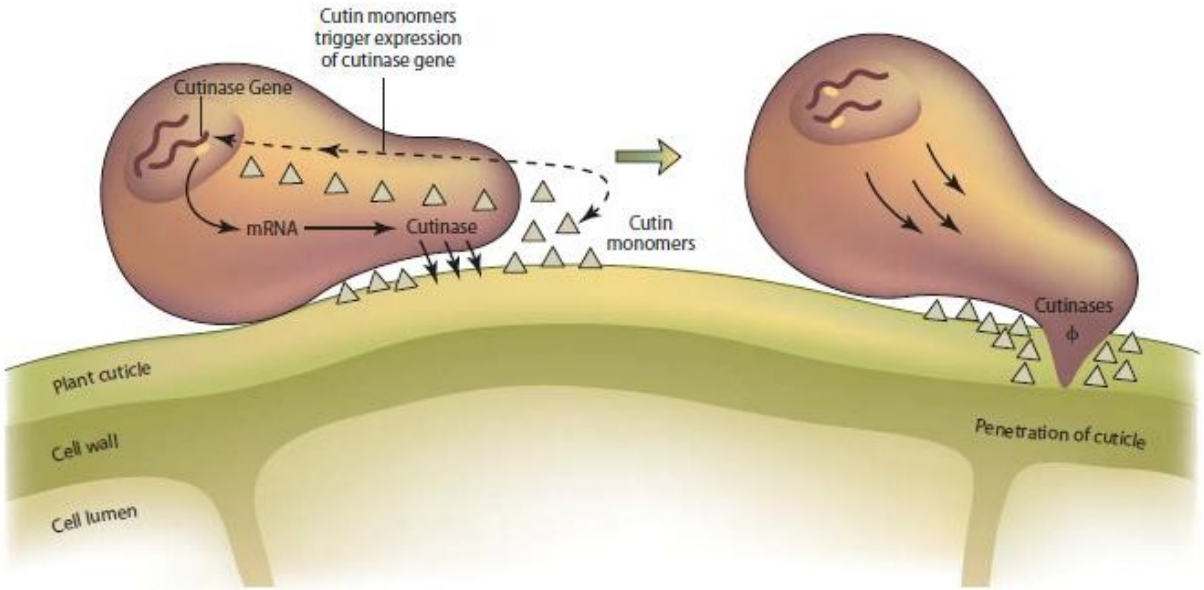



**FIGURE 5-5** Diagrammatic representation of cuticle penetration by a germinating fungus spore. Constitutive cutinase releases a few cutin monomers from the plant cuticle. These trigger expression of the cutinase genes of the fungus, leading to the production of more cutinase(s), which macerates the cuticle and allows penetration by the fungus.

**Toxins That Affect a Wide Range of Host Plants**

Living plant cells are complex systems in which many interdependent biochemical reactions are taking placeconcurrently or in a well-defined succession. These reactions result in the intricate and well-organizedprocesses essential for life. Disturbance of any of thesemetabolic reactions causes disruption of the physiologicalprocesses that sustain the plant and leads to thedevelopment of disease. Among the factors inducingsuch disturbances are substances that are producedby plant pathogenic microorganisms and are calledtoxins. Toxins act directly on living host protoplasts,seriously damaging or killing the cells of the plant.

Some toxins act as general protoplasmic poisons andaffect many species of plants representing differentfamilies. Others are toxic to only a few plant species orvarieties and are completely harmless to others. Manytoxins exist in multiple forms that have differentpotency.Fungi and bacteria may produce toxins in infectedplants as well as in culture medium. Toxins, however,are extremely poisonous substances and are effective invery low concentrations. Some are unstable or reactquickly and are bound tightly to specific sites within theplant cell.

Toxins injure host cells either by affecting thepermeability of the cell membrane (Fig. 5-2) or by inactivatingor inhibiting enzymes and subsequently interruptingthe corresponding enzymatic reactions. Certaintoxins act as antimetabolites and induce a deficiency foran essential growth factor.

**Toxins That Affect a Wide Range of Host Plants**

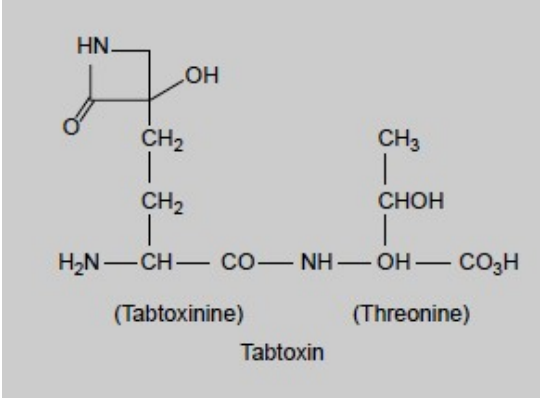
Several toxic substances produced by phytopathogenic microorganisms have been shown to produce all or part of the disease syndrome not only on the host plant, but also on other species of plants that are not normally attacked by the pathogen in nature. Such toxins, called nonhost-specific or nonhost-selective toxins. These toxins increase the severity of disease caused by a pathogen, i.e., they affect the virulence of the pathogen, but are not essential for the pathogen to cause disease, i.e., they do not determine the pathogenicity of the pathogen. Several of these toxins, e.g., tabtoxin and phaseolotoxin, inhibit normal host enzymes, thereby leading to increases in toxic substrates or to depletion of needed compounds. Several toxins affect the cellular transport system, especially H+/K+ exchange at the cell membrane. Some, e.g., tagetitoxin, act as inhibitors of transcription in cell organelles, such as the chloroplasts. Others, e.g., cercosporin, act as photosensitizing agents, causing the peroxidation of membrane lipids.

Several toxic substances produced by phytopathogenic microorganisms have been shown to produce all or part of the disease syndrome not only on the host plant, but also on other species of plants that are not normally attacked by the pathogen in nature. Such toxins, called **nonhost-**

**specific or nonhost-selectivetoxins**.

**Tabtoxin**

Tabtoxin is produced by the bacterium *Pseudomonas syringae*; pv. tabaci, which causes the wildfire disease of tobacco; by strains of pv. tabaci occurring on other hosts such as bean and soybean; and by other pathovars (subspecies) of *P. syringae*, such as those occurring on oats, maize, and coffee. Toxin-producing strains cause necrotic spots on leaves, with each spot surrounded by a yellow halo.




**Phaseolotoxin**

Phaseolotoxin is produced by the bacterium *Pseudomonas syringae* pv. phaseolicola, the cause of halo blight of bean and some other legumes. Phaseolotoxin is a modified ornithine–alanine–arginine tripeptide carrying a phosphosulfinylgroup.

**Tentoxin**

Tentoxin is produced by the fungus *Alternaria alternate* (previously called *A. tenuis*), which causes spots and chlorosis in plants of many species. Tentoxin is a cyclic tetrapeptide that binds to and inactivates a protein (chloroplast-coupling factor) involved in energy transfer into chloroplasts. The toxin also inhibits the light-dependent phosphorylation of ADP to ATP.

**Cercosporin**

Cercosporin is produced by the fungus *Cercospora* and by several other fungi. It causes damaging leaf spot and blight diseases of many crop plants, such as Cercospora leaf spot of zinnia and gray leaf spot of corn.

**Host-Specific or Host-Selective Toxins**

A host-specific or host-selective toxin is a substance produced by a pathogenic microorganism that, at physiological concentrations, is toxic only to the hosts of that pathogen and shows little or no toxicity against nonsusceptibleplants.

**Victorin, or HV Toxin**

Victorin, or Hv-toxin, is produced by the fungus *Cochliobolus* (*Helminthosporium*) *victoriae*. This fungus appeared in 1945 after the introduction and widespread use of the oat variety Victoria and its derivatives, all of which contained the gene Vb for resistance to crown rust disease. *C. victoriae* infects the basal portions of susceptible oat plants and produces a toxin that is carried to the leaves, causes a leaf blight, and destroys the entireplant.

**T Toxin [*Cochliobolus* (*Helminthosporium*) heterostrophus Race T Toxin]**

T toxin is produced by race T of *C. heterostrophus* (*Bipolaris maydis*), the cause of southern corn leaf blight. Race T, indistinguishable from all other *C. heterostrophus* races except for its ability to produce the T toxin, appeared in the United States in 1968. By 1970, it had spread throughout the corn belt, attacking only corn that had the Texas male-sterile (Tms) cytoplasm. T toxin is a mixture of linear, long (35 to 45 carbon)polyketols.

**HC Toxin**

Race 1 of *Cochliobolus* (*Helminthosporium*) *carbonum* (*Bipolaris zeicola*) causes northern leaf spot and ear rot disease in maize. It also produces the hostspecific HC toxin, which is toxic only on specific maize lines.

***Other Nonhost-Specific Toxins***

Numerous other nonhost-specific toxic substances have been isolated from cultures of pathogenic fungi andbacteria and have been implicated as contributing factors in the development of the disease caused by the pathogen. Among such toxins produced by fungi are fumaric acid, produced by *Rhizopus* spp. in almond hullrot disease; oxalic acid, produced by *Sclerotium* and *Sclerotinia* spp. in various plants they infect and by *Cryphonectria parasitica*, the cause of chestnut blight; alternaric acid, alternariol, and zinniol produced by*Alternaria* spp. in leaf spot diseases of various plants; ceratoulmin, produced by *Ophiostoma ulmi* in Dutchelm disease; fusicoccin, produced by *Fusicoccum amygdale* in the twig blight disease of almond and peach trees; ophiobolins, produced by several *Cochliobolus* spp. in diseases of grain crops; pyricularin, produced by *Pyricularia grisea* in rice blast disease; fusaric acid and lycomarasmin, produced by *Fusarium oxysporum* in tomato wilt; and many others.

**Host-Specific or Host-Selective Toxins**

A **host-specific** or **host-selective** toxin is a substance produced by a pathogenic microorganism that, at physiological concentrations, is toxic only to the hosts of that pathogen and shows little or no toxicity against non susceptible plants. Most host-specific toxins must be present for the producing

microorganism to be able to cause disease. So far, host-specific toxins have been shown to be produced only by certain fungi (*Cochliobolus*, *Alternaria*, *Periconia*, *Phyllosticta*, *Corynespora*, and *Hypoxylon*), although certain bacterial polysaccharides from *Pseudomonas* and *Xanthomonas* have been reported to be host specific.

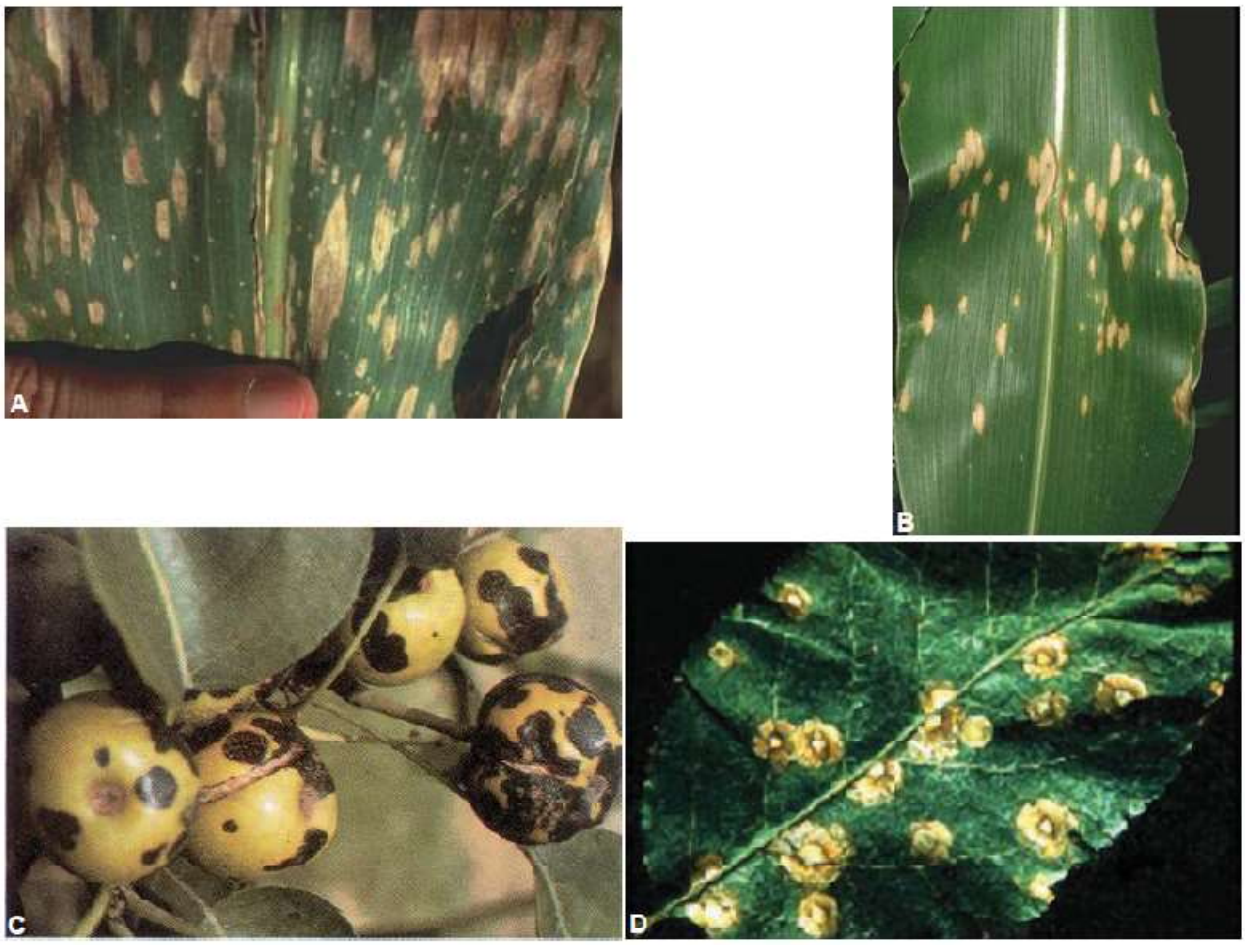



FIGURE 5-15 Symptoms caused by host-selective toxins. (A) Southern corn leaf blight symptoms caused by two race T of the fungus *Cochliobolus (Helminthosporium) heterostrophus* and its toxin, T toxin, on a corn plant containing Texas male-sterile cytoplasm. (B) Northern corn leaf spot symptoms caused by the fungus *Cochliobolus carbonum* and its toxin, HC toxin, on corn. (C) Fruit spots on Japanese pear caused by one of the strains of the fungus *Alternaria alternata* and its toxin, AK toxin. (D) Leaf spots caused by the AM toxin produced by another strain of the fungus *A. alternata* and its toxin, AM toxin, on apple leaves. [Photographs courtesy of (A) C. Martinson and (B) G. Munkvold, Iowa State University, (C) T. Sakuma, and (D) J. W. Travis, Pennsylvania State University.]

**Entry through specific parts ororgans:**

In such cases the pathogens exhibit specificity of host parts or organs (stem, leaf, root hairs, floral parts, coleoptile, buds, etc.) during their entry in thehost.

Some examples are:

*Synchytrium endobioticun* infects and remains confined in the epidermal cells of potato tuber, *Erysiphe graminis* infects the epidermal cells of leaves of cereals and grasses, *Ustilago nuda* causes infection only in the flowers of wheat, *Claviceps purpurea* infects the ovaries of rye flower, certain wood rotting fungi are – *Merulius lacrimans* and *Fomes annosus*; soil inhabiting pathogens inter through root hairs – *Plasmodiophora brassicae*.

**Post-Penetration Stage:**

Usually with the success of the penetration process, post-penetration is successful. But the entry of the pathogen in the host tissue may not always ensure immediate infection leading to disease development. The process may be delayed or there may be failure for various reasons.

The delay may be in cases where the pathogen has incubation period and infection is established only after the expiry of the incubation period. The success of post-penetration process depends largely upon competition of pathogen for nutrition, and production of enzymes and toxic substances and their effects on host metabolic activities.

Again due to toxic effect of host cytoplasm, the pathogen may fail to establish biological relationship with the host. Host-pathogen interaction may also result hypersensitivity of the host tissue, whereby rapid death of the affected cells prevents the further spreading of the pathogen due to shortage of nutrition.

But in most of the plant diseases, host infection is followed by invasion, a condition when a pathogen grows rapidly in the host tissue. For example, bacteria invade host tissues intracellular and destroy them. Whereas, fugal mycelia invade inter- or intracellular but may or may not cause destruction immediately after invasion. Viruses always invade host tissues intracellular.

They multiply in the living host cells by directing them to manufacture viral nucleic acid and viral protein, their movement from cell to cell is through plasmodesmata. Again fungal hyphae and spores, and bacterial cells may move through vascular tissues once they gain entrance inthem. Successful host invasion of the pathogen is invariably associated with disease syndromes of various types of varyingdegrees.

After penetrating the host cell walls, the pathogen comes in contact with the host cytoplasm from

which it gets its required nutrition. In response to the activities of the pathogen, the host metabolic processes (osmoregulation, respiration, photosynthesis, etc.) get upset.

**Signal transduction:**

**Perception of pathogen presence**

Plant defense signaling is activated by the pathogen-detecting receptors that are described in an above section. The activated receptors frequently elicit reactive oxygen and nitric oxide production, calcium, potassium and proton ion fluxes, altered levels of salicylic acid and other hormones and activation of **MAP** kinases and other specific protein kinases. These events in turn typically lead to the modification of proteins that control gene transcription, and the activation of defense-associated gene expression.

**Transcription factors and the hormone response**

Numerous genes and/or proteins as well as other molecules have been identified that mediate plant defense signal transduction. Cytoskeleton and vesicle trafficking dynamics help to orient plant defense responses toward the point of pathogen attack.

**Mechanisms of transcription factors and hormones**

Plant immune system activity is regulated in part by signaling hormones such as: Salicylic acid , Jasmonic acid, Ethylene. There can be substantial cross-talk among these pathways

**Regulation by degradation:**

As with many signal transduction pathways, plant gene expression during immune responses can be regulated by degradation. This often occurs when hormone binding to hormone receptors stimulates ubiquitin-associated degradation of repressor proteins that block expression of certain genes. The net result is hormone-activated gene expression. Examples:

**Auxin:** binds to receptors that then recruit and degrade repressors of transcriptional activators that stimulate auxin-specific gene expression.

**Jasmonic acid:** similar to auxin, except with jasmonate receptors impacting jasmonate-response signaling mediators such as **JAZ** proteins.

**Gibberellic acid:** Gibberellin causes receptor conformational changes and binding and degradation of Della proteins.

**Ethylene:** Inhibitory phosphorylation of the **EIN2** ethylene response activator is blocked by ethylene binding. When this phosphorylation is reduced, **EIN2** protein is cleaved and a portion of the protein moves to the nucleus to activate ethylene-response gene expression.

**Ubiquitin and E3 signaling**

Ubiquitination plays a central role in cell signaling that regulates processes including protein degradation and immunological response. Although one of the main functions of ubiquitin is to target proteins for destruction, it is also useful in signaling pathways, hormone release, apoptosis and translocation of materials throughout the cell. Ubiquitination is a component of several immune responses. Without ubiquitin's proper functioning, the invasion of pathogens and other harmful molecules would increase dramatically due to weakened immune defenses.

**E3 signaling**

The E3 Ubiquitin ligase enzyme is a main component that provides specificity in protein degradation pathways, including immune signaling pathways. The E3 enzyme components can be grouped by which domains they contain and include several types. These include the Ring and U-box single subunit, HECT, and CRLs. Plant signaling pathways including immune responses are controlled by several feedback pathways, which often include negative feedback; andthey are be regulated by De-ubiquitination enzymes, degradation of transcription factors and the degradation of negative regulators of transcription.

6. **Defense mechanisms of plants against infection: Pre-existing structural and biochemical defense, induced structural and biochemical defense, hypersensitive reaction, phytoalexins, PR proteins, systemic acquired resistance, induced systemic resistance; Gene for gene hypothesis; concept of horizontal and vertical resistance**

In general, plants defend themselves against pathogens by a combination of weapons from two arsenals:

**(1)** structural characteristics that act as physical barriers and inhibit the pathogen from gaining entranceand spreading through the plant and **(2)** biochemical reactions that take place in the cells and tissues of theplant and produce substances that are either toxic to the pathogen or create conditions that inhibit growthof the pathogen in the plant. The combinations of structural characteristics and biochemical reactions employed in the defence of plants are different in different host–pathogen systems. In addition, even withinthe same host and pathogen, the combinations vary with the age of the plant, the kind of plant organ and tissue attacked, the nutritional condition of the plant, and the weather conditions.

**I. Structural Defense:**

In plants some structures are already present to defend the attack while in others, the structures to defend the host develops after the infection. In this way, structural defense can be characterised as **(A)** Preexisting defense structures and **(B)** Defense structures developed after the attack of the pathogen.

**(A) Preexisting Defense Structures:**

**(i) CuticularWax:**

Wax-mixtures of long chain aliphatic compounds get deposited on the cuticular surface of some plants. Deposition of wax on the cuticular surface is thought to play a defensive role by forming a hydrophobic surface where water isrepelled.

As a result, the pathogen does not get sufficient water to germinate or multiply. In addition, a negative charge usually develops on the leaf surface due to the presence of fatty acids – the main component of cuticle. The negative charge prevents/reduces the chance of infection by many pathogens.

**(ii) CuticleThickness:**

The thickness of cuticle is most important for those which try to enter the host through the leaf surface. The cuticle thickness obstructs the path of pathogen. In addition, a thick cuticle checks the exit of the pathogen from inside the host, thus reducing the secondary infection.

**(iii) Structure of Epidermal CellWalls:**

Tough and thick outer walls of epidermal cells may directly prevent the entry of the pathogen completely or make the entry difficult. The presence or absence of lignin and silicic acid in the cell walls may show variation in resistance to penetration of the pathogen.Most outer walls of epidermal cells of rice plants are lignified and are seldom penetrated by blast disease of rice pathogen. In resistant varieties of potato tubers (resistant to *Pythium debaryanum*) the epidermal cells contain higher fibre content than the susceptible ones.

**(iv) Structure of Naturalopenings:**

Structure of natural openings like stomata lenticels etc. also decide the fate of the entry of the pathogen. In Szincum variety of citrus, the stomata are small and possess very narrow openings surrounded by broad lipped raised structures which prevent entry of water drops containing citrus cankerbacterium.

In the same way, the size and internal structures of lenticels may play a defensive role against the pathogens. Varieties having small lenticels in the apple fruits prevent the entry of the pathogen while those having large openings easily allow the pathogen to enter.

Nectaries provide openings in the epidermis and may play a defensive role due to high osmotic concentration of the nectar. In resistant varieties of apple, presence of abundant hairs in the nectaries acts as a defense mechanism while susceptible varieties are devoid of abundant hairs.

**Internal Defense Structures:**

There are many preexisting internal defense structures inside the plant that prevent the entry of pathogen beyond these structures. In some plants, cell walls of certain tissues become thick and tough due to environmental conditions and this makes the advance of the pathogen quite difficult. In case of stems of cereal crops, vascular bundles or extended areas of sclerenchyma cells checks the progress of rust pathogen. Leaf veins effectively obstruct the spread of pathogen like the angular leaf spot pathogen.

**(B) Defense Structures Developed after the Attack of thePathogen:**

After the pathogen has successfully managed to overcome the preexisting defense mechanisms of

the host, it invades the cells and tissues of thehost.

In order to check the further invasion by the pathogen, the host plants develop some structures/mechanisms which may be defense reactions in the cytoplasm, cell wall defense structures, defense structures developed by the tissues and ultimately the death of the invaded cell i.e. necrosis. These will be briefly discussedhere.

**(i) Defense Reactions in theCytoplasm:**

The cytoplasm of the invaded cell surrounds the hyphae of the pathogen and the nucleus of the host cell gets stretched to break into two. In some host cells, the cytoplasm and the nucleus of the infected cells enlarge.

The cytoplasm becomes granular and dense and develops granular particles. These result in the disintegration of the pathogen mycelium and thus the invasion stops. Such cytoplasmic defence mechanisms can be seen in weak pathogens like Annillaria and some mycorrhizal fungi.

**(ii) Cell Wall DefenseStructures:**

Cell wall defense structures are of limited help to the host. These include morphological changes in the cell wall of the host.

Three types of cell wall defense structures are generally observed:

**(i)** Cell walls thicken in response to the pathogen by producing a cellulose material, thus preventing the entry of thepathogen

**(ii)** The outer layer of cell walls of the parenchyma cells in contact with invading bacterial cells produce an amorphous fibrillar material that traps the bacteria thus preventing them to multiply a

**(iii)** Callose papillae get deposited on the inner layers of the cell walls due to invasion by fungal pathogens.In raw cases, the hyphal tips of the infecting fungal pathogen penetrating the cell wall and thereafter growing into the cell lumen get enveloped by callose material that, later become infused with phenolics forming a sheath around the hyphae.

**(iii) Defense Structures Developed by the Tissues:**

The following four developments take place in the tissues after penetration:

**(a) GumDeposition:**

Plants produce a variety of gummy substances around lesions or spots as a result of infection. These gummy substances inhibit the progress of the pathogen. The gummy substances are commonly produced in stone fruits.

**(b) Abcission Layers:**

Abscission layers are usually formed to separate the ripe fruits and old leaves from the plant. But in some stone fruit trees, these layers develop in their young leaves in response to infection by several fungi, bacteria or viruses. An abscission layer is a gap formed between two circular layers of cells surrounding the point ofinfection.

This gap is created by the dissolution of one or two layers of the middle lamella, one or two layers of cells surrounding the infected loci resulting in the infected locus becoming unsupported, shrivels, dies and falls down along with the pathogen. Abscission layer formation protects the healthy leaf tissue from the attack of thepathogen.

Different defence structures developed after infection

**(C) Tyloses:**

Tyloses are out growths of protoplasts of adjacent live parenchyma cells protruding into xylem vessels through pits under stress or in response to attack by the vascular pathogens. Their development blocks the Xylem vessels, obstructing the flow of water and resulting in the development of wilt symptoms.

However, tyloses are formed in some resistant plants ahead of infection and the prevent the plant from being attacked.

**(D) Formation ofLayers:**

Some pathogens like certain bacteria, some fungi and even some viruses and nematodes stimulate the host to form multilayered cork cells in response to infection, these develop as a result of stimulation of host cells by substances secreted by thus,pathogen.

These layers inhibit the further invasion by the pathogen and also block the flow of toxic substances secreted by the pathogen. Cork layers also stop the flow of nutrients of the host thus also depriving the pathogen of the nutrients.

Examples of cork layer formation as a result of infection are: soft not of potato caused by *Rhizopus* sp., potato tuber disease caused by *Rhizoctonia* sp., Scab of potato caused by *Streptomyces scabies* and necrotic lesions on tobacco caused by tobacco mosaic virus.

**IV. Necrosis or Hypersensitive Type of Defense:**

Necrosis or hypersensitive type of defense is another defense mechanism adopted by some pathogens like *Synchytrium endobioticum* causing wart disease of potato, *Phytophthora infestans* causing late blight disease of potato and *Pyricularia oryzae* causing blast of rice etc.

In such diseases, the host nucleus moves toward the pathogen when the latter comes in contact with the protoplasm of the host. The nucleus soon disintegrates into brown granules which first accumulate around the pathogen, later dispersing throughout the host cytoplasm.

Soon the cell membrane swells and finally the cell bursts and dies. These cause the pathogen

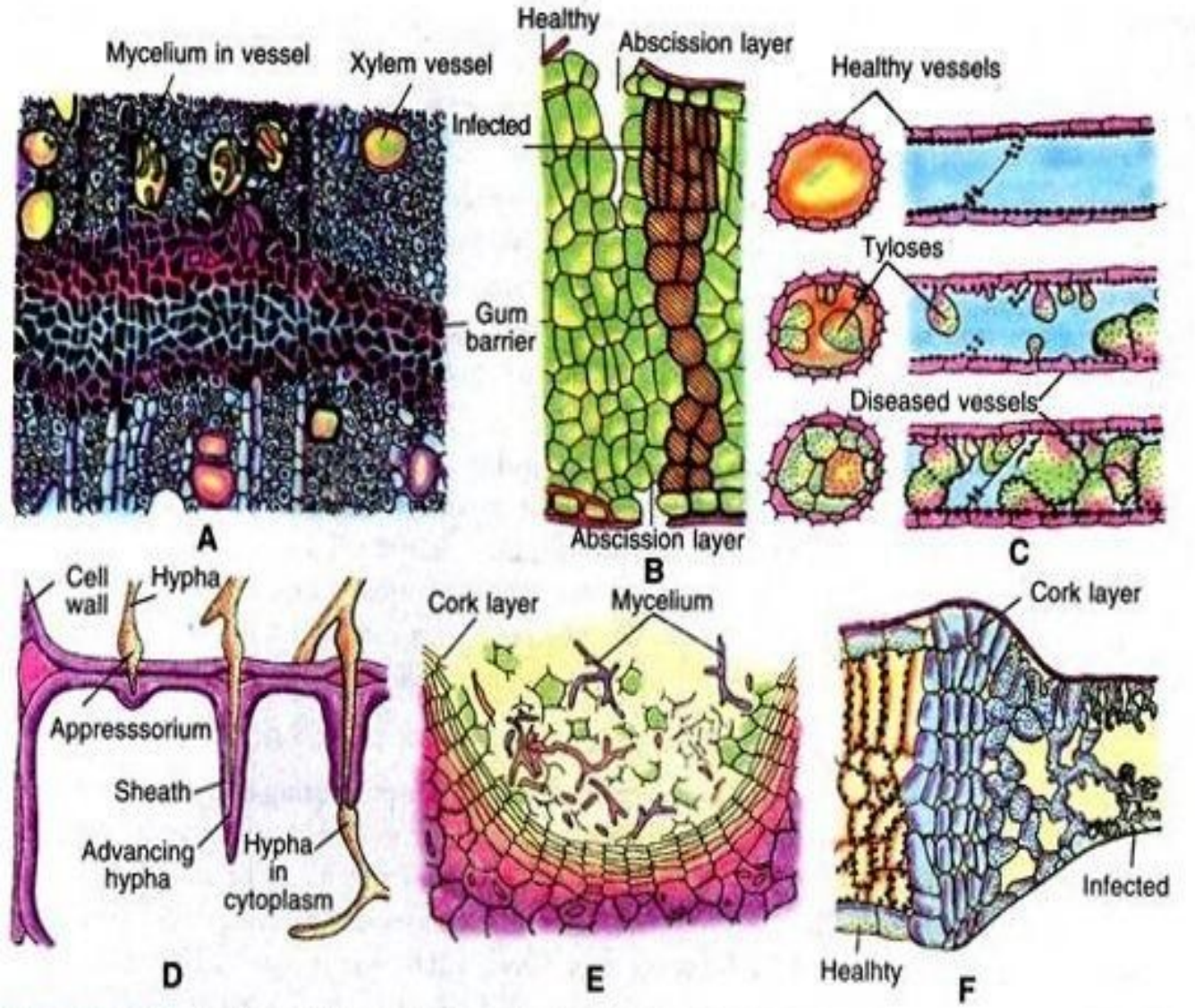



Fig. 21.1. (A-F). Different defence structures developed after infection : (A) Gum deposition in cells ; (B) Abscission layer ; (C) Tyloses ; (D) Formation of sheath around developing hyphae (E, F)./ Development of cork layer in tuber (E) and leaf (F).

**II. Biochemical Defense:**

Although structural defense mechanisms do prevent the attack of the pathogen, the defense mechanism also includes the chemical substances produced in the plant cells before or after the infection.

It has now been established that biochemical defense mechanisms play more important role than the structural defense mechanisms. This has been supplemented by the fact that many pathogens entering non host plants naturally or artificially inoculated fail to cause infections in absence of any structural barriers.This does suggest that chemical defense mechanisms rather than structural mechanisms are responsible for resistance in plants against certain pathogens.

**(A) Preexisting BiochemicalDefense:**

**(i) Inhibitors Released in the Prepenetration Stage:**

Plant generally exudes organic substance through above ground parts (phyllosphere) and roots (rhizosphere). Some of the compounds released by some plants are known to have an inhibitory effect on certain pathogens during the prepenetration stage.

For example fungistatic chemicals released by tomato and sugar beet prevent the germination of *Botrytis* and *Cercospora*. Presence of phenolics like protocatechuic acid and catechol in scales of red onion variety inhibit the germination of conidia of *Colletotrichum circinans* on the surface of red onion.

Inhibitors present in high concentrations in the plant cells also play an important role in defense of plants. Presence of several phenolics, tannins and some fatty acid like compounds such as dienes in cells of young fruits, leaves or seeds afford them resistance to Botrytis.

The tubers of resistance vars of potato against potato scab disease contain higher concentrations of chlorogenic acid around the lenticels and tubers than the susceptible vars. Several other compounds like saponin tomatin in tomato and avinacin in oats have antifungal activity. Some enzymes like glucanases and chitinases present in cells of some plants may break down the cell wall components of pathogens.

**(ii)** Lack of nutrients essential for the pathogen is another preexisting biochemical defense mechanism. Plant varieties or species which do not produce any of the chemicals essential forthe growth of pathogen may act as resistantvariety.

For example, a substance present in seedling varieties susceptible to *Rhizoctonia* initiates hyphae cushion formation from which the fungus sends penetration hyphae inside the host plants. When this substance is not present, hyphal cushions are not formed and the infection does not occur.

**(iii) Absence of Common Antigen in Hostplant:**

It is now clear that the presence of a common protein (antigen) in both the pathogen and host determines diseases occurrence in the host. But if the antigen is present in the host and absent in the host or vice-versa, it makes the host resistant to the pathogen.For example, varieties of linseed which have an antigen common to their pathogen are susceptible to the disease rust of linseed caused by *Melampsoralini*.In contrast, the absence of antigen in linseed varieties but occurring in the pathogen are resistant to the pathogen. Another example is leaf spot disease of cotton caused by *Xanthomonas campestris* pv. malvacearum.


FIGURE 6-2 Onion smudge, caused by the fungus *Colletotrichum circinans*, develops on white onions but not on colored ones, which, in addition to the red or yellow pigment, also contain the phenolics protocatechuic acid and catechol, both of which are toxic to the fungus. (Photograph courtesy of G. W. Simone.)

**(B) Post-Infection-Biochemical Defense Mechanism:**

In order to sight infections caused by pathogens or injuries caused by any other means, the plant cells and tissues produce by synthesis many substances (chemicals) which inhibit the growth of causal organism.

These substances are generally produced around the site of infection or injury with the main aim at overcoming the problem.

Some such important chemicals are described below:

**(i) PhenolicCompounds:**

These are the most common compounds produced by plants in response to injury or infection. The synthesis of phenolic compounds takes place either through "**acetic acid pathway**" or "**Shikimic acid pathway**".

Some common phenolic compounds toxic to pathogens are chlorgenic acid, caffeic acid and ferulic acid. These phenolic compounds are produced at a much faster rate in resistant varieties than in susceptible varieties.

Probably that the combined effect of all phenolics present is responsible for inhibiting the growth of the infection.

**(ii) Phytoalexins:**

Phytoalexins are toxic antimicrobial substances synthesized 'de novo' in the plants in response to injury, infectious agents or their products and physiological stimuli. The term phytoalexin was first used by the two phytopathologists Muller and Borger (1940) for fungi static compounds produced by plants in response to mechanical or chemical injury or infection.

All phytoalexins are lipophilic compounds and were first detected after a study of late blight of potato caused by Phytophthora infestans. Phytoalexins are believed to be synthesized in living cells but surprisingly necrosis follows very quickly.

According to Bill (1981), peak concentration of phytoalexins almost always coincides with necrosis. Although the exact mechanism of production of phytoalexin has not been properlyunderstood, it is considered that a metabolite of the host plant interacts with specific receptor on the pathogen's membrane resulting in the secretion of "phytoalexin elicitor" which enters the host plant cells and stimulates the phytoalexinsynthesis.

Phytoalexins are considered to stop the growth of pathogens by altering the plasma membrane and inhibiting the oxidative phosphorylation.

Phytoalexins have been identified in a wide variety of species of plants such as Soyabean, Potato, sweet potato, barley, carrot, cotton etc. are being investigated. Some common phytoalexins are Ipomeamarone, Orchinol, Pistatin, Phaseolin, Medicarpin, Rishitin, Isocoumarin, 'Gossypol' Cicerin, Glyceolin, Capisidiol etc.

The following Table gives a list of phytoalexins, chemical nature the host and the pathogens in response to which these are produced:

| | Phytoalexin | Host | Pathogen | Chemical Nature of the phytoalexin |
|---|---|---|---|---|
| 1. | Ipomeamarone | *Ipomoea batata* | *Ceratocystis fimbriata* | Furanosesquiterepene ketone. |
| 2. | Orchinol | *Orchid malitaris* | *Rhizoctonia repens* | Phenanthrene |
| 3. | Pistatin | *Pisum sativum* | *Ascochyta pisi* | Kievitone |
| 4. | Phaseolin | *Phaseolus* sp. | *Monilia fruticola* | |
| 5. | Medicaripin | *Medicago lupulina* | *Colletotrichum phomoides* | Dimethyl-homoptero-carpin |
| 6. | Rishitin | *Solanum tuberosum* var. *rishiri* | *Phytophthora infestans* | Nonsequiter-pene |
| 7. | Isocoumarin | *Daucus carrota* | *Fusarium oxysporum f. lycopersici* *Rhizopus solonifer* | – |
| 8. | *Cicerin* | *Cicer arietinum* | *Ascochyta rabie* | |
| 9. | Gossypol | *Gossypium* sp. | *Verticillium alboatrum* | |
| 10. | Capisidiol | *Capsicum fruitescens* | *Peronospora trifolium* | |
| 11. | Wyerone acid | Broad bean | *Botrytis fabae* | |

**(iii)Substances Produced in Host to Resist Enzymes Produced byPathogen:**

Some hosts produce chemicals which neutralize the enzymes produced by pathogen, thus defending the host. Therefore these substances help plants to defend themselves from the attack of the pathogen.

In bean plants, infection with *Rhizoctonia solani* causes necrosis. In resistant bean varieties, the entry of pathogen causes the separation of methyl group from methylated pectic substances and forms polyvalent cations of pectic salts which contain calcium.

The calcium ions accumulate in infected as well as neighbouring healthy tissues and because of the calcium accumulation, the pathogen fails to disintegrate middle lamella by its polygalacturonase enzymes. These are known to dissolve the middle lamella of healthy tissue in susceptible varieties.

**(iv) Detoxification of Pathogen Toxins andEnzymes:**

In some cases, the plants produce chemicals which deactivate the toxins produced by the pathogens. For example, *Pyricularia oryzae* which causes blast disease of rice produces Picolinic acid and pyricularin as toxins.

Although resistant varieties convert these toxins into N-methyl picolininic acid pyrecularin into other compounds, the susceptible varieties do get affected by these toxins. Similarly in case of

cotton and tomato wilts, the toxin fusaric acid produced by the pathogen gets converted into non-toxic N-methyl-fusaric acid amide in resistant varieties.As in case of detoxification of toxins, the toxic enzymes produced by the pathogen is deactivated by phenolic compounds or their oxidation products. Some varieties of cider apple are resistant to brown not disease caused by S*clereotinia fructigena*.It may be because of the resistant varieties producing pheolic oxidation products which inactivate the pectinolytic enzymes produced by thepathogen.

**(v) BiochemicalAlterations:**

It has been observed that infection of the host by the pathogen brings about biochemical changes in the host which may prove toxic to the pathogenic microorganisms and cause resistance to the pathogen. Production of certain new enzymes and other compounds are synthesized and accumulated in higher concentration. This may also add to the resistance of the plant by being toxic to pathogenic microorganisms.

**INDUCED STRUCTURAL DEFENSES**

Despite the preformed superficial or internal defensestructures of host plants, most pathogens manage topenetrate their hosts through wounds and natural openingsand to produce various degrees of infection. Evenafter the pathogen has penetrated the preformed defensestructures, however, plants usually respond by formingone or more types of structures that are more or lesssuccessful in defending the plant from further pathogeninvasion. Some of the defense structures formed involvethe cytoplasm of the cells under attack, and the processis called **cytoplasmic defense reaction**; others involve thewalls of invaded cells and are called **cell wall defensestructures**; and still others involve tissues ahead of thepathogen (deeper into the plant) and are called **histologicaldefense structures**. Finally, the death of theinvaded cell may protect the plant from further invasion.

This is called the **necrotic** or **hypersensitive defense reaction**and is discussed here briefly

**Cytoplasmic Defense Reaction:**

In a few cases of slowly growing, weakly pathogenicfungi, such as weakly pathogenic *Armillaria* strains andthe mycorrhizal fungi, that induce chronic diseases ornearly symbiotic conditions, the plant cell cytoplasmsurrounds the clump of hyphae and the plant cellnucleus is stretched to the point where it breaks in two.

In some cells, the cytoplasmic reaction is overcome andthe protoplast disappears while fungal growth increases.In some of the invaded cells, however, the cytoplasm andnucleus enlarge. The cytoplasm

becomes granular anddense, and various particles or structures appear in it.Finally, the mycelium of the pathogen disintegrates andthe invasion stops.

**Cell Wall Defense Structures**

Cell wall defense structures involve morphologicalchanges in the cell wall or changes derived from the cellwall of the cell being invaded by the pathogen. Theeffectiveness of these structures as defensemechanismsseems to be rather limited, however. Three main typesof such structures have been observed in plant diseases.

(**1**) The outer layer of the cell wall of parenchyma cellscoming in contact with incompatible bacteriaswells andproduces an amorphous, fibrillar material that surroundsand traps the bacteria and prevents them frommultiplying. (**2**) Cell walls thicken in response to severalpathogens by producing what appears to be a cellulosicmaterial. phenolic substances that are cross-linked and furtherincrease its resistance to penetration. (**3**) Callose **papillae**are deposited on the inner side of cell walls inresponse to invasion by fungal pathogens.Insome cases, hyphal tips of fungi penetrating a cell walland growing into the cell lumen are enveloped by cellulosic(callose) materials that later become infused withphenolic substances and form a sheath or lignituberaround the hypha (Fig. 6-4).

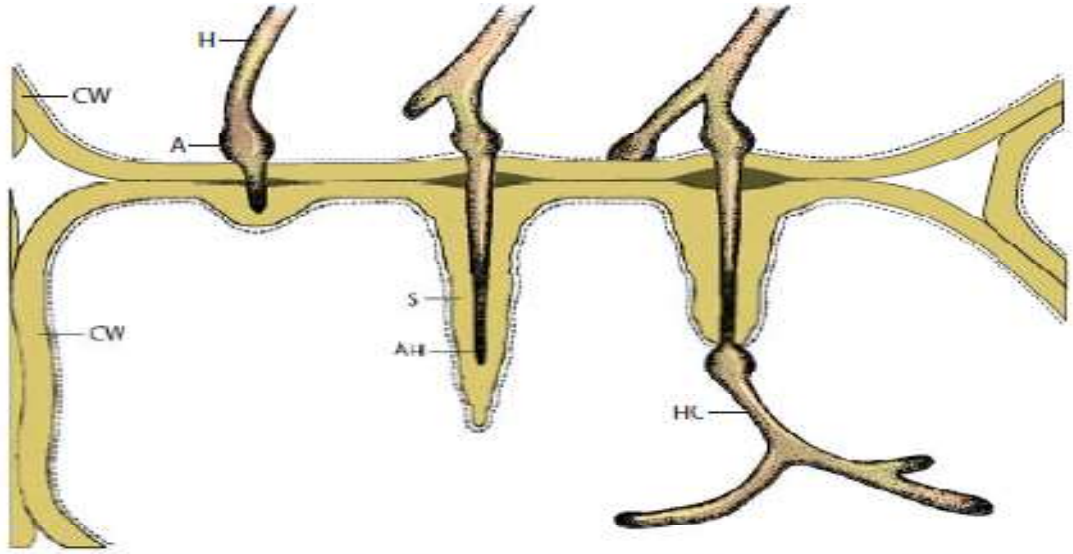



**FIGURE 6-4** Formation of a sheath around a hypha (H) penetrating a cell wall (CW). A, appressorium; AH, advancing hypha still enclosed in sheath; HC, hypha in cytoplasm; S, sheath.

**Histological Defense StructuresFormation of Cork Layers**

Infection by fungi or bacteria, and even by some virusesand nematodes, frequently induces plants to formseveral layers of cork cells beyond the point of infection(Figs. 6-5 and 6-6), apparently as a result ofstimulationof the host cells by substances secreted by the pathogen.The cork layers inhibit furtherinvasion by the pathogenbeyond the initial lesion and also block the spread ofany toxic substances that the pathogen may secrete.Furthermore, cork layers stop the flow of nutrients andwater from the healthy to the infected area and deprivethe pathogen of nourishment.

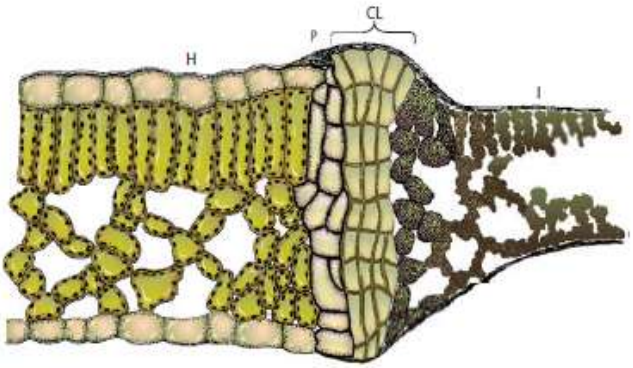



FIGURE 6-5 Formation of a cork layer (CL) between infected (I) and healthy (H) areas of leaf. P, phellogen. [After Cunningham (1928). *Phytopathology* **18**, 717–751.]

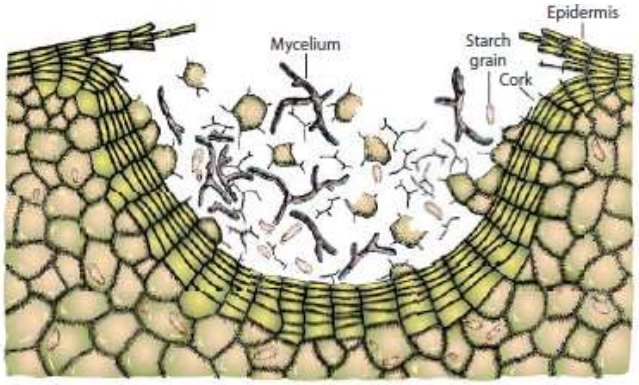
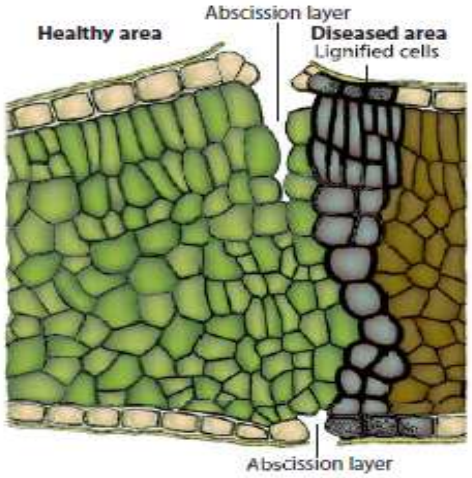



FIGURE 6-6 Formation of a cork layer on a potato tuber following infection with *Rhizoctonia*. [After Ramsey (1917). *J. Agric. Res.* **9**, 421–426.]

**Formation of Abscission Layers**

Abscission layers are formed on young, active leaves ofstone fruit trees after infection by any of several fungi,bacteria, or viruses. An abscission layer consists of a gapformed between two circular layers of leaf cells surroundingthe locus of infection. Upon infection, themiddle lamella between these two layers of cells isdissolved throughout the thickness of the leaf, completelycutting off the central area of the infectionfrom the rest of the leaf (Fig. 6-7).



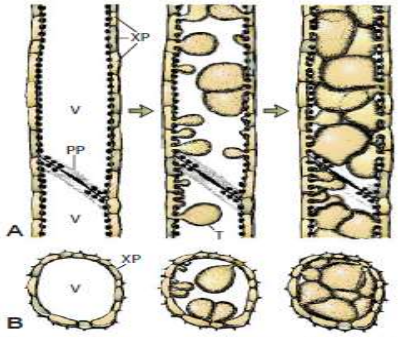




FIGURE 6-8 Development of tyloses in xylem vessels. Longitudinal (A) and cross section (B) views of healthy vessels (left) and of vessels with tyloses. Vessels at right are completely clogged with tyloses. PP, perforation plate; V, xylem vessel; XP, xylem parenchyma cell; T, tylosis.

**Formation of Tyloses**

Tyloses form in xylem vessels of most plants undervarious conditions of stress and during invasion by mostof the xylem-invading pathogens. Tyloses are overgrowthsof the protoplast of adjacent livingparenchymatouscells, which protrude into xylem vessels throughpits (Fig. 6-8).

**Deposition of Gums**

Various types of gums are produced by many plantsaround lesions after infection by pathogens or injury.

Gum secretion is most common in stone fruit trees butoccurs in most plants. The defensive role of gums stemsfrom the fact that they are deposited quickly in the intercellular spaces and within the cells surrounding the locusof infection, thus forming an impenetrable barrier thatcompletely encloses the pathogen. The pathogen thenbecomes isolated, starves, and sooner or later dies.

**Necrotic Structural Defense Reaction:**

**Defensethrough the Hypersensitive Response**

The hypersensitive response is considered a biochemicalrather than a structural defense mechanism but isdescribed here briefly because some of the cellularresponses that accompany it can be seen with thenakedeye or with the microscope. In many host–pathogencombinations, as soon as the pathogenestablishescontact with the cell, the nucleus moves toward theinvading pathogen and soon disintegrates. At the sametime, brown, resin-like granules form in the cytoplasm,first around the point of penetration of the pathogenand then throughout the cytoplasm. As the browningdiscoloration of the plant cellcytoplasm continues anddeath sets in, the invading hypha begins to degenerate(Fig. 6-9). In most cases the hypha does not grow outof such cells, and further invasion is stopped. In bacterialinfections of leaves, the hypersensitive responseresults in the destruction of all cellular membranes ofcells in contact with bacteria, which is followed bydesiccation and necrosis of the leaf tissues invaded bythe bacteria.

Although it is not quite clear whether the **HR** is thecause or the consequence of resistance, this type ofnecrotic defense is quite common, particularly in diseasescaused by obligate fungal parasites and by viruses(Fig. 6-10A), bacteria (Fig. 6-10B), and nematodes.

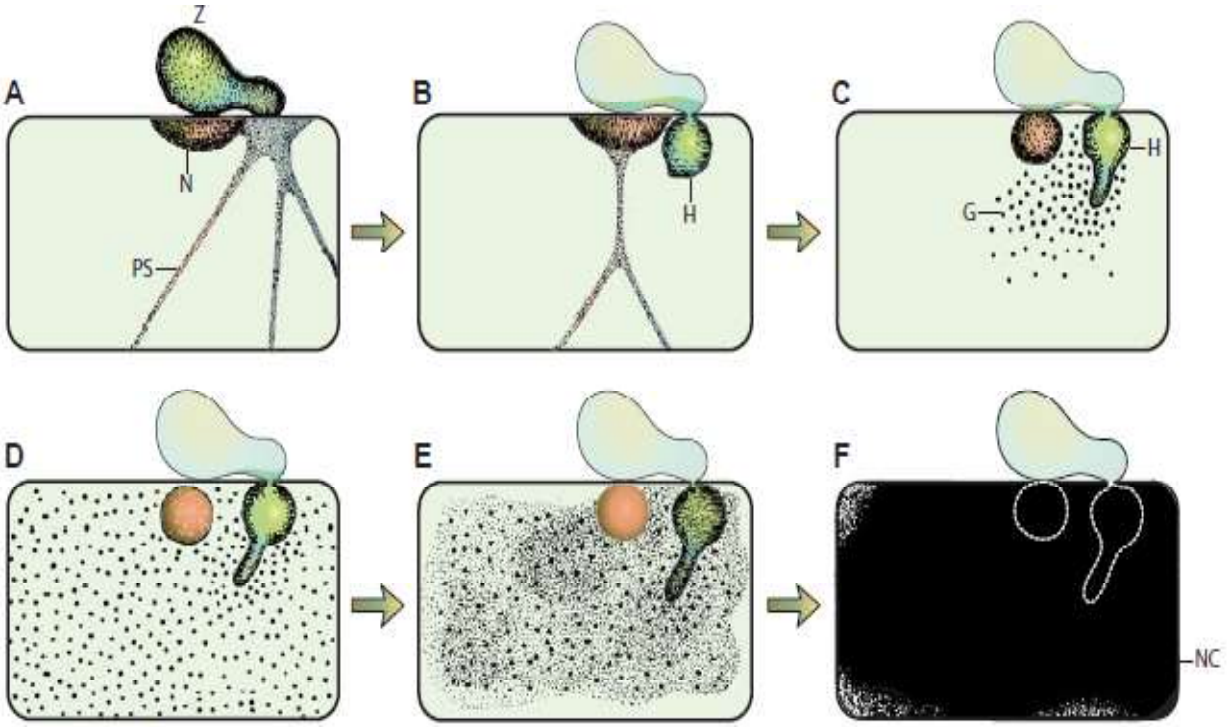



FIGURE 6-9 Stages in the development of the necrotic defense reaction in a cell of a very resistant potato variety infected by *Phytophthora infestans*. N, nucleus; PS, protoplasmic strands; Z, zoospore; H, hypha; G, granular material; NC, necrotic cell. [After Tomiyama (1956). *Ann. Phytopathol. Soc. Jpn.* 21, 54–62.]

**Induced biochemical defenses:**

Induced Biochemical Nonhost ResistanceAs mentioned earlier, nonhost resistance is the resistancethat keeps a plant protected from pathogens that are,through evolution, incompatible with that host.

Although the nature of nonhost resistance is unknown,for a pathogen it can be as big a gap to bridge as thedifference between the features of a potato plant andan oak tree, or as close as the difference between thefeatures of potato and tomato, or barley and wheat.

It appears, however, that in some plant/pathogeninteractions of taxonomically unrelated plants (e.g.,potato and oak or oak and wheat), nonhost resistanceis controlled by constitutive defenses and/or defensesinduced by nonspecific stimuli in a nonspecific manner.

Such defenses include physical topography and thestructures present on the plant, the presence of toxic orthe absence of essential compounds, and so on.

**Induced Biochemical Defenses in Quantitative(Partial, Polygenic, General, or Horizontal)Resistance**

In quantitative (partial, polygenic, multigenic,general, field, durable, or horizontal) resistance, plantsdepend on the action of numerous genes, expressed constitutivelyor upon attack by a pathogen (induced resistance).

These genes provide the plants with defensivestructures or toxic substances that slow down or stopthe advance of the pathogen into the host tissues andreduce the damage caused by the pathogen. Quantitativeresistance is particularly common in diseases causedby nonbiotrophic pathogens. Quantitative resistancemay vary considerably, in some cases being specificagainst some of the strains of a pathogen, in others beingeffective against all strains of a pathogen, or providingresistance against more than one pathogen. Genes forquantitative resistance are present and provide a basallevel of resistance to all plants against all pathogensregardless of whether the plant also carries major (or **R**)genes against a particular pathogen.

**Function of Gene Products in QuantitativeResistance**

Unlike most major (or **R**) genes involved in monogenicresistance, which appear to code for components thathelp the host recognize the pathogen and to subsequentlyexpress the hypersensitive response, genes forquantitative resistance seem to be involved directly inthe expression or production of some sort of structuralor biochemical defense. Quantitative resistance defensesare basically the same ones that follow the hypersensitiveresponse in monogenic resistance; in quantitativeresistance, however, defenses generally do not follow ahypersensitive response and cell death because the latterdo not usually occur in quantitative resistance. Genesinvolved in quantitative resistance are present in thesame areas of plant chromosomes that contain the genesinvolved in defense responses, such as the production ofphenylalanine ammonia lyase, hydroxyproline-rich glycoproteins,and pathogenesis–related proteins.

**Pathogenesis-Related Proteins:**

Pathogenesis-related proteins, often called PR proteins, are a structurally diverse group of plant proteins that are toxic to invading fungal pathogens. They are widely distributed in plants in trace amounts, but are produced in much greater concentration following pathogen attack or stress. PR proteins exist in plant cells intracellularly and also in the intercellular spaces, particularly in the cell walls of different tissues. Varying types of PR proteins have been isolated from each of several crop plants. Different plant organs, e.g., leaves, seeds, and roots, may produce different sets of PR proteins. Different PR proteins appear to be expressed differentially in their hosts in the field when temperatures

become stressful, low or high, for extended periods. The several groups of PR proteins have been classified according to their function, serological relationship, amino acid sequence, molecular weight, and certain other properties. PR proteins are either extremely acidic or extremely basic and therefore are highly soluble and reactive. **At least 14 families of PR proteins** are recognized.The better known PR proteins are **PR1** proteins (antioomycete and antifungal), **PR2** (b-1,3- glucanases), **PR3** (chitinases), **PR4** proteins (antifungal), PR6 (proteinase inhibitors), thaumatine-like proteins, defensins, thionins, lysozymes, osmotinlike proteins, lipoxygenases, cysteine-rich proteins, glycine-rich proteins, proteinases, chitosanases, and peroxidases. There are often numerous isoforms of each PR protein in various host plants. Although healthy plants may contain trace amounts of several PR proteins, attack by pathogens, treatment with elicitors, wounding, or stress induce transcription of a battery of genes that code for PRproteins.

**Systemic acquired resistance:**

Induced resistance is at first localized around the point of plant necrosis caused by infection by the pathogen or by the chemical, and it is then called local acquired resistance. Subsequently, resistance spreads systemically and develops in distal, untreated parts of the plant and is called systemic acquired resistance. It is known now that several chemical compounds, e.g., salicylic acid, arachidonic acid, and 2,6-dichloroisonicotinic acid, may induce localized and systemic resistance in plants at levels not causing tissue necrosis. Jasmonic acid is another type of compound, derived primarily from oxidation of fatty acids, that leads to systemic acquired resistance, often in cooperation with salicylic acid and ethylene, leading to the production ofdefensins. Probenazole, a synthetic chemical used in Asia for the control of rice blast disease caused by the fungus *Magnaporthe grisea*, has been shown to act upstream from the salicylic acid transcribing gene and, thereby, causing accumulation of salicylic acid. Probenazole induces systemic acquired resistance in rice against rice blast, in tomato against the bacterial pathogen *P. syringae* pv. tabaci, and in tobacco against the tobacco mosaicvirus.

Systemic acquired resistance acts nonspecifically throughout the plant and reduces the severity of disease caused by all classes of pathogens, including normally virulent ones. It has been observed in many dicot and monocot plants, but has been studied most in cucurbits, solanaceous plants, legumes, and gramineous plants following infection with appropriate fungi, bacteria, and viruses. Systemic acquired resistance is certainly produced in plants following expression of the hypersensitiveresponse.

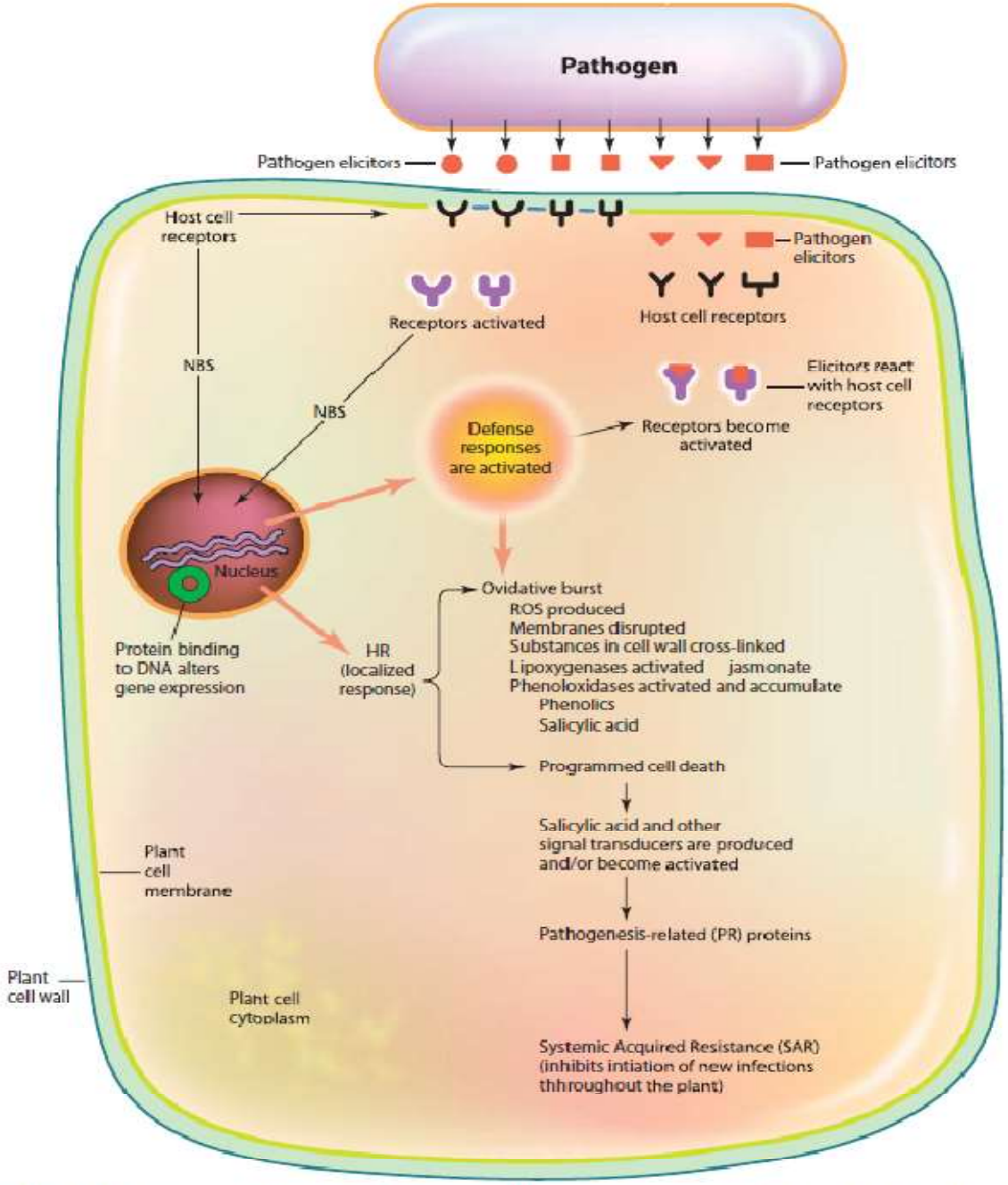



**FIGURE 6-11** Diagram of the hypothetical steps in the hypersensitive response defense of plants following interaction of an elicitor molecule produced by a pathogen avirulence gene with a receptor molecule produced by the matching host R gene.

**Gene for gene hypothesis:**

For each gene conditioning rust reaction in the host there is a specific gene conditioningpathogenicity in the parasite.For each resistance gene in the host there is a corresponding gene for avirulence in the pathogen conferring resistance and viceversa.Flor (1946, 47) showed correlation between inheritance of pathogenicity and resistance to linseedrustcausedby*Melampsoralini*whichisnowcommonlyknownasgene-for–genehypothesis.
that "for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite.

The concept has been applied with varying degree of proof to other host pathogen combinations including viruses, bacteria, fungi, nematodes, insects and a flowering plant (Orobanche).

RR = homozygous resistant ; Rr = heterozygous resistant; rr = homozygous susceptible
AVR AVR = homozygous avirulent; AVR avr = heterozygous avirulent; avr avr = homozygous virulent

| | | HOST GENOTYPE | |
|---|---|---|---|
| | | RR or Rr | rr |
| PATHOGEN GENOTYPE | AVR avr or AVR AVR | Disease-resistant | Susceptible to disease |
| | avr avr | Susceptible to disease | Susceptible to disease |

**Gene-for-gene hypothesis Multifactor Interactions**

| Avirulence/virulence | Resistance/susceptibility | | | |
|---|---|---|---|---|
| | R1 R2 | r1 R2 | R1r2 | r1r2 |
| A1A2 | - | - | - | + |
| a1A2 | - | - | + | + |
| A1a2 | - | + | - | + |
| a1a2 | + | + | + | + |

where, – = Resistance
+ = Susceptible

(Agrios 2007)

**Resistance genes**

**Classes of resistance gene**

There are several different classes of R Genes. The major classes are the NBS-LRR genes and thecellsurfacepatternrecognitionreceptors(PRR).TheproteinproductsoftheNBS-LRRRgenes contain a nucleotide binding site (**NBS**) and a leucine rich repeat (**LRR**). The protein products of the PRRs contain extracellular, juxtamembrane, transmembrane and intracellular non-RD kinase domains.

Within the NBS-LRR class of R genes are two subclasses:

One subclass has an amino-terminal Toll/Interleukin 1 receptor homology region (**TIR**). This includes the *N* resistance gene of tobacco against tobacco mosaic virus (**TMV**).

The other subclass does not contain a TIR and instead has a leucine zipper region at its aminoterminal.

The protein products encoded by this class of resistance gene are located within theplantcell cytoplasm.

The PRR class of R genes includes the rice XA21 resistance gene that recognizes the ax21 peptide and the *Arabidopsis* FLS2 peptide that recognizes the flg22 peptide from flagellinhere are other classes of R genes, such as the extracellular LRR class of R genes; examples include rice Xa21D for resistance against *Xanthomonas* and the *cf* genes of tomato that confer resistance against *Cladosporium fulvum.*

The *Pseudomonas* tomato resistance gene (***Pto***) belongs to a class of its own. It encodes a er/Thr kinase but has no LRR. It requires the presence of a linked NBS-LRR gene, *prf*, foractivity.

**Specificity of resistance genes**

R gene specificity (recognising certain *Avr* gene products) is believed to be conferred by the leucine rich repeats. LRRs are multiple, serial repeats of a motif of roughly 24 amino acids in length, with leucines or other hydrophobic residues at regular intervals. Some may also contain regularly spaced prolines andarginines.

LRRs are involved in protein-protein interactions, and the greatest variation amongst resistance genes occurs in the LRR domain. LRR swapping experiments between resistance genes in flaxrust resulted in the specificity of the resistance gene for the avirulence gene changing.

**Recessive resistance genes**

Most resistance genes are autosomal dominant but there are some, most notably the *mlo* gene in barley, in which monogenic resistance is conferred by recessive alleles. *mlo*protects barley against nearly all pathovars of *powderymildew*.

**Avirulence genes**

The term "avirulence gene" remains useful as a broad term that indicates a gene that encodes any determinant of the specificity of the interaction with the host. Thus, this term can encompass some conserved microbial signatures (also called pathogen or microbe associated molecularpatterns (**PAMPs or MAMPs**) and pathogen effectors (e.g. bacterial type III effectors and oomycete effectors) as well as any genes that control variation in the activity of those molecules.

There is no common structure between avirulence gene products. Because there would be no evolutionary advantage to a pathogen keeping a protein that only serves to have it recognised by the plant, it is believed that the products of *Avr* genes play an important role in virulence in genetically susceptible hosts.

*Example:* AvrPto is a small triple-helix protein that, like several other effectors, is targeted to the plasma membrane by N-myristoylation. *AvrPto* is an inhibitor of PRR kinase domains. PRRs signal plants to induce immunity when PAMPs are detected. The ability to target receptor kinases is required for the virulence function of AvrPto in plants. However, Pto is a resistant gene that can detect AvrPto and induce immunity as well.AvrPto is an ancient effector that is conserved in many *P. syringae* strains, whereas Pto R gene is only found in a few wild tomato species. This suggests recent evolution of the Pto R gene and the pressure to evolve to target AvrPto, turning a virulence effector to an avirulenceeffector.

Unlike the MAMP or PAMP class of avr genes that are recognized by the host PRRs, the targets of bacterial effector avr proteins appear to be proteins involved in plant innateimmunity signaling, as homologues of Avr genes in animal pathogens have been shown to do this. For example, the AvrBs3 family of proteins possessesDNA binding domains, nuclearlocalisation signals and acidic activation domains and are believed to function by altering host celltranscription.

**Biotrophy and gene for gene systems:**

All the parasites in which gene for gene relationship has been proved are essentially biotrophic or biotrophs at least for some time after start ofinfection.

(*Xanthomonas campestris* pv. *malvacearum*, *Phytophthora infestans*, *Venturia inaequalis* (Vander Plank,1978).

The genes-for-gene systems thus involvebiotrophy.

But the converse is not necessarily true. For example, *Plamodiophora brassicae* , the cause of club root of crucifers, is biotrophic but no evidence has yet been presented in the literature to suggest that host-pathogen interaction in them is based on a gene for gene systems.

According to Van der Plank (1978), specificity in gene for gene relationships lies in susceptibility.

**Table. The diagonal check for specificity in a gene-for gene relationship**

| Pathogen | Plant | | | | |
|---|---|---|---|---|---|
| | $R_1R_1$[b] | $R_2R_2$ | $R_3R_3$ | $R_4R_4$ | $R_5R_5$ |
| $v_1v_1$ | S | R | R | R | R |
| $v_2v_2$ | R | S | R | R | R |
| $v_3v_3$ | R | R | S | R | R |
| $v_4v_4$ | R | R | R | S | R |
| $v_5v_5$ | R | R | R | R | S |

a. Plant reaction when resistance gene $R_1, R_2, R_3, R_4, R_5$ at interact with virulence genes $v_1, v_2, v_3, v_4, v_5$ at five loci pathogen

b. Resistance is assumed to be dominant and RR can be r by Rr. Virulence is assumed to be recessive. However, re resistance and dominant virulence are also known.

R= resistant S= susceptible

- He explains it with the help of interactions of five host and five pathogens attacking them specifically.
- Suppose there are five host varieties with five d different R genes; R1, R2, R3--------R5. A plant with resistance gene R1 is attacked by a pathogen having virulence gene v1 and not to pathogen without this particular resistance gene irrespective of how many the virulence genes it mayhave.

Vander Plank (1978) elaborated protein for proteins hypothesis as a biochemical explanation of gene for geneinteraction.

The protein for protein hypothesis states that in gene for gene diseases the mutual recognition of host and pathogen is not by the genes themselves but by their coded proteins.

Vander Plank (1978) hypothesized that in susceptibility the pathogen excretes a protein

(virulence for product) into the host cell which copolymenizes with a complementary host protein (resistance gene product). This co-polymerization interferes with one auto regulation of the host gene that codes for the protein and by so doing turns the gene on to produce more protein.

In resistance, the protein specified by the gene for avirulence in the pathogen and excreted into the host does not polymerize with the protein coded for by the gene for resistance. It is not recognized by the host atall.

**From a practical point of view, gene for gene relationship can be used to study the following:**

1. The source of pathogenic variability inpathogens
2. The mutability of resistance and virulencegenes
3. Why host resistance is expressed under one set of conditions and notothers
4. Prediction of putative genotypes
5. Racenomenclature
6. Genetic dissection of complexloci
7. Cataloguing and storing of R genes in the form of plant seeds or cuttings and V genes in the form of pathogen strains
8. Management and deployment of resistance genes in space and tome
9. Detection of linkage and allelic relationship
10. Geographic distribution of R and V genes
11. Synthesis of multilines and multigene cultivars.

**Vertifolia effect:**

The vertifolia effect was discovered by Van der Plank (1963) who named it after a potato cultivar of this name, in which the effect was very pronounced. The vertifolia effect is a loss of horizontal resistance which occurs during breeding for vertical resistance. Its meaning was later extended to include the loss of horizontal resistance that occurs during breeding under the protection ofpesticides.

The level of horizontal resistance can only be assessed by the level of parasitism. Clearly, if there is no parasitism because of a functioning vertical resistance, or a pesticide, the level of horizontal resistance cannot be assessed. Because individual plants with a high level of horizontal resistance are rather rare in a mixed screening population, the chances are that individuals with a relatively

low level of horizontal resistance will then be selected on the basis of their other attributes. The loss is usually quite small in a single breeding cycle but, after many cycles, it can become very serious indeed.

The prime example of the vertifolia effect is the loss of horizontal resistance to potato blight (*Phytophthora infestans*) that has continued ever since both the discovery of Bordeaux mixture in the late nineteenth century, and the discovery of vertical resistance in the twentieth century. A loss of horizontal resistance to cotton pests has continued ever since the discovery of DDT in the 1940s.

The vertifolia effect is a very modern phenomenon. Its overall consequences are seen in the high levels ofhorizontal resistance in heritage cultivars, when they are compared to modern cultivars. This is the main reason why heritage cultivars are so valued by organic farmers. One of the main objectives of most amateur plant breeders will be to restore the horizontal resistances that were lost to the vertifolia effect.

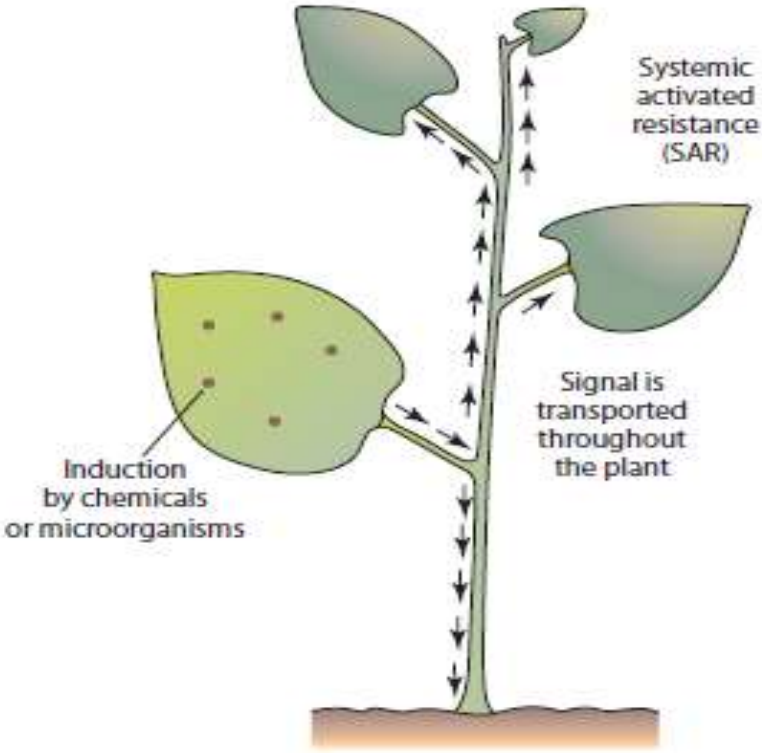



**FIGURE 6-24** Principle of systemic activated (or acquired) resistance. A leaf treated with certain chemicals or with pathogens causing necrotic lesions produces a signal compound(s) that is transported systemically throughout the plant and activates its defense mechanisms, making the entire plant resistant to subsequent infections.

**Immunization of plants againstpathogens:**

**Defense through Plantibodies:**

In humans and animals, defenses against pathogens areoften activated by natural or artificial immunization,

i.e., by a subminimal natural infection with thepathogen or by an artificial injection of pathogen proteins

and other antigenic substances. Both events resultin the production of antibodies against the pathogenand, thereby, in subsequent prolonged protection(immunity) of the human or animal from infection byany later attacks of the pathogen.

Plants, of course, do not have an immune system likethat of humans and animals, i.e., they do not produceantibodies. In the early 1990s, however, transgenic plantswere produced that were genetically engineered to incorporatein their genome, and to express, foreign genes,such as mouse genes that produce antibodies againstcertain plant pathogens. Such antibodies, encoded byanimal genes but produced in and by the plant, are calledplantibodies. It has already been shown that transgenicplants producing plantibodies against coat proteins ofviruses, e.g., *artichoke mottle crinkle virus*, to which theyare susceptible, can defend themselves and show someresistance to infection by these viruses. It is expected that,in the future, this type of plant immunization will yielddividends by expressing animal antibody genes in plantsthat will produce antibodies directed against specificessential proteins of the pathogen, such as viral coat proteinsand replicase or movement proteins, and fungal andbacterial enzymes of attack.

**Systemic acquired resistance:**

**Induction of Plant Defenses by Artificial Inoculation with Microbes or by Treatment with Chemicals:**

Induced resistance is at first localized around the point of plant necrosis caused by infection by the pathogen or by the chemical, and it is then called local acquired resistance (Fig. 6-22A). Subsequently, resistance spreads systemically and develops in distal, untreated parts of the plant and is called systemic acquired resistance (Fig. 6-22B). It is known now that several chemical compounds, e.g., salicylic acid, arachidonic

acid, and 2,6-dichloroisonicotinic acid, may induce localized and systemic resistance in plants at levels not causing tissue necrosis. Jasmonic acid is another type of compound, derived primarily from oxidation of fatty acids, that leads to systemic acquired resistance, often in cooperation with salicylic acid and ethylene, leading to the production of defensins. Probenazole, a synthetic chemical

used in Asia for the control of rice blast disease caused by the fungus *Magnaporthe grisea*, has been shown to act upstream from the salicylic acid transcribing gene and, thereby, causing accumulation of salicylic acid. Probenazole induces systemic

acquired resistance in rice against rice blast, in tomato against the bacterial pathogen *P. syringae* pv. *tabaci*, and in tobacco against the *tobacco mosaic virus*.

Similarly, riboflavin was shown to induce systemic acquired resistance but it activates it in a distinct mannernot involving salicylic acid.Salicylic acid and isonicotinic acid are true SAR activatorsbecause not only do they induce resistance to thesame spectrum of pathogens and induce expression ofthe same genes as pathogens, but these chemicals haveno antimicrobial activity.

**R Gene Resistance, Race-Specific, Monogenic, or Vertical Resistance:**

Many plant varieties are quite resistant to some races of a pathogen while they are susceptible to other races of the same pathogen. In other words, depending on the race of the pathogen used to infect a variety, the variety may appear strongly resistant to one pathogen race and susceptible to another race (**race specific)** under a variety of environmental conditions. Such resistance differentiates clearly between races of a pathogen, as it is effective against specific races of the pathogen and ineffective against others (Figs. 4-9 and 4-10). Such resistance is sometimes called strong, major, race-specific, qualitative, or differential resistance, but it was more commonly referred to in the past as **vertical resistance**.

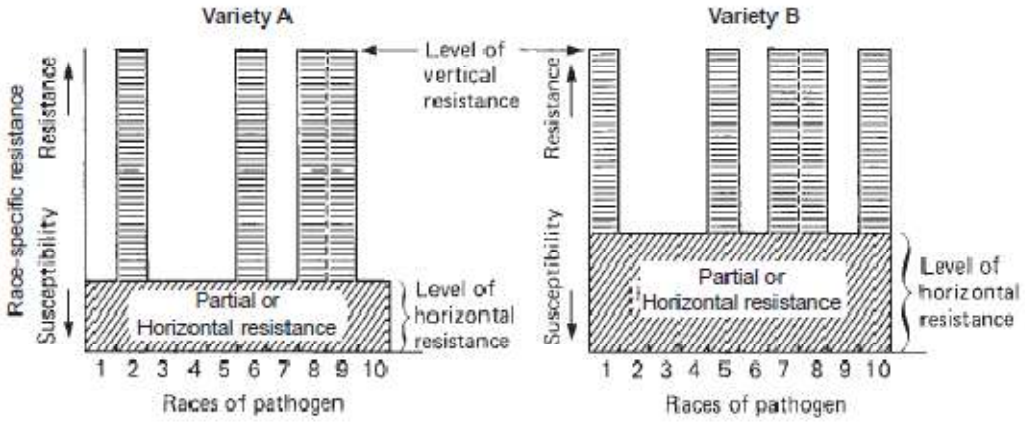



FIGURE 4-9 Levels of horizontal and vertical resistance of two plant varieties toward 10 races of a pathogen. [After Vanderplank (1984).]

Race-specific resistance is always controlled by one or a few genes (thereby the names **monogenic** or **oligogenic resistance**). These genes, referred to as R genes, control a major step in the recognition of the pathogen by the host plant and therefore play a major role in the expression of resistance. In the presence of race-specific resistance, the host and pathogen appear incompatible. The host may respond with a hypersensitive reaction, may appear immune, or may inhibit pathogen reproduction. Often, race-specific resistance inhibits the initial establishment of pathogens that arrive at a field from host plants that lack, or have different, major genes for resistance. Race-specific resistance inhibits the development of epidemics by limiting the initial inoculum or by limiting reproduction after infection.

Complete resistance may be provided by a single resistance gene. Often, it is desirable to combine, or **pyramid**, more than one resistance gene (R1R2, R1R3, R1R2R3) in the same plant, which then is resistant to all the pathogen races to which each of the genes provides resistance. A plant species may have as many as 20 to 40 resistance genes against a particular pathogen, although each variety may have only one or a few of these genes. For example, wheat has 20 to 40 genes for resistance against the leaf rust fungus *Puccinia recondita*, barley has a similar number of genes against the powdery mildew fungus *Erysiphe graminis hordei*, and cotton has almost as many against the bacterium *Xanthomonas campestris* pv. *malvacearum*. Each gene for resistance, such as R2, makes the plant resistant to all the races of the pathogen that contain the corresponding gene for avirulence. This pathogen race and its avirulence gene (**A2**), however, are detected because the pathogen attacks plants that lack the particular gene for resistance (**R2**).

Whether partial or race specific, true resistance is generally controlled by genes located in the plant chromosomes in the cell nucleus. There are, however, several plant diseases in which resistance is controlled by genetic material contained in the cytoplasm of the cell.

Such resistance is sometimes referred to as **cytoplasmic resistance**. The two best-known cases of cytoplasmic resistance occur in the southern corn leaf blight caused by *Bipolaris (Helminthosporium) maydis* and the yellow leaf blight caused by *Phyllosticta maydis*. Resistance in these is conferred by the lack of a gene in mitochondria of normal cytoplasm of various types of corn that encodes a receptor for the host-specific toxin produced by each pathogen. The presence of such a gene in mitochondria of Texas male-sterile cytoplasm makes all corn lines with Texas male-sterile cytoplasm susceptible to these pathogens.

Varieties with race-specific (monogenic or oligogenic) resistance generally show complete resistance to a specific pathogen under most environmental conditions, but a single or a few mutations in the

pathogen mayproduce a new race that may infect the previously resistant variety. On the contrary varieties with partial (polygenic) resistance are less stable and may vary in their reaction to the pathogen under different environmental conditions, but a pathogen will have to undergo many more mutations to completely break down the resistance of the host. As a rule, a combination of major and minor genes for resistance against a pathogen is the most desirable makeup for any plant variety.

| **Horizontal resistance** | **Vertical resistance** |
|---|---|
| Controlled by many genes (Poly-/ multigene resistance) | Controlled by one or a few genes (Monogenic or oligogenic) |
| Each gene plays a minor role | The gene plays a major role in expression resistance |
| Non-differential types of Resistance (Non- specific) | Differentiates between races of a pathogen. (host variety resistant to one race but not otherrace) |
| Affected by environmental conditions. | Less affected by environmental conditions. Provide complete resistance |
| Does not provide complete protection but slows down the disease spread. | Qualitative in nature |
| Quantitative innature | In plant varieties with vertical resistance, resistance may break down if there is a single or a few mutations in the pathogen producing a new race that may infect the previously resistantvariety. |
| Varieties with partial resistance though less stable and may vary in their reaction to the pathogen under different environmental conditions, but a pathogen will have to undergo several mutations to completely break down the resistance of thehost. | |

## 7. Physiological (photosynthesis, respiration, translocation of water and nutrients) and molecular (protein and nucleic acid) changes in diseasedplants

### A. Effect on photosynthesis:

In disease symptoms such as cause chlorosis, necrotic lesions, reduced growth, mosaic, leaf spot, blight, and other kinds of diseases in which there is destruction of leaf tissue, e.g., cereal rusts, viral mosaics, yellowing and stunting diseases etc. photosynthesis is affected. Photosynthesis is also reduced in defoliations which causes decrease in the photosynthetic surface of the plant. Plant pathogens reduce photosynthesis, especially in the late stages of diseases, by affecting the chloroplasts and causing their degeneration. Photosynthesis is reduced because the toxins, such as tentoxin and tabtoxin, which inhibit some of the enzymes that are involved directly or indirectly inphotosynthesis.

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 (Figs. 3-1A–3-1C), bacterial leaf spots (Fig. 3-1D), viral mosaics (Fig. 3-1E) and

yellowing and stunting diseases (Fig. 3-1F), 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.

**B. Effect of pathogens on host plantrespiration**

Respiration is the process by which cells, through the enzymatically controlled oxidation (burning) of the energy-rich carbohydrates and fatty acids, liberateenergy in a form that can be utilized for the performanceof various cellular processes. Plant cells carry outrespiration in, basically, two steps. The first stepinvolves the degradation of glucose to pyruvate and iscarried out, either in the presence or in the absence ofoxygen, by enzymes found in the ground cytoplasm ofthe cells. The production of pyruvate from glucosefollows either the glycolytic pathway, otherwise knownas glycolysis, or, to a lesser extent, the pentose pathway.

The second step, regardless of the pathway, involves thedegradation of pyruvate, however produced, to $CO_2$ andwater. This is accomplished by a series of reactionsknown as the Krebs cycle, which is accompanied by theso-called terminal oxidation and is carried out in themitochondria only in the presence of oxygen. The energy produced through respiration is utilizedby the plant for all types of cellular work, such as accumulationand mobilization of compounds, synthesis ofproteins, 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. 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 defensemechanisms 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.

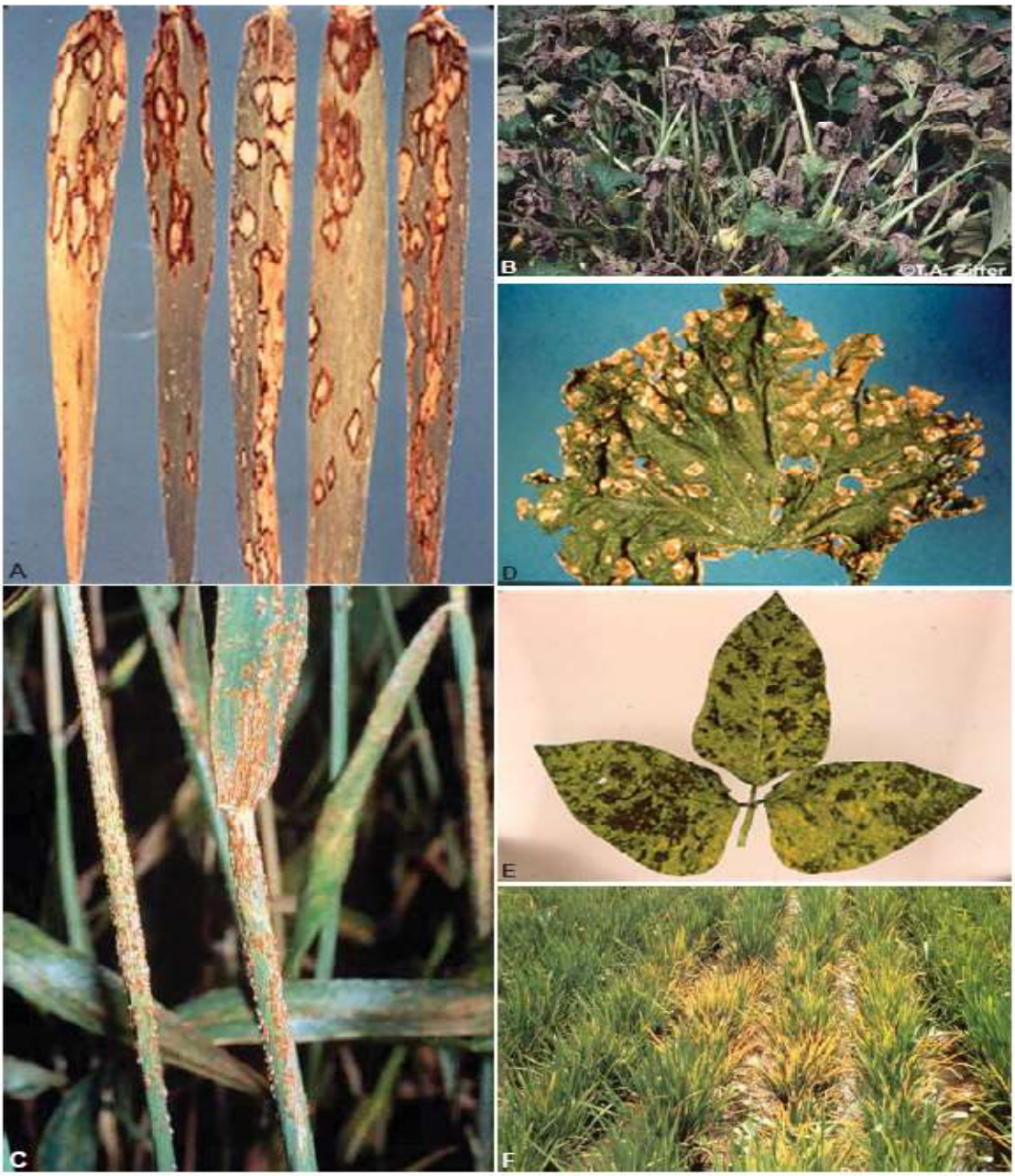



**FIGURE 3-1** Ways in which pathogens reduce photosynthetic area and, thereby, photosynthesis in plants. (A) Spots on barley leaves caused by the fungus *Rhynchosporium sp.* (B) Nearly complete destruction of pumpkin leaves infected heavily with the downy mildew oomycete *Pseudoperonospora cubensis*. (C) Countless tiny lesions on stems and leaves of wheat plant infected with the stem rust fungus *Puccinia graminis f.sp. tritici*. is. (D) Angular leaf spots on cucumber leaf caused by the bacterium *Pseudomonas lacrymans*. (E) Reduced chlorophyll in yellowish areas of virus-infected plants, such as cowpea infected with *cowpea chlorotic mottle virus* or (F) by stunting and yellowing of rice plants infected with the *rice tungro virus*. [Photographs courtesy of (A) Plant Pathology Department, University of Florida, (B) T. A. Zitter, Cornell University (C) I. Evans and (D) R. J. Howard, W.C.P.D., and (F) H. Hibino.]

### C. Effect on translocation of water & nutrients

In plant diseases in which the pathogen infects theleaves, transpiration is usually increased. This is theresult of destruction of at least part of the protection afforded the leaf by the cuticle, an increase in the permeabilityof leaf cells, and the dysfunction of stomata.In diseases such as rusts, in which numerous pustulesform and break up the epidermis (Figs. 3-6A and 3-6B),in most leaf spots (Fig. 3-6E), in which the cuticle, epidermis,and all the other tissues, including xylem, maybe destroyed in the infected areas, in the powderymildews, in which a large proportion of the epidermalcells are invaded by the fungus (Fig. 3-6C), and in applescab (Fig. 3-6D), in which the fungus grows betweenthe cuticle and the epidermis—in all these examples,the destruction of a considerable portion of the cuticleand epidermis results in an uncontrolled loss of waterfrom the affected areas. If water absorption and translocationcannot keep up with the excessive loss of water,loss of turgor and wilting of leaves follow. The suction

forces of excessively transpiring leaves are increasedabnormally and may lead to collapse or dysfunction ofunderlying vessels through the production of tylosesand gums.

**Interference with Translocation of OrganicNutrients 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 (Fig. 3-7) and eventually, again through plasmodesmata, into the protoplasm of living nonphotosynthetic 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 increasedrespiration. 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 cankersdevelop (Figs. 3-8A–3-8C), the pathogen attacks andremains confined to the bark for a considerable time.

During that time the pathogen attacks and may destroythe phloem elements in that area, thereby interferingwith the downward translocation of nutrients.


**FIGURE 3-2** Examples of reduction of water absorption by plants. (A) Destruction of roots of young seedlings by the damping-off oomycete *Pythium sp.* (B) Roots and stems of pepper plants killed by *Phytophthora sp.* (C) Wheat roots at different stages of destruction by the take-all fungus *Gaeumannomyces tritici.* (D) Infection of crown and roots of corn plant with the fungus *Fusarium.* (E) Numerous galls caused by the bacterium *Agrobacterium tumefaciens* on roots of a cherry tree. (F) Root knot galls caused by the nematode *Meloidogyne sp.* on roots of a cantaloupe plant. [Photographs courtesy of (A) Plant Pathology Department, University of Florida, (B) K. Pernezny, University of Florida, (C) W. McFadden, W.C.P.D., (D) Plant Pathology Department, Iowa State University, (E) Oregon State University, and (F) B. D. Bruton, USDA, Lane, Oklahoma.]

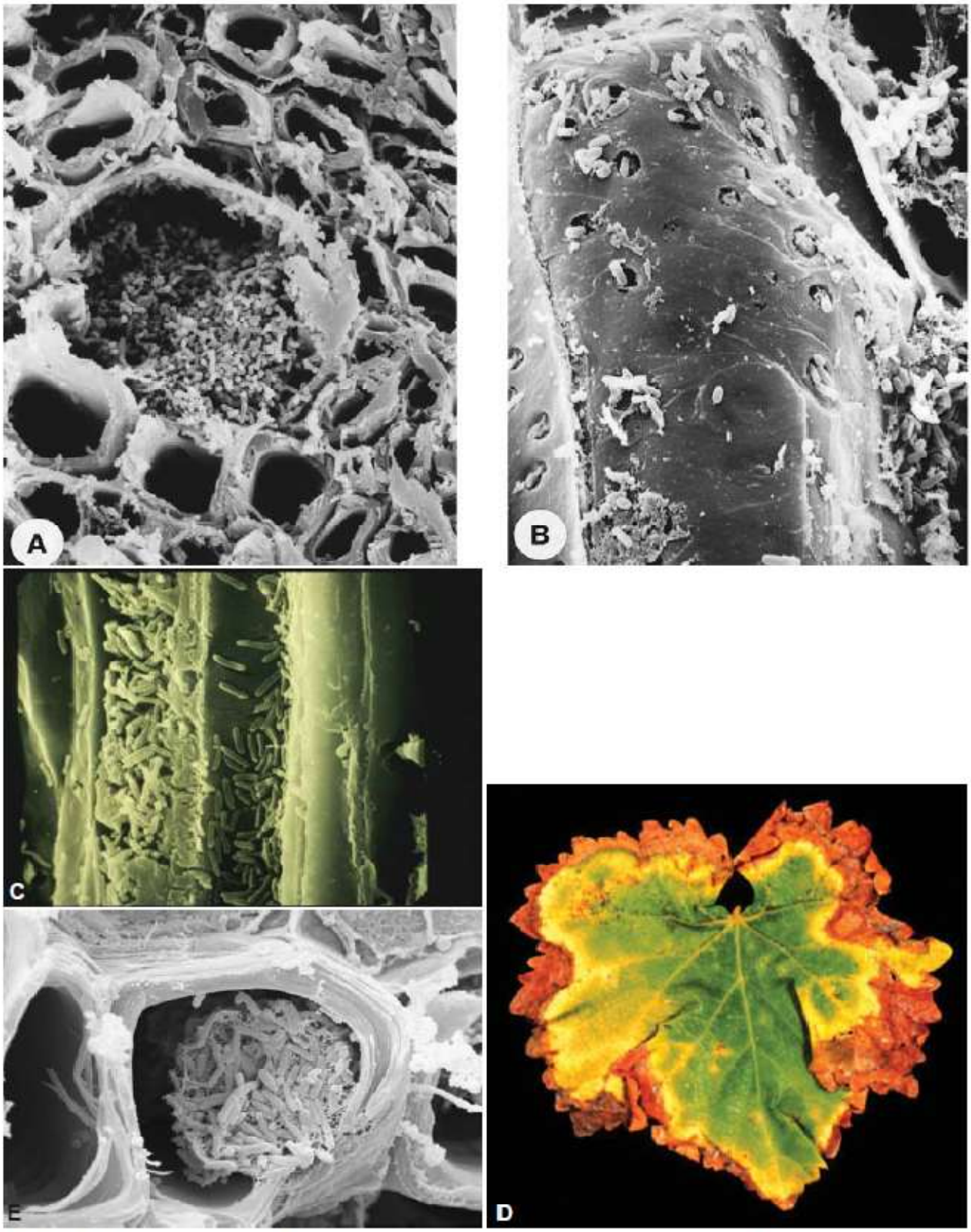



**FIGURE 3-4** (A) *Pseudomonas* bacteria clogging a xylem vessel of a young plant shoot. (B) Bacteria moving from one vessel to another and to adjacent parenchyma cells through xylem pits. (C) Bacteria of the xylem-inhabiting *Xylella fastidiosa* in a vessel of a grape plant. (D) Marginal scorching of a grape leaf from a plant infected with *X. fastidiosa*, the cause of Pierce's disease of grape. (E) *Xylella* bacteria in a cross section of a xylem vessel of an infected grape leaf. [Photographs courtesy of (A and B) E. L. Mansvelt, I. M. M. Roos, and M. J. Hattingh (1500×), (C) D. Cooke, provided by E. Hellman, Texas A&M University, (D) E. Hellman, and (E) E. Alves, Federal University of Lavras, Brazil.]

**Molecular (protein & nucleic acid) changes in diseased plants**

The most common effect of pathogenesis are changes in cell membrane permeability i.e. loss of **electrolytes** from the cell. 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. 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.

**Effect on 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 affectranscription 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 defense mechanisms of plantcells.

**Effect on 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 (defense) reactions to infection. Although a certain amount of some ofthese 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 defense reactions of plants.

## 8. Predisposition, survival of pathogen in nature and its spread, diseaseepidemics

**Predisposition**

Yarwood (1976) defined predisposition as 'The tendency of treatments and conditions acting before inoculation or before introduction of the incitant, to affect susceptibility to biotic and abiotic pathogens.'

**Sources of survival of pathogens**

**1)** Infected host as reservoir of inoculum (or) survival in vital association with livingplants.

**2)** Survival as saprophytes outside thehost.

**3)** Survival by means of specialized resting structures in or on the host or outside thehost.

**4)** Survival in association with insects, nematodes andfungi

**1. Survival in vital association with livingplants**

Survival of the plant pathogens in vital association with living plants is grouped into

**a. Seed**

The pathogen of loose smut of wheat, *Ustilago nuda tritici*, enters the stigma and style and infects the young seed, in which it survives as mycelium. The seed-infecting pathogens that cause loose smut of wheat and loose smut of barley are strikingly different from other smut fungi that attack cereal crops. Most of the others survive from season to season either in nonpathogenic association with seed or as spores in the soil. *Colletotrichum lindemuthianum*, the causative organism of bean anthracnose, can also infect the seed; unless the seed is killed, the fungus in newly sprouted bean seedlings initiates new infections. The bacteria that cause bean blights and bacterial blight of cotton survive the winter in infected seed. In Mexico, the fungus of late blight of potatoes (*Phytophthora infestans*) produces oospores but in colder regions of the world, the fungus overwinters as mycelium in diseased tubers.

**b. Collateralhosts**

Collateral hosts are those, which are susceptible to the plant pathogens of crop plants and provide adequate facilities for their growth and reproduction of these pathogens. Weeds, which survive and live during non-cropping season provide for the continuous growth and multiplication of the pathogen. For example, the fungal pathogen for blast disease of rice, *Pyricularia oryzae* can infect the grass weeds like *Bracguarua mutica, Dinebra retroflexa, Leersia hexandra, Panicum repens*, etc., and survive during off-season of rice-crop.

**c. Alternatehosts**

The role of alternate hosts is not important as of collateral hosts. However, when a pathogen has very wide host-range and is tolerant to wide range of weather conditions the alternate hosts become very, important source of survival of the pathogen. These alternate hosts are very important for the completion of the life cycle of heteroecious rust pathogens. e.g. in temperateregions the alternate host *Berberis vulgaris* of *Puccinia graminis tritici* (black/stem. rust pathogen on wheat), the barberry bush, grows side by side with the cultivated host,wheat.

**d. Self sown crops**

Self-sown plants, voluntary crops and early sown crops are reservoirs of many plant pathogens e.g., groundnut rust pathogen, *Puccinia arachidis* and ring mosaic of groundnut caused by tomato spotted wilt virus. Self-sown rice plants harbour the pathogen as well as vector. e,g., rice tungro virus and its vector, *Nephotettix virescens*.

**Survival of plant pathogens:**

Most plant pathogens can survive only by repeatedly reinfecting host plants. Thisprocess of repeated infection is known as an infection chain. If the infection chainis broken the pathogen dies out. The Swiss plant pathologist Ernst Gd.umann(1950) in his book Principl.es oJPlantInJection divided infection chains into twotypes depending on how the inoculum of the pathogen survives between leavingthe infected host (the distributor host) and arriving on the host to be infected (thereceiver host). In continuous infection chains the pathogen survives bycontinually infecting susceptible plants in an uninterrupted fashion.

Discontinuous infection chains occur when the disease cycle is interrupted byan epiphytic phase, a saprophytic phase or a resting stage.

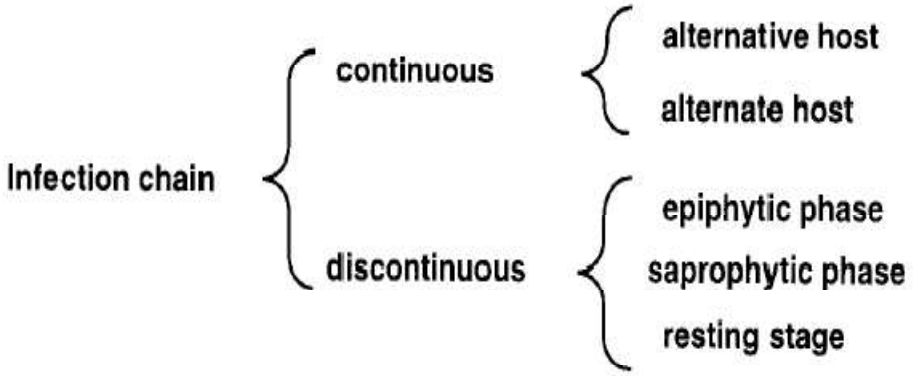



**Figure 12.1** Five types of infection chains available to plant pathogens.

**Continuous infection chains involving the same or alternative hosts**

Continuous infection chains involving the same or alternative hosts occur whenthe parasite survives by continually infecting plants of the same host species orplant species other than the main crop species. Such plants are calledalternative hosts and are often related to the crop species. If the alternativehost, which is often a weed species, does not show symptoms of disease, it iscalled a disease carrier. The parasite does not form resting structures nor does itsurvive saprophytically or epiphytically on plant surfaces. Survival therefore isdependent upon the presence of susceptible host plants. Such infection chainsare common in all major groups of plant parasites.

In tropical regions where year-round cropping of plant species such as rice androot crops (e.g. cassava, sweet potato, taro and yams) is a common practice, thecontinuous presence of host plants enables parasites to survive by continuouslyinfecting new hosts. In temperate regions, it is not uncommon for self-sown,volunteer crop plants to grow along roadsides, fencelines and irrigation channelsand as weeds in paddocks during periods when the main crop is not growing.These self-sown plants enable parasites to survive during intercrop periods andserve as sources of inoculum when the main crop is grown.The wheat growing areas of northern NSW and southern Queensland, wherecrops are sown in late autumn/early winter and harvested in late spring/earlysummer. are often referred to as rust liable areas. This is because the summerstorms which are prevalent in the region allow seed that has fallen to the groundduring the previous season's harvest to germinate. Susceptible self-sown plantsbecome infected and this enables the fungus to oversummer. In the southernregions of Australia the climate is more Mediterranean-like with winter rains andsummer drought. Under these conditions self-sown wheat plants do not occur insummer (except in irrigation areas), the fungus cannot survive and epidemics arerare. However, grasses such as barley grass (Hordeum Leporinum) and commonwheat grass (Agropgron scabrum) can serve as alternative hosts to various specialforms of Puccinia graminis. A similar infection chain occurs with other pathogensof wheat.

Many plant pathogens survive intercrop periods by infecting alternative hosts.For example, the black rot bacterium of brassicas (*Xanthomonas campestrus* pv.campestris) can survive on related weed species such as wild radish. Similarly,root-knot and lesion nematodes have a wide host range and infected alternativehosts play a major role in their survival.

**Continuous infection chains involving alternate hosts**

Some plant pathogens cannot be directly transmitted from an infected plant toanother plant of the same species. They must alternate between two completelyunrelated biological species. Even with

the modern technologies available today,scientists cannot transmit most plant viruses and phytoplasmas directly from

one plant to another. They can only be transmitted via a vector, usually but notalways, an insect. In many cases viruses enter into a biological relationship withtheir vector and in some instances multiply in the vector formore details on virus-vector relationships). The vector therefore acts as analternate or intermediate host and the infection chain alternates between the twounrelated species. Thus, the infection chain consists of plant-insect vector-plant.If the alternate host {the vector) is absent the infection chain is broken.Some heteroecious rust fungi also require an alternate host in the infectionchain. They cannot complete their disease cycle in the absence of the alternatehost. An example is the European pear or cluster rust fungus (Ggmnosporangium ) which is present in Europe, Asia Minor and North Africa. It has beenintroduced into North America but is currently absent from the SouthernHemisphere. The fungus forms clusters of aecia opposite spermagonia on theunderside of pear leaves (Purus communis) for more detail on theplant rusts). The aeciospores cannot infect pear. They can only infect thealternate host, the g,.rnnosperm Juniper (Juniperus spp.). Teliospores formed onthe alternate host cannot re-infect juniper plants, they can only infect pear. Thefungus does not form urediniospores and therefore cannot reproduce asexuallyon Junipents.

**Discontinuous infection chains involving an epiphytic phase**

Several fungal and bacterial pathogens survive on the surfaces of their hosts andother plants in a non-parasitic relationship as epiphytes. For example, the peachleaf curl fungus (*Taphrina dejormans*) causes leaves to develop yellow to reddishareas which progressively thicken and pucker, causing the leaf to curl and dropprematurely. Naked asci containing ascospores are produced on the uppersurface of the curled leaves. Ascospores germinate to form budding, yeast-likeconidia that can only infect very young leaves which are unavailable at the timewhen the budding colonies are formed.

The fungus survives during the summer months (when only mature leaves arepresent on trees) and through the winter months (when no leaves are present onthe deciduous peach trees) as an epiphyte on the bark and on the scales of thefollowing year's leaf buds. In the following spring, when the leaf buds swell andyoung leaves begin to emerge, the budding cells infect the young leaves and forma mycelium in young, undifferentiated host tissue. The fungus produces asci inthe early summer and the life cycle is completed. Satisfactory control of peachleaf curl can be achieved by a single application of a protectant fungicide (e.g.copper based sprays)just before bud-burst in the spring.

Bacterial canker of stone fruit caused by Pseudomonas sgrtngae pv. Syringaecauses sunken cankers, which frequently exude gum, on the woody tissue ofaffected trees. Infection can lead to premature

death of whole trees or infectedbranches. The bacterium overwinters in cankers and in spring it multiplies andspreads by rain splash to leaves where large epiphytic populations develop. Theseepiphytic populations provide the main source of inoculum for wound and leafscar infections in the following autumn. The same bacterium can also causebacterial blight of peas. Again, the bacterium is present as part of the normalleaf-surface microflora of healthy peas. Infection occurs following wounding ofplants.

**Discontinuous infection chains involving a saprophytic phase**

Many plant pathogenic bacteria and fungi survive during intercrop periods assaprophytes on diseased plant debris or on organic material present in or on soil.For example, the apple scab fungus (*Venturia tnaequalls)* infects apple leaves andfruits during spring and summer. The fungus overwinters as pseudothecialinitials in infected leaves and fruit that fall to the ground during autumn.Following the union of mycelium of compatible mating types in autumn, blackpseudothecia develop in the fallen leaves. The fungus then overwinters in thefallen leaves. In the following spring, the pseudothecia produce ascospores whichserve as the primary inoculum which starts the disease cycle in the new season.The primary infections resulting from ascospore infection produce conidia whichserve as the secondary inoculum that contributes towards the development ofepidemics. Disease levels can be reduced by preventing the fungus from overwintering in fallen leaves. This can be achieved by spraying with a systemicfungicide before leaves fall during autumn so that pseudothecial development isprevented. The application of nitrogen (e.g. urea) to the leaf litter accelerates leafdecomposition under mild winter conditions reducing the survival rate in leaves.

Many root-infecting pathogens of annual crops have a saprophytic phase intheir life cycle. When infected plants die, either as a result of disease or naturalsenescence, the pathogen survives saprophytically on the infected debris. Forexample the take-all fungus of wheat (GaeumannomAces gramtnis var. tritici)persists as a saprophyte in infected host debris during the intercrop period. Somepathogens that survive in diseased plant debris compete poorly as saprophyteswith the normal microbial inhabitants of soil. Consequently, their numbersdecline during the intercrop period. Such pathogens have been called rootinhabitants or soil lnvaders.

Other plant pathogenic bacteria {e.g. *Agrobacterium rhizogenes* which causescrown gall, *Ralstonta sotanacean-um* which causes bacterial wilt and Streptomgcesscabies which causes common scab of potato) and fungi (e.g. damping-off fungi and several species of *Fusarium, Pythium and Rhizoctonia*) can compete veryeffectively as saprophytes with the normal soil microflora. These parasites areoften referred to as soil inhabitants because they are part of the normal soilmicroflora.

**Discontinuous infection chains involving a resting stage**

Plant pathogenic bacteria, phytoplasmas and viruses do not form resting structures and can survive only by means of continuous infection chains or in the case of bacteria as saprophytes or epiphytes. Plant pathogenic bacteria do not form endospores. Some viruses retain their infectiousness in infected plant debris for long periods of time. For example, leaves infected with tobacco mosaic virus retain their infectiousness when kept dry under laboratory conditions for over 50 years. However, most viruses lose their infectiousness soon after their host plants die.

Fungi and nematodes on the other hand often form resting structures that enable them to survive, often for long periods, during the absence of suitable host plants or when the environment is unfavourable. The downy mildews produce oospores, the rusts and smuts form teliospores and many fungi produce chlamydospores. In some instances, resting spores can survive for periods of up to twenty years or more. The resting spores of Plasmodiophora brassicae (the cause of club root of brassicas) and Spongospora subterranea (the cause of

powdery scab of potato) can survive in soil for many years. Moreover, the spores of both species are stimulated to germinate only by exudates secreted from the roots of certain plant species reducing the probability of spores germinating in a 'no win' situation when potential hosts are absent. Many fungi produce sclerotia (e.g. Sclerotium roLfsii, ScLerotinta scLerotiorum, VerticilLium dahliae) which can survive in soil or in infected plant debris for periods ranging from months to many years, depending on the species. Other resting structures formed by fungi include sexual fruiting structures such as cleistothecia, perithecia andpseudothecia.

Nematodes also have adaptations that enable survival for long periods in soilin the absence of host plants. Eggs of the cyst nematodes (*Globodera* and*Heterodera* spp.) survive in egg cysts while those of root-knot nematodes(*Meloidogyne* spp.) survive in gelatinous egg masses. Both structures reduce therate of egg desiccation. Some species such as *Angutna funesta* and *Ditglenchusdipsaci* can survive for several years in an anhydrobiotic state (metabolically inactive with loss of water from body fluids). Some cyst nematodes (e.g. thepotato cyst nematode, *Globodera rostochiensrs*) will not hatch in the absence ofhost root exudates.

Some fungal pathogens infect their host, enter into a dormant state andresume activity at a later stage of host development. For example, the loose smutfungus of wheat (*Ustilago tritici*) infects developing wheat embryos duringflowering. Within a week of flowering, the embryo becomes resistant to infection.

The fungus survives as dormant mycelium within the embryo of the seed. Wheninfected seed germinates, the pathogen is activated. As plants approach maturity,the inflorescence is replaced by masses of smut teliospores. Some of the ripe-rotsor postharvest rots of fruits are caused by fungi that infect the young developingfruit and then enter into a quiescent or latent state. They later resume activitywhen fruits commence to ripen. The black spot fungus of citrus, *Guignardiacttricarpa*, is an example of such a fungus. Ascospores produced in pseudotheciaon leaves that fell to the ground 6 weeks to 6 months previously infect veryimmature citrus fruits (between fruit set and 4 months development). From theappressorium, an infection peg penetrates the cuticle and forms a small mass ofsubcuticular (between the cuticle and epidermis) mycelium. The fungus thenremains in a quiescent state for about six months until the fruits are fully grownand mature. It resumes growth and grows into the rind tissue to produce blackspot symptoms. Thus, the disease cycle involves a saprophytic state in fallenleaves and a resting state in immature fruits.

**A. Survival assaprophytes**

The ability to live saprophytically enables many plant pathogens to survive in the absence of growing susceptible plants. Saprophytic survival usually occurs in the soil. Waksman (1971) distinguished between soil inhabitants soil invaders; the former comprise the basic fungal flora of the soil, whereas the latter are short-lived exotics. As applied to the root infecting fungi soil inhabitants are unspecialized parasites with a wide host range that are able to survive-indefinitely in the soil as saprophytes; soil invaders (root inhabiting fungi) are more specialized parasites that survive in soils inclose association with their hosts. Most plant pathogenic fungi and bacteria are soil invaders, but some pathogens, notably *Rhizoctonia solani* and *Pythium debaryanum* that cause seedling blights and root rots, live saprophytically in thesoils.

**B. Survival by means of specialized restingstructures**

Enduring structures of plant pathogens may be as simple as conidia or as complex as perithecia. Apparently, ascospores or conidia derived from them, serve to carry the pathogen causing peach- leaf curl (*Taphrina deformans*) over the winter. Conidia of *Alternaria solani*, the pathogen of early blight of potato and tomato, survive for eighteen months in dried diseased leaves. Specialized thick-walled chlamydospores of *Fusarium* and other Imperfect fungi, spores of many smut fungi and the amphiospores, uredospores and teliospores of certain rust fungi also are important enduring structures. The resting spores of *Plasmodiophora brassicae* may survive for ten years in soils infested upon the disintegration of clubbed roots.

**C.Survival in association with Insect, nematodes andfungi**

Many insects are carriers of inocula during the growing season and several important plant pathogens survive between growing seasons within insects. Some bacterial plant pathogens may survive within the insect body and over winter therein. The com flea beetle, *Chaetocnema pulicaria* Melsh carries inside its body, the com wilt pathogen, *Xanthomonas stewartii* and thus helps in its overwintering. The cucumber beetles, *Diabrotica vitata* Fabr. and *D. duodecimpunctata* Oliv., which chew the plant parts affected by *Erwiniatra cheiphila* carry the pathogen inside their body, where it over winters.

Plant viruses like wheat mosaic, wheat spindle streak virus, lettuce big vein, tobacco necrosis, tobacco rattle and tobacco ring spot viruses survive with nematodes or fungi found in the soil between crop seasons. Tobacco ring spot virus is associated with the nematode, *Xiphinema americanum*. The fungus, *Polymyxa graminis* (Barley yellow mosaic, oat yellow mosaic, wheat soil-borne mosaic, wheat spindle-streak mosaic) and *Spongospora subterranea* (potato mop top) carry the viruses internally and transmit.them through their resting spore.

**Transmission of Plant Diseases:**

Rational and most effective control of parasitic plant diseases is possible only if –

1. the disease is correctly diagnosed,
2. the nature of transmission of the disease is known and
3. life cycle stages of the involved parasite i.e its mode of reproduction active structures produced under the favourable condition for repaid and wide dispersal and the structures produced to overcome adverse condition are known.

The detail study of life cycles of different parasites being beyond the scope of this article it would be quite sufficient to understand and remember.one of the general modes of reproduction of different plant parasites viz.fungi, bacteria, viruses (not true parasites) and nematodes and the structures which enable these parasites to survive under most unfavourable condition like the off season of suitable host crop(s) and / or exceptional high temperature and dry weather conditions prevailing in the summer months in a tropical country like India.

All parasitic as well as viral diseases are transmissible, the parasites or issues being infectious to suitable host plants with ability to spread from host to host and from one area to another. The microscopic parasites or sub-microscopic infectious agents viz. Viruses causing plant diseases are technically termed as ‘pathogens’.

Wherever a disease is established in a particular area or country, transmission of the pathogen from host to host or from one place to another is termed as 'dissemination' or 'dispersal' of the pathogen. Dissemination of plant diseases is recognized in relation to different phases of diseases as under:

1. **Primary infection:** Contact of a pathogen with a suitable host plant and initiation of the disease first time in the season of a crop is called 'primary infection'. Often a few or several plants in the crop are likely to get primarily infected.
2. **Secondary spread**: When a plant or few plants are primarily infected, rapid multiplication of the pathogen sets in under favorable climatic conditions, which helps 'secondary spread' of the disease.

In case of fungal diseases the primary infections are usually caused by the resting or dormant structures of the fungal pathogen while the secondary spread occurs through the asexually produced, countless and short-lived spores

Although in majority of plant diseases the above two phases occur sequentially, primary infections occurring only once in the season and secondary spread often repeating several times in the same season thus causing rapid. Transmission over wide areas, this type of cycle is not observed in many other diseases. In such cases there is no secondary spread in the same season i.e. the pathogen multiplies only once during the crop period and the spread of disease is observed only during the following crop season by way of increased primary infection. Mildews, leafspots, blasts, blights etc are the common examples of former type while some smut disease of cereal or grain crops where black powder is formed in place of grains or inflorescence fall in the latter type. Control measures, therefore, in the former type need to be directed to avoid the primary infection and also to check the secondary spread.

**Modes of primary infections:**

According to the general mode of primary infection plant diseases are recognized as:-

1. Soil borne
2. Seed borne, including diseases carried with planting material.
3. Wind borne
4. Insect borne etc.

This knowledge is helpful in adopting suitable control measures.

**Continuous and discontinuous transmission: -**

Transmission of disease is termed as 'continuous' when it occurs naturally by way of growth, multiplication and spread of the pathogen in an area or country where the disease is established. At times, however, in an area or country where a particular disease has never occurred, it may get

introduced through the agency of man carrying diseased material to a new locality or to a distant country for the purpose of introduction of new plants, crops, varieties etc. such transmission, of course, is unnatural and regarded as 'discontinuous' transmission. Altogether different control measures viz. Quarantine Regulations are enforced to check such discontinuous transmission.

**Direct and indirect transmission:-**

For classifying the methods of disease transmission in relation to the methods of suitable control measures, the following two groups can be conveniently recognized.

**1. Direct transmission:** - Disease transmission where the pathogen is carried externally or internally on the seed or planting material like cuttings, sets, tubers, bulbs etc.

**2. Indirect transmission:-** The pathogen spreading itself by way of its persistent growth or certain structures of the pathogen carried independently by natural agencies like wind, water, animals, insects, mites, nematodes, birds etc. are the different methods of indirect transmissions.

**Direct transmission:** -

**1. Internal transmission through seed or planting material:-** False smut disease as well as Helminthosporin Blight disease of wheat are the common examples of fungal diseases carried internally through apparently healthy seed. Ring rot and Brown rot of potato caused by bacteria are carried internally through the tubers. The well known whip smut and red rot of sugarcane are fungal diseases carried internally in the planting sets. Mosaic and leaf roll of potato which are viral diseases are also carried inside the infected tubers.

**2. External transmission through seed or planting material:-** In this mode of transmission the pathogen is carried externally over the surface of seed or vegetatively propagated plant parts like sets, tubers, bulbs etc. or may even be carried as a physical mixture of fungal structures with the seed. The common grain smut of jowar is an example of the former type while the fungal structures called 'sclerotia' having the size of a grain or slightly bigger in case of the Ergot disease of bajra are often likely to be transmitted in the form of physical mixture with the seed.

**Indirect transmission:** -

**1. Autonomous transmission:-** It takes place by continuous and persistent growth of the threads or 'hyphae' of the causal fungi in soil, characteristic of several wood rotting fungi attacking forest trees and some fruit plants. Some root rotting fungi infecting certain seasonal crops also are transmitted by this method. The autonomous dispersal of such soil fungi may range from few cm. To several (8 to 10) meters in a single season. Some plant parasitic nematodes also exhibit active but limited mobility in the soil.

**2. Wind dispersal: -** Fungal spores produced externally on host surfaces are most easily carried by wind currents and this is the most dangerous mode of transmission of plant pathogenic fungi like those causing powdery and downy mildews, leaf spots, blasts, blights and rust diseases. The black stem rust disease of wheat in India perpetuates on wild grasses in the Nilgiri hills in the south India from where the rust spores are carried to south, central & then to north India by wind currents every year. Spores may be carried from low to very high altitudes of 12,000 to 14,000 feet and from short distances to very long distances of several hundred kilometers.

Extensive and severe epidemics of plant diseases are mostly the results of wind transmission of the pathogens. Wind dissemination involves four stages relating to the spores viz. Production of countless spores, their liberation in the wind currents, dispersal alongwith the wind and deposition on new susceptible host surfaces where they cause infection under favourable climatic conditions. Apart from spores, bits of fungal threads and nematode cysts are also amenable to wind transmission in certain cases.

**3. Water dissemination:** Disease transmission through the agency of water in different ways is comparatively less important as compared to the wind transmission. Splashing rain drops mostly transmit the foliar diseases from leaf to leaf, from shoot to shoot and even from plant to plant in case of closely spaced crops. Such transmission is usually accompanied by wind dispersal as well. Plant pathogens requiring high humidity conditions like the fungi causing downy mildew diseases or bacteria causing canker of citrus are well adapted to this kind of short distance water dispersal.

Certain soil inhabiting pathogenic fungi and bacteria causing root and collar rots, wilts, foot, rots, etc are likely to be transmitted to much longer distances through the agencies like irrigation water, streams and rivers, etc. It is also an important agency in transmission of seeds of higher flowering parasites like dodder and striga.

**4. Animals:** Farm animals serve as disease transmitting agents in some cases. They are likely to carry the pathogen externally on their body surface, particularly on legs and hoofs, etc. or internally through their intestinal tract. Commonly, the soil inhabiting fungi causing rots and wilts are carried externally while certain smut fungi causing diseases to grain crops are transmitted through the intestinal tract.

**5. Birds:** Although birds play a very minor role in disease transmission, in cases of dispersal of seeds of higher flowering parasite. Loranthus sp. Parasitising certain trees like mango, etc. their role is of great significance. They transmit loranthus both externally and internally.

**6. Implements and Tools:** Farm implements used for cultivation of soil are often likely to transmit plant pathogens from one place to another. The pathogens in this case are usually carried in the form of bits of plant disease debris lying in the soil. Similarly tools used for carrying out operations like cutting, pruning, budding, grafting, thinning,etc. also help in the transmission of certain diseases from plant to plant. Several viral diseases are disseminated through the budding and grafting operations.

**7. Insects:** Most of the viral diseases of plants are transmitted through the agency of different insects. Both types of insects viz. sucking and chewing or/biting are capable of transmitting viral diseases. The transmission may be simply `mechanical' or it may be `biological'. In the latter case the specific insect and the specific viral pathogen have some kind of association or relationship between the two. Insects in such cases are called the `vectors' for the particular viral pathogen. In case of mechanical transmission the pathogen is simply carried externally or internally by the insect. Viruses carried `biologically' by the insect vectors are of two types:

**1.** Non-persistent-viral pathogen requiring no latent or incubation period in the insect body.

**2.** Persistent: viral pathogens requiring certain incubation period inside the vector body before they are inoculated or transmitted to healthy host.The insects responsible for transmission of viral diseases belong to the species of aphids, jassids (leaf hoppers), white flies, mealy bugs, etc. Certain bacterial and several fungal pathogens are also known to be carried by insects.

**1. Mites:** Mites in contrast to insects are wingless anthropods resembling ticks and having four pairs of legs and no antennae. It is suspected that some viral diseases of chillies, tomato, brinjal, etc. have vector relationship with mites.

**2. Nematodes:** Nematodes have been observed to transmit viral, bacterial and fungal plant diseases. Nematodes feeding externally on host plant roots cause injuries to roots which become the avenues for entrance of fungal and bacterial pathogens infecting plant roots. The Fan-leaf virus of grapevine is a well known example of transmission through a species of nematodes.

**3. Biological transmission**: Dodder which is higher flowering parasite is known to transmit certain viral diseases which remain `persistent' in the dodder plant. The flowering parasite after acquiring the virus from infected plant does not show any symptom itself but remains capable of transmitting the virus to healthy hosts.

**4. Human dispersal**: Man is often responsible for transmission of plant diseases in two ways viz.

**1.** Workers handling seedlings, other planting material or fruits are likely to get personally in contact with plant pathogens like fungi or bacteria. While handling the diseased material and

unknowingly and indirectly transmit the pathogens to healthy seedlings or plant parts through his contaminated hands. This is a kind of `continuous' mode of transmission.

**2.** The other or`discontinous' mode of transmission for which only man is responsible is the most efficient and equally dangerous phenomenon of transmission of plant diseases between distant geographical areas often separated by physical barriers like oceans, mountains or deserts, etc. Such long distances transmission of a disease to an area or country hitherto free from the disease is usually accomplished by the transport of infected seed, nursery stock or timber, etc. Thus it is a kind of direct transmission through propagating material.

In the history of plant pathology this mode of transmission has often resulted in to some of the worst plant disease epidemics in new areas, the local host plant stock being not adapted to the pathogen and consequently remaining highly susceptible. Fungal diseases viz. Late blight of potato and downy mildew of grapes, Bunchy top of banana, which is a viral disease and Bacterial blight of paddy are the examples of some severe and important diseases introducing in India from other countries.

Practically all the countries of the world have suffered from such introduction of new plant diseases through the agency of man. Concerned international authorities, therefore, are engaged in promoting studies in this respect and designing regulations to reduce the danger.

**Disease Epidemics:**

When a pathogen spreads to and affects many individuals within a population over a relatively large area and within a relatively short time, the phenomenon is called an epidemic. An epidemic has been defined as any increase of disease in a population.

A similar definition of an epidemic is the dynamics of change in plant disease in time and space. The study of epidemics and of the factors that influence them is called epidemiology. Epidemiology is concerned simultaneously with populations of pathogens and host plants as they occur in an evolving environment, i.e., the classic disease triangle. As a result, epidemiology is also concerned with population genetics of host resistance and with the evolutionary potential of pathogen populations to produce pathogen races that may be more virulent to host varieties or more resistant to pesticides. Epidemiology, however, must also take into account other biotic and abiotic factors, such as an environment strongly influenced by human activity, particularly as it relates to disease management.

Plant disease epidemics, sometimes called epiphytotics, occur annually on most crops in many parts of theworld. Most epidemics are more or less localized and cause minor to moderate losses. Some epidemics arekept in check naturally, e.g., by changes in weather conditions. Others are kept in

check by chemical spraysand other control measures. Occasionally, however, some epidemics appear suddenly, go out of control, andbecome extremely widespread or severe on a particular plant species. Some plant disease epidemics, e.g., wheat rusts, southern corn leaf blight (Fig. 8-1), and grape downy mildew, have caused tremendous losses ofproduce over rather large areas. Others, e.g., chestnut blight (Fig. 1-8), Dutch elm disease, and coffee rust,have threatened to eliminate certain plant species from entire continents. Still others have caused untold suffering to humans. *The Irish potato famine of 1845–1846 was caused by the Phytophthora late blight epidemic of potato, and the Bengal famine of 1943 was caused by the Cochliobolus (Helminthosporium) brown spot epidemic of rice.*Epiphytotic, rate of increase of disease is mathematically analogous to compound interest in money eg rusts of cereals, powdery mildews

**The elements of an epidemic:**

Plant disease epidemics develop as a result of the timelycombination of the same elements that result in plantdisease: susceptible host plants, a virulent pathogen, andfavorable environmental conditions over a relativelylong period of time. Humans may unwittingly help initiateand develop epidemics through some of their activities,e.g., by topping or pruning plants in wet weather.

More frequently, humans may stop the initiation anddevelopment of epidemics by using appropriate controlmeasures under situations in which epidemics wouldalmost certainly occur without human intervention.Thus, the chance of an epidemic increases when the susceptibilityof the host and virulence of the pathogen aregreater, as the environmental conditions approach theoptimum level for pathogen growth, reproduction, andspread, and as the duration of all favorable combinationsis prolonged or repeated.

The interactionof the four components can be visualized as a tetrahedron,or pyramid, in which each plane represents one ofthe components. This figure is referred to as the diseasetetrahedron or disease pyramid (Fig. 8-2). The effect oftime on disease development becomes apparent whenone considers the importance of the time of year (i.e.,the climatic conditions and stage of growth when hostand pathogen may coexist), the duration and frequencyof favorable temperature and rains, the time of appearanceof the vector, the duration of the infection cycle ofa particular disease, and so on.

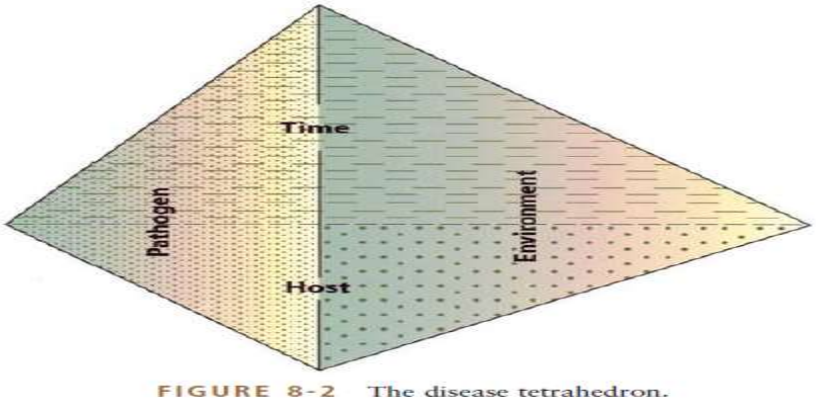



FIGURE 8-2 The disease tetrahedron.

**Host factors that affect the development of epidemics:**

Several internal and external factors of particular host plants play an important role in the development of epidemics involving those hosts.

**i) Levels of Genetic Resistance or Susceptibility of the Host**

Obviously, host plants carrying race-specific (vertical) resistance do not allow a pathogen to become established in them, and thus no epidemic can develop. Host plants carrying partial (horizontal) resistance will probably become infected, but the rate at which the disease and the epidemic will develop dependson the level of resistance and the environmental conditions.

Susceptible host plants lacking genes for resistance against the pathogen provide the ideal substrate forestablishment and development of new infections. Therefore, in the presence of a virulent pathogen anda favorable environment, susceptible hosts favor the development of disease epidemics.

**ii) Degree of Genetic Uniformity of Host Plants**

When genetically uniform host plants, particularly withregard to the genes associated with diseaseresistance,are grown over large areas, a greater likelihood existsthat a new pathogen race will appear that can attacktheir genome and result in an epidemic. This phenomenonhas been observed repeatedly, for example, in the*Cochliobolus* (*Helminthosporium*) blight on Victoriaoats and in southern corn leaf blight oncorn carrying Texas male-sterile cytoplasm. For similarreasons of genetic uniformity, the highest rates ofepidemic development generally occur in vegetativelypropagated crops, intermediate rates in self-pollinatedcrops, and the lowest rates in cross-pollinated crops.This explains why most epidemics develop rather slowlyin natural populations, where plants of varying geneticmakeup are intermingled.

**iii) Type of Crop**

In diseases of annual crops, such as corn, vegetables,rice, and cotton, and in foliar, blossom, or fruit diseasesof trees and vines, epidemics generally develop muchmore rapidly (usually in a few weeks) than they do indiseases of branches and stems of perennial woody cropssuch as fruit and forest trees. Some epidemics of fruitand forest trees, e.g., tristeza in citrus, pear decline,Dutch elm disease, and chestnut blight, take years todevelop.

**iv) Age of Host Plants**

Plants change in their reaction (susceptibility or resistance)to disease with age. The change of resistancewith age is known as ontogenic resistance. In someplant–pathogen combinations, e.g., *Pythium* dampingoff and root rots, downy mildews, peach leaf curl, systemicsmuts, rusts, bacterial blights, and viral infections,the hosts (or their parts) are susceptible only during thegrowth period and become resistant during the adultperiod (adult resistance). Withseveral diseases, such as rusts and viral infections, plantparts are actually quite resistant to infection while stillvery young, become more susceptible later in theirgrowth, and then become resistant again before they arefully expanded.

**Pathogen factors that affect development of epidemics:**

**i) Levels of Virulence**

Virulent pathogens capable of infecting the host rapidlyensure a faster production of larger amounts of inoculum,and, thereby, disease, than pathogens of lesservirulence.

**ii) Quantity of Inoculum near Hosts**

The greater the number of pathogen propagules (bacteria,fungal spores and sclerotia, nematode eggs, virusinfectedplants, etc.) within or near fields of host plants,the more inoculum reaches the hosts and at an earliertime, thereby increasing the chances of an epidemicgreatly.

**iii) Type of Reproduction of the Pathogen**

All pathogens produce many offspring. Some of them, such as most fungi, bacteria, and viruses, produce agreat many offspring, while a few fungi, all nematodes, and all parasitic plants produce relatively small numbers of offspring. Some plant pathogenic fungi, bacteria, and viruses have short reproduction cycles and therefore are polycyclic, i.e., they can produce many generations in a single growing season. Polycyclic pathogens include fungi that cause rusts, mildews, and leaf spots and are responsible for most of the sudden, catastrophic plant disease epidemics in the world. Some soil fungi, such as *Fusarium* and *Verticillium*, and most nematodes usually have one to a few (up to four) reproductive cycles per growing season. For these latter pathogens, the smaller number of offspring and, especially, the conditions of their dispersal limit their potential to cause sudden and widespread epidemics in a single season.

**iv) Ecology of the Pathogen**

Some pathogens, such as most fungi and all parasitic higher plants, produce their inoculum (spores and seeds, respectively) on the surface of the aerial parts of the host. From there, spores and seeds can be dispersed with ease over a range of distances and can cause wide spread epidemics. Other pathogens, such as vascular fungi and bacteria, mollicutes, viruses, and protozoa, reproduce inside

the plant. In this case, spread of the pathogen is rare or impossible without the help of vectors. Therefore, such pathogens can cause epidemics only when vectors are plentiful and active.

**v) Mode of Spread of the Pathogen**

The spores of many plant pathogenic fungi, such as those causing rusts, mildews, and leaf spots, are releasedinto the air and can be dispersed by air breezes or strong winds over distances varying from a few centimeters up to several kilometers. These kinds of fungi are responsible for the most frequent and most widespread epidemics.

In terms of their ability to cause sudden and widespread epidemics, the next most important group of pathogens includes those whose inoculum is carried by airborne vectors. Many of the viruses are transmittedby aphids, whiteflies, and some other insects. Mollicutes and fastidious bacteria are transmitted by leafhoppers, plant hoppers, or psyllids. Some fungi (such as the cause of Dutch elm disease), bacteria (such as the cause of bacterial wilt of cucurbits), and even nematodes (such as the cause of pine wilt disease) are disseminated primarily by beetles. Pathogens that are transmitted by windblown rain (primarily fungi causing diseases such as anthracnoses and apple scab, and most bacteria) are almost annually responsible for severe but somewhat localized epidemics within a field, a country, or a valley. Pathogens carried with the seed or other vegetative propagative organs (such as tubers or bulbs) are often placed in the midst of susceptible plants, but their ability to cause epidemics depends on the effectiveness of their subsequent transmission to new plants.

**Environmental factors that affect development of epidemics:**

The majority of plant diseases occur wherever the host is grown but, usually, do not develop into severe andwidespread epidemics. The concurrent presence in the same areas of susceptible plants and virulent pathogens does not always guarantee numerous infections, much less the development of an epidemic. This fact dramatizes the controlling influence of the environment on the development of epidemics. The environment may affect the availability, growth stage, succulence, and genetic susceptibility of the host plants. It may also affect the survival, vigor, rate of multiplication, sporulation, and ease, direction, and distance of dispersal of the pathogen, as well as the rate of spore germination and penetration.

**i) Moisture**

Abundant, prolonged, orrepeated high moisture, whether in the form of rain, dew,or high humidity, is the dominant factor in the developmentof most epidemics of diseases caused by oomycetesand fungi (blights, downy mildews, leaf spots, rusts, andanthracnoses), bacteria (leaf spots, blights, soft rots), andnematodes. Moisture not only promotes new succulentand susceptible growth in the host, but,

more importantly,it increases sporulation of fungi and multiplication of bacteria. Moisture facilitatesspore release by many fungi and theoozing of bacteria to the host surface, and it enablesspores to germinate and zoospores, bacteria, and nematodesto move. The presence of high levels of moistureallows all these events to take place constantly andrepeatedly and leads to epidemics.

**ii) Temperature**

Epidemics are sometimes favored by temperatureshigher or lower than the optimum for the plant becausethey reduce the plant's level of partial resistance. Atcertain levels, temperatures may even reduce or eliminatethe race-specific resistance of host plants. Plantsgrowing at such temperatures become "stressed" andpredisposed to disease, provided the pathogen remainsvigorous.

Low temperature reduces the amount of inoculumof oomycete fungi, bacteria, and nematodes that survivescold winters. High temperature reduces the inoculumof viruses and mollicutes that survives hot summertemperatures. In addition, low temperatures reduce thenumber of vectors that survive the winter. Low temperaturesoccurring during the growing season can reducethe activity of vectors.

The most common effect of temperature on epidemics,however, is its effect on the pathogen during thedifferent stages of pathogenesis, i.e., spore germinationor egg hatching, host penetration,pathogen growth or reproduction,invasion of the host, and sporulation. Whentemperature stays within a favorable range for each ofthese stages, a polycyclic pathogen can complete itsinfection cycle within a very short time (usually in a fewdays). As a result, polycyclic pathogens can producemany infection cycles within a growing season. Becausethe amount of inoculum is multiplied manyfold (perhaps100 times or more) with each infection cycle and becausesome of the new inoculum is likely to spread tonew plants, more infection cycles result in more plantsbecoming infected by more and more pathogens, thusleading to the development of a severe epidemic.

In reality, moisture and temperature must be favourable and act together in the initiation and developmentof the vast majority of plant diseases and plant diseaseepidemics.

**Effect of human cultural practices and control measures:**

Many activities of humans have a direct or indirecteffect on plant disease epidemics, some of them favouring and some reducing the frequency and the rate ofepidemics.

**i) Site Selection and Preparation**

Low-lying and poorly drained and aerated fields, especiallyif near other infected fields, tend to favor theappearance and development of epidemics.

**ii) Selection of Propagative Material**

The use of seed, nursery stock, and other propagativematerial that carries various pathogens increases theamount of initial inoculum within the crop and favorsthe development of epidemics greatly. The use ofpathogen-free or treated propagative material canreduce the chance of epidemics greatly.

**iii) Cultural Practices**

Continuous monoculture, large acreages planted to thesame variety of crop, high levels of nitrogen fertilization,no-till culture, dense plantings, overheadirrigation, injury by herbicide application, and poorsanitation all increase the possibility and severity ofepidemics.

**iv) Disease Control Measures**

Chemical sprays, cultural practices (such as sanitationand crop rotation), biological controls (such as using resistant varieties), and other control measures reduce or eliminate the possibility of an epidemic. Sometimes, however, certain controls, e.g., the use of a certain chemical or planting of a certain variety, may lead to selection of virulent strains of the pathogen that either are resistant to the chemical or can overcome the resistance of the variety and thus lead to epidemics.

**Patterns of epidemics:**

Interactions of the structural elements of epidemics, as influenced over time by factors of the environmentand by human interference, are expressed in patterns and rates. The pattern of an epidemic in terms of thenumbers of lesions, the amount of diseased tissue, or thenumbers of diseased plants is given by a curve, calledthe **disease–progress curve**, that shows the progress ofthe epidemic over time. The point of origin and theshape of a disease–progress curve reveal informationabout the time of appearance and amount of inoculum,changes in host susceptibility during the growing period,recurrent weather events, and the effectiveness of culturaland control measures.

Disease–progress curves,because they are affected by weather, variety, and so on,vary somewhat with location and time, but they are generallycharacteristic for some groups of diseases. Forexample, a saturation-type curve is characteristic formonocyclic diseases, a sigmoid curve is characteristicfor polycyclic diseases, and a bimodal curve is characteristicfor diseases affecting different organs(blossoms, fruit) of the plant. Knowledge ofdisease–progress curves also allows disease forecastingand selection of the best control strategy for the particulardisease and time.

**Comparison of epidemics:**

For better comparison of epidemics of the same diseaseat different times, different locations, or under differentmanagement practices or to compare different diseases,the patterns obtained for disease–progress curvesand disease–gradient curves are frequently transformedmathematically into straight lines. The slopes of theselines can then be used to calculate epidemic rates.

**In monocyclic diseases**, the amount of inoculum doesnot increase significantly during the season. In such diseases,therefore, the rate of disease increase is affectedonly by the inherent ability of the pathogen to inducedisease and by the ability of the environmental factorsand cultural practices to influence host resistance andthe virulence of the pathogen.

In contrast, the initial inoculum for diseases causedby polycyclic pathogens, although extremely important,has relatively less importance than the number of infectioncycles in the final disease outcome.Pathogens that have many infection cycles also havenumerous opportunities to interact with the host. Therefore,the same factors mentioned earlier, namely theinherent ability of the pathogen to induce disease, environmentalfactors, host resistance, and cultural practices,have an opportunity to influence the dispersal,penetration, multiplication, size of lesion, rate of lesionformation, and rate and amount of sporulation, but theycan do that not once but several times during the samegrowth season. The continuous or, sometimes, intermit-tent increase of the amount of inoculum and disease mayresult in highly variable infection rates for individualshort-term intervals during the growth season, and quitevariable epidemic rates for the entire season.

In addition to the epidemics caused by monocyclicand polycyclic pathogens, there are also polyeticepidemics. Pathogens causing polyetic epidemics arepresent for one year or more in the infected plant beforethey produce effective inoculum

**Development of epidemics:**

For a disease to become significant in a field, particularlyif it is to spread over a large area and develop intoa severe epidemic, specific combinations of environmentalfactors must occur either constantly or repeatedly,and at frequent intervals, over a large area. Even in asingle, small field that contains the pathogen, plantsalmost never become severely diseased from just one setof favorable environmental conditions. It takes repeatedinfection cycles and considerable time before a pathogenproduces enough individuals to cause an economicallysevere epidemic in the field. Once large populationsof the pathogen are available, however, they canattack, spread to nearby fields, and cause a severe epidemicin a very short time, often in just a few days.A plant disease epidemic can occur in a garden, agreenhouse, or a small field, but “epidemic” generallyimplies the development and rapid spread of a pathogenon a particular kind of crop plant cultivated over a largearea, such as a

large field, a valley, a section of a country,the entire country, or even part of a continent. Therefore, the first component of a plantdisease epidemic is a large area planted to a geneticallyuniform crop plant, with the plants and the fields beingclose together. The second component of an epidemicis the presence or appearance of a virulent pathogen.

Such cohabitations of host plants and pathogens occur,of course, daily in countless locations. Most of these,however, cause local diseases of varying severity, destroycrop plants to a limited extent, and do not develop intoepidemics. Epidemics develop only when the combinationsand progression of the right sets of conditionsoccur. These include appropriate temperature, moisture,and wind or insect vector coinciding with the susceptiblestage or stages of the plant and with the production, spread, inoculation, penetration, infection, and reproductionof the pathogen.

## 9. Principles of plant disease control: exclusion, eradication, protection and therapy

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

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

although it is often difficult to distinguish between the two concepts, especially in the application of specific measures.

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

**Exclusion:**

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

In the United States, the Animal and Plant Health Inspection Service (**APHIS**), a division of the U.S. Department of Agriculture, is responsible for promulgating and enforcing plant quarantine measures. There are also state agencies that deal with local quarantines. Internationally, eight regional plant protection organizations (**PPOs**) were established in 1951 by the International Plant Protection Convention sponsored by the Food and Agricultural Organization of the United Nations. This was revised in 1997 and now includes nine regional PPOs. The European and Mediterranean Plant

Protection Organization (EPPO) is the oldest of the regional PPOs. The regional PPOs have no regulatory authority such as APHIS or other governmental agency, but function to develop strategies against the introduction and spread of pests and to coordinate the use of phytosanitary regulations to ensure agreement among the different member countries.

An important and practical strategy for excluding pathogens is to produce pathogen-free seed or planting stock through certification programs for seeds and vegetatively propagated plant materials such as potatoes, grapes, tree fruits, etc. These programs utilize technologies that include isolation of production areas, field inspections, and removal of suspect plants to produce and maintain pathogen-free stocks. Planting stock that is freed of pathogens can be increased by tissue culture and micropropagation techniques as well as be maintained in protective enclosures such as green houses to exclude pathogens and their vectors. Exclusion may be accomplished by something as simple as cleaning farming equipment (Figure 2) to remove contaminated debris and soil that can harbor pathogens such as *Verticillium*, nematodes or other soil borne organisms and prevent their introduction into non-infested fields.

**Eradication:**

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

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

Eradication can also be on a more modest scale such as the removal of apple or pear branches infected by the fire blight bacterium (*Erwinia amylovora*) or pruning to remove blister rust cankers (caused by *Cronartium ribicola*) on white pine branches. Or, it can be the sorting and removal of diseased flower bulbs, corms or rhizomes. Hot water seed-treatment of cereal seeds to kill smut

mycelium in the seed and heat treatment to eliminate viruses from fruit tree budwood for grafting are other examples of pathogen eradication.

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

Eradication may also be accomplished by destroying weeds that are reservoirs of various pathogens or their insect vectors. Elimination of potato cull piles is an effective method of eradicating overwintering inoculum of the late blight pathogen.

Soil fumigation has been a widely used eradication strategy. This technology involves introducing gas-forming chemicals such as carbon disulfide, methyl bromide, or chloropicrin into soil to kill target pathogens. However, undesirable side effects such as killing beneficial organisms, contamination of groundwater, and toxicity of these chemicals have resulted in less reliance on this approach for disease management. Volatile fumigants like methyl bromide are injected into soil and sealed with a plastic film. Some water-soluble fumigants like metam-sodium can be injected into the soil and the soil simply compacted to form a seal.

Crop rotation is a frequently used strategy to reduce the quantity of a pathogen, usually soil-borne organisms, in a cropping area. Take-all of wheat (caused by *Gaeumannomyces graminis*) and soybean cyst nematode (*Heterodera glycines*) are two examples of soilborne diseases that are easily managed by short rotations of 1 and 2 years, respectively, out of susceptible crops, which may include susceptible weed hosts such as grasses in the case of take-all.

Burning is an effective means of eradicating pathogens and is often required by law to dispose of diseased elm trees affected by Dutch elm disease (DED) citrus trees infected by citrus canker or of bean fields infected by halo blight bacteria (*Pseudomonas syringae* pv. *phaseolicola*). Propane flaming can effectively destroy *Verticillium* microsclerotia in mint stems, and flaming potato stems prior to harvest may prevent tuber infection by the late blight pathogen. However, burning

agricultural fields is controversial because the smoke creates human health and safety and environmental concerns.

**Protection:**

This principle depends on establishing a barrier between the pathogen and the host plant or the susceptible part of the host plant. It is usually thought of as a chemical barrier, e.g., a fungicide, bactericide or nematicide, but it can also be a physical, spatial, or temporal barrier. The specific strategies employed assume that pathogens are present and that infection will occur without the intervention of protective measures. For example, bananas are covered with plastic sleeves as soon as the fruit are set to protect the fruit from various pests including fruit decay fungi.

Protection often involves some cultural practice that modifies the environment, such as tillage, drainage, irrigation, or altering soil pH. It may also involve changing date or depth of seeding, plant spacing, pruning and thinning, or other practices that allow plants to escape infection or reduce severity of disease. Raising planting beds to assure good soil water drainage is an example of cultural management of plant diseases such as root and stem rots.

Fungicides have been used for more than a hundred years and new fungicides continue to be developed.

**Bordeaux mixture**, a basic copper sulfate fungicide, was the first widely used fungicide and is still used today in various forms. The earliest fungicides were simple elements like sulfur or metallic compounds of copper or mercury, and these are generally classed as inorganic fungicides. In the early to mid-1900s organic fungicides such as thiram, captan, and the bisdithiocarbamates were developed. These are broad-spectrum, contact or protectant fungicides that control a wide range of fungal diseases. Starting in the 1960s the “systemic” fungicides were developed. Most of these are not truly systemic in plants but have some limited mobility, usually translaminar, and often give some post-infection benefits. Some of the “systemic” fungicides move upward in the plant’s vascular system, but currently only one (fosetyl-Al) has ambimobile distribution (both upward and downward) that would constitute a truly systemic fungicide. In addition to the SBI and DMI fungicides mentioned earlier, a recent group of systemic fungicides are the strobilurins. Some fungicides have narrow ranges of activity and are used primarily for control of specific groups of diseases such as downy mildews, rusts, smuts or powdery mildews while others are active against a wider range of diseases.

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

Fungicides can be applied by any of several methods: ground sprayers, airplanes or through irrigation systems, but to be effective applications must be done properly. First, the fungicide must be legally registered for use on the plant involved and against the target disease. Several different chemicals may be registered for the same crop or disease. If the different fungicides are similar in effectiveness, cost, ease of application, and safety, then timing of application becomes the most critical factor. If applied too early much of the chemical will be wasted before it can be effective; if applied too late, it will be largely ineffective. The benefits of properly applied fungicides can often be striking. Distribution of the spray droplets is important; the finer the spray the more complete the coverage on the plant surface. However, very small droplets form a mist that is easily displaced by wind.

Many cultural practices can be modified to manage the occurrence, intensity or severity of plant diseases. These include selection of suitable growing sites for the crop, adequate tillage to bury pathogen-infested plant residues, rotation to nonsusceptible crops, selecting pathogen-free planting stocks, orientation of plantings to improve exposure to sun and air currents, pruning and thinning to eliminate sources of infection and improve aeration in and around susceptible plants, water management on both plants and in soil, adequate nutrition, proper cultivation to improve root growth and avoid plant injury, and sanitation procedures to eliminate sources of inoculum.

Biological control involves the use of one living organism to control another, and this management technology has received much attention in recent times. However, the number of biological agents registered for use is relatively small, success has been limited, and application has been largely restricted to intensively managed, high value crops such as greenhouse plants. Two examples of effective biological control are the use of the fungus *Peniophora gigantea* to inoculate tree stumps to prevent infection of adjacent trees by the wood decay fungus *Heterobasidion annosum*, and the

application of the nonpathogenic (i.e., non-tumor-producing) bacterium*Agrobacterium radiobacter* to fruit trees before planting to prevent infection by the crown gall bacterium (*Agrobacterium tumefaciens*)

**Avoidance:**

It involves avoiding disease by planting at time when, or in areas where inoculums is absent or ineffective due to environmental conditions. The major aim is to enable the host to avoid contact with the pathogen or to ensure that the susceptible stage of the plant does not coincide with favourable conditions for the pathogen. The main practices under avoidance are choice of geographical area, selection of the field, choice of sowing/ planting time, selection of seed and planting material, short duration / disease escaping varieties and modification of agronomic/cultural practices. The potato cultivation at high altitude is relatively free from viruses; as prevailing environmental conditions do not permit the buildup of vector populations. Similarly, early planting of potato or wheat, in indo Gangetic plains may escape late blight or stem rust damage respectively.

**Resistance:**

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

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

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

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

Development of resistance has been most successful against the more specialized pathogens such as rust fungi, smut fungi, powdery mildew fungi, and viruses, but less so against general pathogens such as many blight, canker, root rot and leaf spotting pathogens.

A major problem with genetically resistant plants is that host-differentiated pathogenic races can be selected, so that many breeding programs become continuous processes to develop disease resistant plant lines. Disease resistance conferred by a single major gene is sometimes called specific or qualitative resistance and is race-specific. This type of resistance is often unstable, and emergence of a pathogenic race that can attack that genotype can completely overcome this type of resistance. Quantitative resistance or general resistance derives from many different genes for resistance with additive effects to provide more stable (or durable) resistance to pathogens.

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

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

**Immunization:**

Development by natural or artificial means of an immune or highly resistant plant population in the area infected with the pathogen to be combated

**Therapy**

It is the treatment of infected host plant, which is attempted in case of economically important horticulture plants. As a principle of plant disease control, it provides an opportunity to cure or rejuvenate the diseased host plant by use of physical or chemical agents.

Under the concept of disease management these principles have been classified into following five categories:

**1.** Management of physical environment (culturalcontrol)

**2.** Management of associated micro biota (biologicalantagonism)

**3.** Management of host genes (host resistance)

**4.** Management with chemicals (Chemicalcontrol)

**5.** Management with therapy (Physical, chemicaletc)

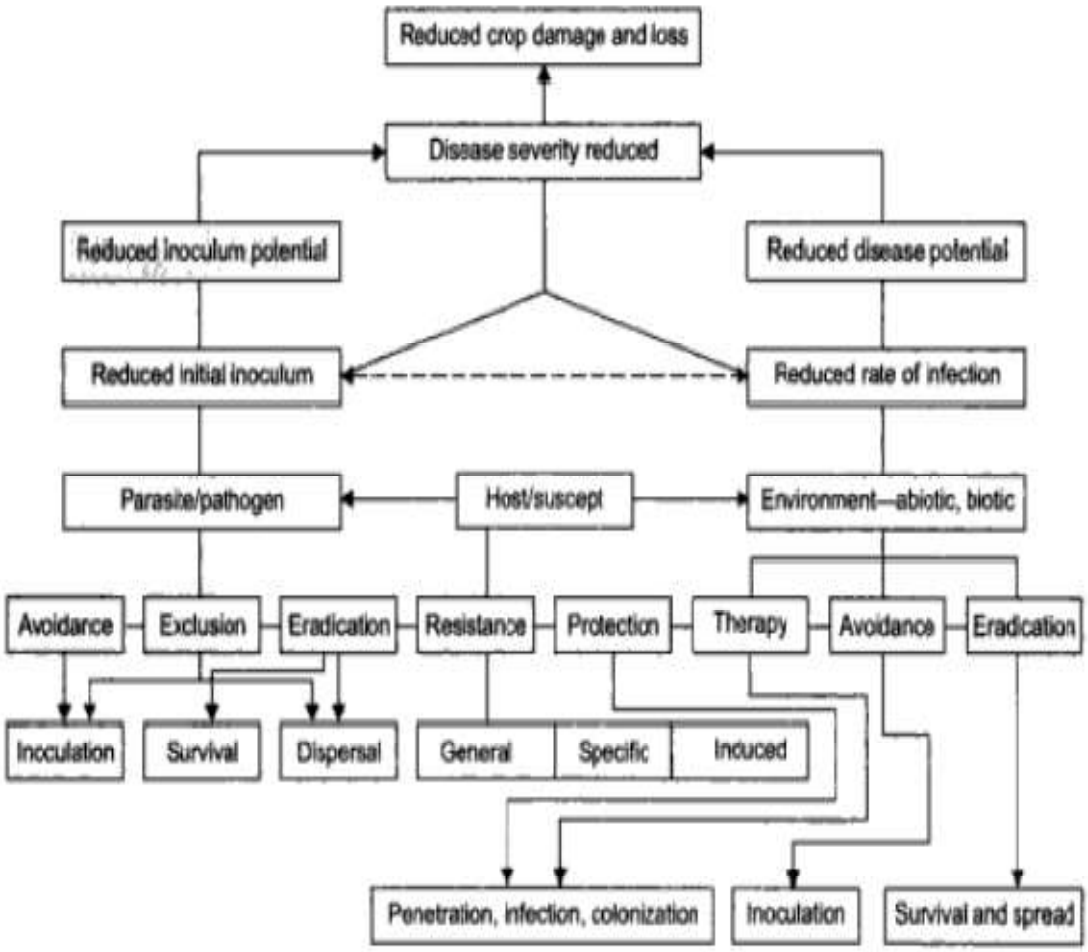
The six principles that characterize the modern concept of plant disease management should be viewed from three stand points

(a) Reduction in the initial inoculums or the rate of diseasedevelopment.

(b) Management of the pathogen population, the cure or induce defense of the suscept or modify the environment as it influences diseaseand

(c) Interruption of dispersal, survival or the course of diseasedevelopment.

These interactions are originally proposed by Baker (1968) and Roberts and Boothroyd (1972) and subsequently modified for the readers are illustrated as below:



## 10. Strategies of plant disease management with special emphasis on cultural and biological management; plant quarantine; integrated pest management

**Depending on the nature of the agents employed, the control methods are:**

**Regulatory control** measures aim at excluding a pathogen from a host or from a certain geographic area. Most **cultural control** methods aim at helping plants avoid contact with a pathogen, creating environmental conditions unfavorable to the pathogen or avoiding favorable ones, and eradicating or reducing the amount of a pathogen in a plant, a field, or an area.

Most **biological** and some **cultural control** methods aim at improving the resistance of the host or favoring microorganisms antagonistic to the pathogen. A new type of biological control involves the transfer of genetic material (DNA) into plants and the generation of transgenic plants that exhibit resistance to a certain disease(s).

**Physical** and **chemical** methods aim at protecting the plants from pathogen inoculum that has arrived, or is likely to arrive, or curing an infection that is already in progress. Some chemicals operate by activating the defenses of the plant (systemic acquired resistance) againstpathogens.

Methods of control vary considerably from one disease to another, depending on the kind of pathogen, the host, the interaction of the two, and many other variables.

**Cultural management:**

**(a) Selection of Geographical area** which on the basis of the favourable temperature and humidity requirement for a particular crop but unflavored to the fungi andbacteria.

**(b) Selection of field:**

Many soil borne diseases are controlled by proper selection of the field. It is quite possible that a particular field soil contains a pathogen species. In that case the particular crop is not sown inthat field for several years. The causal organism of Red rot of sugarcane *Colletotrichum falcatum* survives in the soil for several months. Water drainage is also taken care of while selecting the field.

**(c) Choice of the time ofsowing:**

The susceptible stage of plant growth and the favourable environment for pathogen should not match at the same time.

**(d) Disease escaping varieties:**

Certain varieties of crop due to their growth characteristics are able to escape from disease. This disease escaping characteristics of the crop is not genetic rather it is due to growth habits and time of maturation. Early maturing variety of pea is capable of escaping powdery mildew and rust.

**(e) Selection of seed:**

To avoid seed borne diseases, healthy and disease free seeds are essential.

**(f) Croprotation:**

Crop rotation is essential for controlling soil borne diseases and pathogens.

**(g) Roguing:**

Removal and Destruction of Diseased Plant Organs, eradication of alternate and collateral hosts and sanitation of Fields.

**(h) Modification of cultural Practices:**

Cultural practices such as – distance between the plants, time and frequency of irrigation, transplantation time and method, mixed cropping, amount and property of fertilizer and compost etc. can be changed to reduce losses caused by the disease.

**(i) Eradication of InsectVectors:**

Insects serve as vectors for many diseases. Eradication of such insect vectors is essential for the control of pathogens.

**Biological management:**

Use of antagonists or biocontrol agents, eg. *Trichoderma harzianum, Pseudomonas fluorescens*

Mode of action: parasitism, predation, competition, lysis, antibiosis

Control of soil-borne diseases- using suppressive soil containing antagonistic microorganisms, Post-harvest disease- using yeast, *Candida oleophila*

Control of nematodes using predaceous fungi, *Dactylella* spp.

Control of insect pest using entomogenous fungi, *Beauveria* spp. Control of aphids using trap plants

**Physical Methods:**

**(a)** The hot water treatment method of Jensen was developed in 1887 which was used to control loose smut disease of wheat, barley and Oats. Until the development of systemic fungicide hot water treatment was the only method to control loose smut. Hot water treatment is also effective in the control ofnematodes.

**(b)** Solar energy treatment to control loose smut was first developed by Lutlzra. In this method seeds are first rinsed or soaked in water for 4-5 hrs. before drying them in scorchingsun.

**(c)** Hot air treatment for the control of virus in propagating stocks was first developed by Kunkal in Peachyellow.

**Chemical Methods:**

**(a)** Seed treatment with fungicide beforetransplanting.

**i.** Soil treatingchemicals:

It is used for controlling such soil borne diseases which attack on seeds or seedlings. The examples of such chemicals are – Formaldehyde, Captan, Thiram, Zineb, Organo-mercurials, PCNB, Ethylene dibromide, vapam etc.

**ii.** For Externally seed borne diseases, chemicals such as formalin, copper carbonate, captan, organo-mercurials (Agrosan GN and Ceresan) are used for seedtreatment.

**iii.** For Internally seed borne diseases (i.e. loose smut), hot water treatment and solar treatment areused.

**iv.** Systemic Organic Compounds are effective chemicals for controlling both externally and internally seed borne diseases eg. Oxanthin derivatives (Plantvax and Vitavax), Benlate, Bavistin,Demosan.

**v.** For controlling air borne diseases, foliar application of chemicals is moreeffective.

**vi.** The common copper fungicides are: Perenox, Perelan, Blitox, Cuprokyt, Cuprosanand Fytolan. Its use is comparatively better than that of Bordeauxmixture.

**(b)** Seed dressing with organomercurials and systemicfungicides.

**Plant quarantine:**

Insects, mites, nematodes, fungi, bacteria, viruses, MLOs and other organisms are known to attack various crops of economic importance. These pests and pathogens not only reduce the quantity but also spoil the quality of the produce to a considerable extent. About crop losses due to the pests and diseases, legendary wheat pathologist, E.C. Stakman (1969) said, 'Weeds, insect-pests and plant diseases reduce the potential agricultural production in the U.S. by 23 percent. The potential food that has been aborted, spoiled or damaged would be enough to feed at least 75 million human beings. In Europe, it could feed Norway, Sweden, Finland, Denmark, Netherlands, Belgium, Czechoslovakia, Switzerland and Portugal or the four Scandinavian countries and the Great Britain. In Africa, it could feed Congo, Tanzania, Kenya, Ethiopia and Sudan'. Indeed, the magnitude of losses caused by various pests and diseases to our crops is frightening. The situation

in most of the developing world may still be worse. All out efforts are, therefore, required to atleast reduce these losses through proper management of crops and through pests and disease management.

Various methods of pest/disease control are: exclusion, eradication, protection, therapy, resistance, and biological control. Exclusion or 'keeping out' is fundamental to the concept of plant quarantine while eradication methods are employed to eliminate a newly established pest/pathogen.

**Plant quarantine** may, therefore, be defined as 'Rules and regulations promulgated by governments to regulate the introduction of plants, planting materials, plant products, soil, living organisms, etc. with a view to prevent inadvertent introduction of exotic pests, weeds and pathogens harmful to the agriculture or the environment of a country/region, and if introduced, to prevent their establishment and further spread'. Plant quarantine is thus designed as a safeguard against harmful pests/pathogens exotic to a country or a region.

**Complementary role of plant quarantine:**

Plant genetic stocks are a global resource meant for the welfare of humanity. Plant introductions could be in the form of new crops or new varieties for crop diversification, or germplasm in the form of primitive landraces or wild/weedy relatives of crop plants. Germplasm from centres of origin and crop diversity may possess valuable genes for resistance against pests/pathogens, high yield, early maturity, cold, drought or salinity tolerance and quality traits like increased oil, protein contents, etc. Plant quarantine services are charged with the responsibility of preventing entry of hazardous pests, pathogens and weeds, but to deny entry to the valuable genetic resources would be against national interest. These activities are meant to help agricultural development and they are complementary to each other. Too much conservatism on the part of plant quarantine officials and too liberal an attitude on the part of plant introduction officials/breeders would be harmful. Plant quarantine officials must strive to provide adequate safeguards to allow smooth flow of germplasm resources in a healthy state. They should also try to ensure that the germplasm, when received in quarantine station, is processed promptly and that the delays in release, if any, are purely due to biological considerations alone. At the same time, circumvention of plant quarantine must be avoided at all cost even if it means delay in release or rejection of certain materials based on biological consideration. The two should work in unison as members of a single team. Together they should decide the type, quantity and source of the material, and also the required quarantine safeguards. The plant quarantine officials should conduct research on developing sensitive and

reliable methods of detection and salvaging treatments, or find alternatives to permit introduction of even high risk genera, if introduction of such materials is in the national interest.

**Quarantine regulations:**

Plant quarantine regulations are promulgated by the national and the state governments to prevent the introduction and spread of harmful pests and pathogens. Plant quarantine will be justified only when the pest has no natural means of spread and when they are based on biological considerations only, i.e., pest/pathogen introduction risks and the available safeguards.

In general, risks are more with the introduction of vegetative propagules than with true seed. In case of true seed, risks are more with deep-seated infections than with the surface borne contamination of pests/pathogens. Again, risks are far greater with pathogens like viruses, downy mildews, smuts and many bacteria carried inside the seed without any external symptoms. When vegetative propagules are introduced, rooted plants, and other underground plant parts like rhizomes, suckers, runners, etc. carry higher risks than budwood, scions and unrooted cuttings. In any case, bulk introductions are always risky as thorough examination and treatment in such cases is very difficult and planting area is far too large to prevent the establishment and spread of the introduced pest/disease.

**Based on these factors, plant quarantine regulates the introductions as follows:**

**1.** *Complete embargo/prohibition:* When the pest risk is very high, the safeguards available in the country are not adequate and, therefore, import is prohibited.

**2.** *Post-entry quarantine:* The risk is very high but adequate safeguards in the form of post-entry isolation growing facilities are available.

**3.** *Restricted:* Pest risk is not high and import permit is required stipulating conditions for entry, inspection and treatment.

**4.** *Unrestricted:* Import permit is not required, and material may enter without restriction.

While formulating quarantine regulations, local conditions like crop spectrum and environmental conditions are also to be considered. Since quarantine regulations are designed to break the life cycle of the pest/pathogen involved, the presence of alternate or collateral hosts in the country of import and their introduction should also be taken into account.

**Plant quarantine system in India:**

Plant quarantine activities in India are carried out under the **Destructive Insects and Pests Act** (DIP Act) of 1914, and the rules and regulations framed from time to time thereunder by the Govt. of India (Anonymous, 1975). Seed was not covered under the DIP Act until 1984, when the

Govt. of India brought forward a comprehensive 'Plants, Fruits and Seeds (Regulation of Import into India) Order, 1984' which came into force in June 1985 (Anonymous, 1985).

With a view to provide the farmers the best planting materials available in the world for maximising productivity per unit area and to encourage the private seed industry in India not only to meet the internal requirements but also to develop export potential for high quality planting materials, the Government of India announced a 'New Policy on Seed Development' in September 1988. The new policy covers the import of seeds/planting materials of wheat, paddy, coarse cereals, oilseeds, pulses, vegetables, flowers, ornamentals and fruit crops; procedures for their import and the related plant quarantine procedures/requirements.

This policy also states that absolutely no compromise shall be made with the requirements of plant quarantine procedures to prevent entry into the country of exotic pests, diseases and weeds detrimental to Indian agriculture. Therefore, to meet the plant quarantine requirements in respect to the provisions of the new seed policy, the Govt. of India brought forward, through Gazette Notification,the updated Plants, Fruits and Seeds (Regulation of Import into India) Order, 1989 (Anonymous, 1989).

The main features of the existing plant quarantine regulations in India are as follows:

**1.** No consignment of seeds/planting materials shall be imported into India without a valid 'Import Permit', which is to be issued by a competent authority, to be notified by the Central Government from time to time in the Official Gazette.

**2.** No consignment of seeds/planting materials shall be imported into India unless accompained by a 'Phytosanitary Certificate', issued by the official Plant Quarantine Service of the source country.

**3.** All consignments of plants and seeds for sowing/propagation/planting purposes shall be imported into India through land customs station, seaport, airport at Amritsar, Bombay, Calcutta, Delhi and Madras, and such other entry points as may be specifically notified by the Central Government from time to time, where these shall be inspected and, if necessary, fumigated, disinfested/disinfected by authorised plant quarantine officials, before quarantine clearance.

**4.** Seeds/planting materials requiring isolation growing under detention, shall be grown in post-entry quarantine facility approved and certified by the Designated Inspection Authority (DIA) to conform to the conditions laid down by the Plant Protection Adviser to the Govt. of India.

**5.** Hay, straw or any other materials of plant origin shall not be used as packing material.

**6.** Import of soil, earth, sand, compost, and plant debris accompanying seeds/planting materials shall not be permitted. However, soil can be imported for research purposes under a special permit issued by the Plant Protection Adviser to the Govt. of India.

The DIP Act empowers the Central Government to make rules for regulating the import of seeds/planting materials into India and also the movement of the materials from one State to another within the country. The State Governments are also empowered to enact rules/regulations to regulate the movement of materials from one region/area to another within a State.

The Directorate of Plant Protection, Quarantine & Storage, headed by the Plant Protection Adviser to the Govt. of India, is primarily responsible for enforcing the quarantine rules and regulations framed under the DIP Act in the country. For this purpose, plant quarantine and fumigation stations have been established at various international airports, seaports and land custom stations where the incoming consignments are inspected, fumigated or otherwise disinfested/disinfected before release to indentors. Consignments of plants/seeds for sowing/planting/propagation purposes, however, can only be imported through Amritsar, Bombay, Calcutta, Delhi and Madras quarantine stations, where facilities in respect to well equipped laboratories, quarantine green-houses and trained scientific and technical manpower are being strengthened to adequately meet the quarantine needs. Quarantine and fumigation stations under the Directorate of Plant Protection, Quarantine & Storage handle bulk imports for commerce and for planting.

**Integrated Pest Management:**

IPM is a sustainable approach to managing pest and diseases by combining the use of all practical methods of control including biological, cultural, physical and chemical methods, in a way that minimizes economic, health and environmental risks. This type of management programme to control plant diseases is called Integrated Disease Management i.e., IDM or Integrated Pest Management i.e.,IPM.

**Procedure of Integrated Plant Disease Management:**

The procedure of management is different in respect of the principal component:

**A. Management ofHost:**

It includes:

**(a)** Procedure to improve plantvigour.

**(b)** Induction of disease resistance,and

**(c)** Breeding for diseaseresistance.

**B. Management ofPathogen:**

It includes:

**(a)** Eradication or reduction ofinoculum.

**(b)** Application of chemicals on plant surface, to discourage thepathogen.

**(c)** Prevention through legislation (i.e., keeping the pathogen away from the host) by quarantine, etc.

**C. Management ofEnvironment:**

It includes:

**(a)** Cropmanagement.

**(b)** Soil management,and

**(c)** Watermanagement.

According to G. N. Agrios (1997), the main goals of an integrated plant disease control program are:

**1.** To eliminate or reduce the amount of initialinoculum.

**2.** To reduce the effectiveness of initialinoculum.

**3.** To increase the resistance of thehost.

**4.** To delay the onset of disease,and

**5.** To slow down the secondarycycle.

**Programmes of Integrated Plant Disease Management:**

The programme of Integrated Plant Disease Control can be taken against a particular common disease, such as late blight disease of potato or against all diseases affecting a particular crop such, as Potato, Apple, Citrus etc.

**Integrated Management in an AnnualCrop:**

The following programmes must be undertaken for annual plants like potato against different diseases like late blight (fungus), brown rot and ring rot (bacteria), leaf roll of potato (virus – potato virus-1), etc.:

**1.** Stock tuber should be free from any disease i.e., a healthy tuber isneeded.

**2.** Field should be cleaned from previous years plant debris and tubers, if any, where pathogen may perpetuate for the nextseason.

**3.** Crop rotation must be done with legume or any other crop plant, except Solanaceous

members.

**4.** Plant should be protected from disease through foliar spray at regularintervals.

**5.** Make the soil loose by ploughing and expose it to sunlight for few days in summer months and/or fumigate the soil with chemicals.

**B. Integrated Management in a PerennialCrop:**

For perennial crop like orchard crop (peach, apple, pear etc.); following programme should be undertaken:

**1.** Nursery-stock should be fumigated (to eradicate the nematode, ifpresent).

**2.** The stock should be fumigated (to eradicate the nematode, ifpresent).

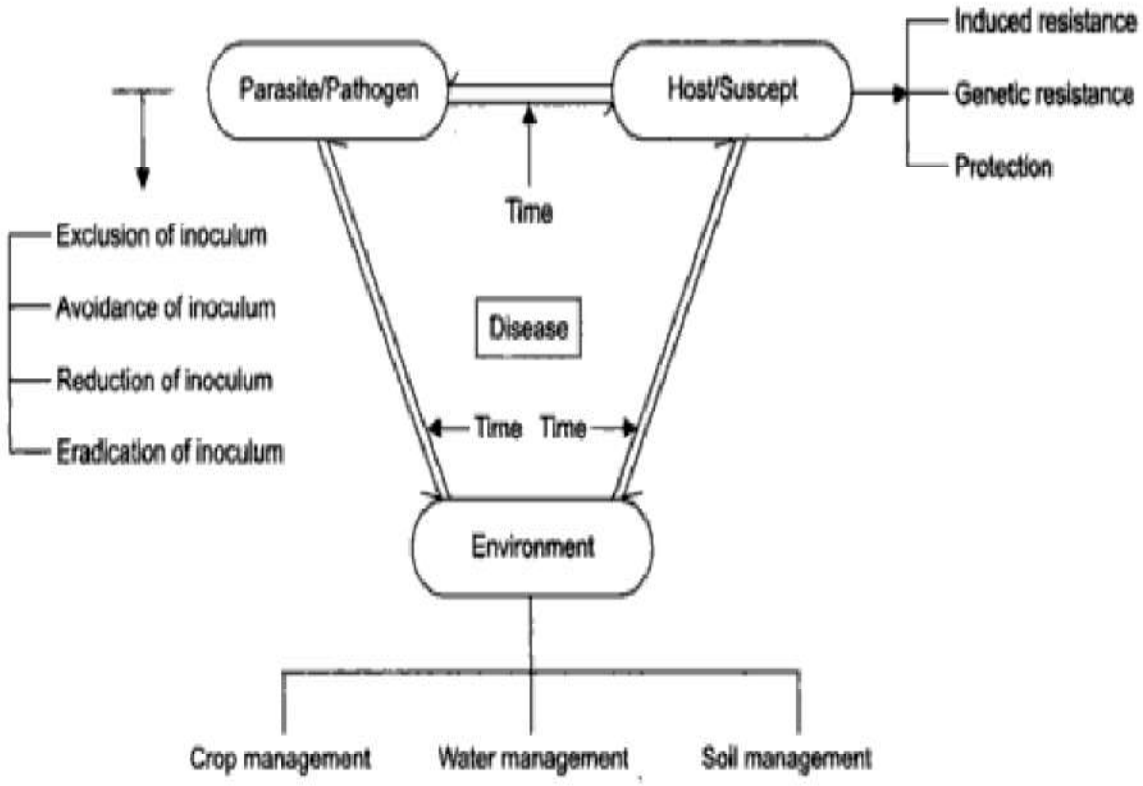
**3.** Thegrowingfieldshouldbefumigatedbeforeplantingtoeradicateanypathogenlike*Armillaria, Phytophthora*, if present.

**4.** Proper drainage in the field should bemaintained.

**5.** Stock should not grow in old field near the old plants that may carry different pathogens like canker, etc.

**6.** The field should be properly irrigated, and supplemented byfertilisers.

**7.** Plants should be sprayed regularly to protect them from different diseases and insectpests.



## 11. Fungicides: types anduses

**Fungicides** are biocidal chemical compounds or biological organisms used to kill parasitic fungi or their spores. A fungistatic inhibits their growth. Fungi can cause serious damage in agriculture, resulting in critical losses of yield, quality, and profit. Fungicides are used both in agriculture and to fight fungal infections in animals. Chemicals used to control oomycetes, which are not fungi, are also referred to as fungicides, as oomycetes use the same mechanisms as fungi to infectplants.[

Based on chemical nature: **inorganic** &**organic**

Based on time of application: **protectant** (use before infection) &**therapeutant** (use after infection)

Based on uptake by and mobility within plant tissue: **Systemic** &**non-systemic fungicides**

**Bordeaux mixture**

It is a mixture of copper sulphate: quick lime (or hydrated lime): water :: 8:8:100. Copper is the only ingredient in the Bordeaux mixture that is toxic to pathogens and, sometimes, to plants, whereas the role of lime is primarily that of a "safener." It controls many fungal (includingoomycete) and bacterial leaf spots, blights, anthracnoses, downy mildews, and cankers. Copper ion ($Cu^{2+}$) is toxic to all cells because it reacts with sulfhydryl (-SH) groups of certain amino acids and causes denaturation of proteins and enzymes.

**Organic protectant fungicides**

**Thiram** used as seed treatment, soil application to control damping off, seedling blights

**Pentachloro nitrobenzene** used as a dip or in the furrow at planting time and selectively toxic to *Rhizoctonia, Sclerotium & Plasmodiophora*

**Captan** used as Leaf spots, blights, and fruit rots on fruit crops, vegetables, ornamentals. The reactive group of captan is $–S-C(Cl)_3$ which interferes with the metabolic processes of sulfur containing enzymes and coenzymes

**Systemic fungicides**

**Metalaxyl** affect nucleic acid (rRNA) synthesis by inhibiting the activity of RNA polymerase I; block uridine incorporation into the RNA chain against Oomycetes, for control of damping off, downy mildew, late blight of potato

**Carbendazim** inhibits mitosis and cell division in target fungi. It interferes microtubule

assembly by binding with β-tubulin. As a result non-disjunction or chromosome loss may occur causing death of the fungalpathogens

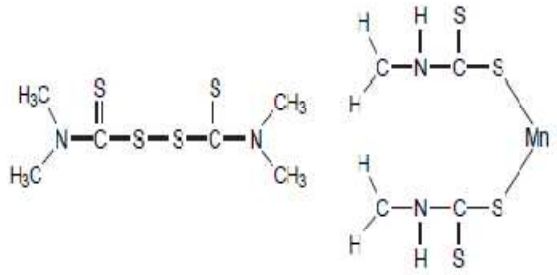
**Plantvax & Vitavax** are the first systemic fungicides to be discovered in 1966 for complete control of internally seed-borne infection of loose smut (*Ustilago nuda*) in barley. They cause dysfunction of succinate dehydrogenase (SDH) in the tricarboxylic cycle and mitochondrial electron transport chain, inhibiting the activity of Complex II and respiration in fungal cells

**Fosetyl-Al** prevents the spore germination and penetration of pathogen into the plant through blocking its mycelial growth and spore production and improve the natural defense mechanism of the plant and induce phytoalexin production against Oomycetes

**Edifenphos** inhibit lipid biosynthesis in fungal cell membrane and inhibit chitin biosynthesis

**Morpholines & triazoles** inhibit sterol biosynthesis

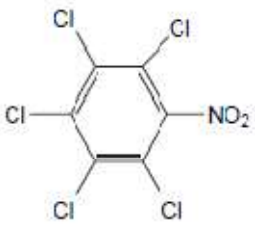
Fungicides are sold commercially as a mixture of active ingredient (that which kills the fungus) and other substances, (i.e., carriers, diluents, solvents, wetting agents, emulsifiers, etc.). The formulation indicates the portion of the product that is active ingredient and the physical form of the product.



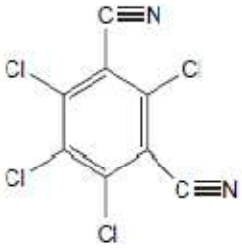
Thiram



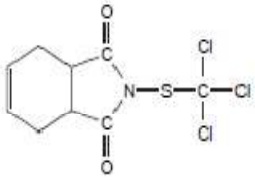
Maneb



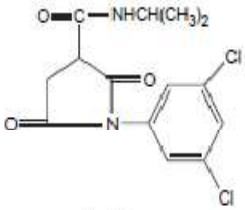
Pentachloro-nitrobenzene



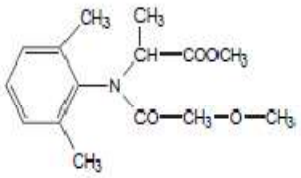
Chlorothalonil



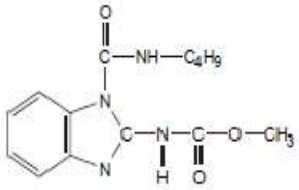
Captan



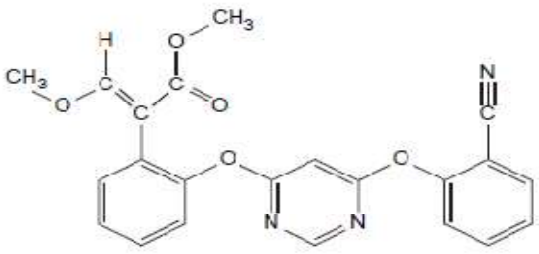
Iprodione



Metalaxyl



Benomyl



Azoxystrobin

**Role of Fungicides in Disease Management:**

Plant diseases are best managed by integrating a number of control practices that may include: crop rotation, selection of disease-tolerant or disease-resistant crop cultivars (cultivars genetically less susceptible than other cultivars), time of planting, level of fertilization, micro-climate modification, sanitation, and application of fungicides. Fungicides are often a vital part of disease management as

(**a**) They control many diseases satisfactorily

(**b**) Cultural practices often do not provide adequate disease control

(**c**) Resistant cultivars are not available or not accepted in the marketplace for many diseases, and (**d**) certain high value crops have an extremely low tolerance for disease symptoms.

In contrast with most human medicines, most fungicides need to be applied before disease occurs or at the first appearance of symptoms to be effective. Unlike with many diseases of humans and animals, applying fungicides cannot heal symptoms already present, even if the pathogen is killed. This is because plants grow and develop differently than animals. Fungicides typically only protect new uninfected growth from disease. Few fungicides are effective against pathogens after they have infected a plant. Those that do have “curative” properties, which means they are active against pathogens that have already infected the plant, have limited ability to do so, often only being active on a pathogen within a few days of infection.

Many fungicides have targeted activity that imparts high efficacy against specific pathogens, which means low potential for toxicity to humans and other organisms, but also results in a high risk of pathogens developing resistance to the fungicide. A resistant pathogen is less sensitive to the action of the fungicide, which results in the fungicide being less effective or even ineffective. Fungicides that are designed to target specific enzymes or proteins made by fungi do not damage plant tissue, thus they can penetrate and move inside leaves enabling curative properties and increasing the amount of plant tissue protected to more than just where fungicide was deposit when applied. Since the mode of action of these fungicides is so specific, small genetic changes in fungi can overcome the effectiveness of these fungicides and pathogen populations can become resistant to future applications.

Disease management strategies that rely heavily upon curative application of fungicides often lead to more resistance problems due to….

(**a**) the large size of the pathogen population when the application is made from which resistant individuals are being selected.

(**b**) the difficultly in eradicating a pathogen entirely from inside the plant. Fungicide resistance is covered in more detail in a separate section.

Growers often use disease forecasting systems or action thresholds, when these are available, to ensure fungicides are applied when needed and to avoid the expense and possible environmental impact of unnecessary applications. Forecasting systems have been developed for a number of diseases based on an understanding of the environmental conditions favourable for their development. Typically these are based on temperature and relative humidity or leaf wetness in the area where the crop is grown. Threshold-based fungicide programs involve routinely scouting the crop for symptoms, then applying fungicides when the amount of symptoms reaches a critical level beyond which the disease cannot be controlled adequately. An example of a critical level is one disease spot per five leaves examined. Knowledge of the disease cycle of the pathogen is important when developing and using forecasting systems and thresholds. Important aspects of the disease cycle include whether the disease is monocyclic (one generation per year) or polycyclic (multiple generations) and latent period (time between infection and production of new inoculum).

Economics often influence the choice of fungicide and application timing. Expensive fungicides and numerous applications are used on valuable plantings that might incur substantial economic loss in the absence of treatment, such as fruit trees and golf courses. Recognizing that with some diseases crop yield is not impacted when severity is low, an economic threshold is used to determine when fungicide treatment is needed. The crop tolerance level, or damage threshold, can vary depending upon the stage of the crop development when attacked, crop management practices, location and climatic conditions.

**Application Methods**

Fungicides are applied as dust, granules, gas, and, most commonly, liquid. They are applied to:

**(a)** Seed, bulbs, roots of transplants, and other propagative organs. These treatments are usually done by the seed company. Some treatments need to be done by the grower on-site at the time of planting. The goal is to kill pathogens that are on the planting material or to protect the young plant from pathogens in the soil.

**(b)** Soil either in-furrow at planting, after planting as a soil drench (including through drip irrigation), or as a directed spray around the base of the plant.

**(c)** Foliage and other aboveground parts of plants by means of a sprayer.

**(d)** Inside of trees via trunk injection.

(**e**) Harvested produce, as a dip or spray in the packinghouse.

Fungicides are used as a formulated product consisting of an active ingredient plus inert ingredients that improve the performance of the product. Fungicides are typically mixed with water then applied by spraying. Application equipment ranges from small hand-held and back-pack sprayers to large spray units carried by tractors or aircraft (Figures 16-22). A few fungicides are applied as dusts. Fungicides can also be applied in greenhouses as smoke, mist, fog or aerosol. Coverage of all parts of the plant susceptible to the disease is critical because very few fungicides can move adequately throughout a plant. Advancements are continually being made to nozzles and sprayers to improve coverage (Figures 17 and 19).

For many diseases, effective control necessitates multiple applications of fungicides, sometimes as frequently as every 5 days. Repeated applications are needed to protect new growth and to replace fungicide lost from the plant by chemical decomposition, UV-light degradation, and erosion by wind and water.

**Fungicide Resistance:**

Fungicide resistance is a stable, heritable trait that results in a reduction in sensitivity to a fungicide by an individual fungus. This ability is obtained through evolutionary processes. Fungicides with single-site mode of action are at relatively high risk for resistance development compared to those with multi-side mode of action. Most fungicides being developed today have a single-site mode of action because this is associated with lower potential for negative impact on the environment, including non-target organisms.

When fungicide resistance results from modification of a single major gene, pathogen subpopulations are either sensitive or highly resistant to the pesticide. Resistance in this case is seen as complete loss of disease control that cannot be regained by using higher rates or more frequent fungicide applications. This type of resistance is commonly referred to as "**qualitative resistance**".

When fungicide resistance results from modification of several interacting genes, pathogen isolates exhibit a range in sensitivity to the fungicide depending on the number of gene changes. Variation in sensitivity within the population is continuous. Resistance in this case is seen as an erosion of disease control that can be regained by using higher rates or more frequent applications. Long-term selection for resistance in the pathogen by repeated applications may eventually result in the highest labeled rates and/or shortest application intervals not being able to adequately control the disease. This type of fungicide resistance is commonly referred to as "quantitative resistance". Comments about resistance risk of fungicides are included.

Fungal isolates that are resistant to one fungicide are often also resistant to other closely-related fungicides, even when they have not been exposed to these other fungicides, because these fungicides all have similar mode of action. This is called cross resistance. Fungicides with the same Group Code are likely to exhibit cross resistance. Occasionally negative cross resistance occurs between unrelated fungicides because the genetic change that confers resistance to one fungicide makes the resistant isolate more sensitive to another fungicide.

Managing fungicide resistance is critically important to extend the period of time that an at-risk fungicide is effective. The primary goal of resistance management is to delay its development rather than to manage resistant fungal strains after they have been selected. Therefore, resistance management programs need to be implemented when at-risk fungicides first become available for commercial use. The objective of resistance management is to minimize use of the at-risk fungicide without sacrificing disease control. This is accomplished by using the at-risk fungicide with other fungicides and with non-chemical control measures, such as disease resistant cultivars, in an integrated disease management program.

It is critical to use an effective disease management program to delay the build-up of resistant strains. At-risk fungicides should be used at the manufacturer's recommended rate (full rate) and application interval. Using full rates is expected to minimize selection of strains with intermediate fungicide sensitivity when resistance involves several genes (quantitative resistance). At-risk

fungicides should be used in alternation with other at-risk fungicides with different modes of action or different chemical groups, and they should be combined or alternated with fungicides that have a low resistance risk.

When one crop could serve as a source of inoculum for a subsequent crop, the alternation scheme among at-risk fungicides should be continued between successive crops such that the first at-risk fungicide applied to a crop belongs to a different cross-resistance group than the last at-risk fungicide applied to the previous crop. Some at-risk fungicides are formulated as premix products with other fungicides to manage resistance. At-risk fungicides should be used only when needed most. The most critical time to use them for resistance management is early in an epidemic when the pathogen population is small. Multi-site contact fungicides should be used alone late in the growing season, where they have been shown to provide sufficient disease control to protect yield. Another important component of resistance management is assessing disease control and reporting any loss of efficacy potentially due to resistance.

To promote resistance management, companies registering fungicides are voluntarily putting on the labels guidelines developed recently by **EPA** through a joint effort with the Canadian Pest Management Regulatory Agency (**PMRA**) under the North American Free Trade Agreement (**NAFTA**).

## 12. Study of some plant diseases with reference to symptoms, etiology and controlmeasures

### Fungaldiseases:

#### Wart disease of potato

Wart disease of potato also known as Black wart of potato, was first described in 1895 from Hungary. In India, the disease was first reported from Darjeeling by Ganguly and Paul (1953) and continues to be restricted to thatarea.

#### Symptoms of Wart Disease:

The symptoms of the disease appear only on underground parts except roots of the planti.e. tubers, buds of stems, and stolons. These are characterised by warty, tuberous and dirty Cauliflower like outgrowths on infectedparts.

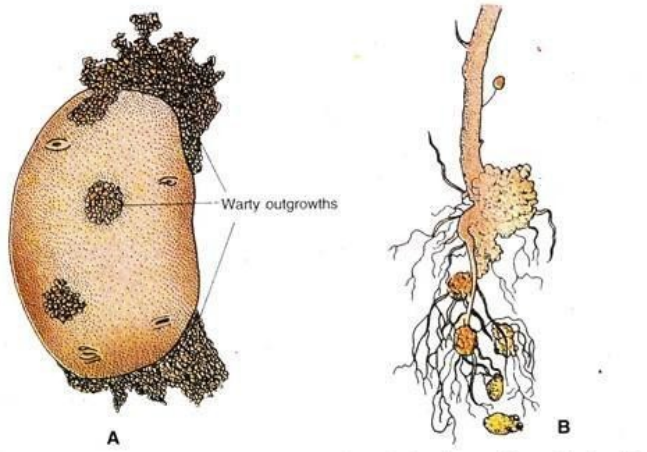


Fig. 22.1. Symptoms of wart disease of potato on a potato tuber (A) and underground parts of a plant (B).

The warty outgrowths vary in size from small protuberances to large intricately branched systems. Early in the growing season they are green or greenish-white in colour ifexposedto light but are cream coloured or black on underground parts.

On the tubers, mostly the warts are larger than the tuber itself covering the whole tuber. In advanced stages, the warts become dark black in colour and may sometimes be attacked by saprophytic fungi.

The wart usually consists of distorted proliferated branched structures grown together into a mass of hypertrophiedtissue,

**Causal Organism:**

The causal organism is *Synchytrium endobioticum* (Schilb.) Peres.- a holocarpic endoparasite.

**Disease Cycle:**

The primary infection of the young potato crops available in the field takes place through the germination of resting spores present in the soil into Zoospores.

These zoospores after swimming for a while land on the underground shoot parts, withdraw their flagella and enter the host cells where they settle down at the bottom of the host cells.

Each of the neighbouring cells start dividing repeatedly followed by swelling, ultimately resulting in the formation of wart. There warts are the main Symptoms and contain the material for secondary infection. The sporangia contained in the warts produce zoospores upon disintegration of thehosts.

These zoospores are responsible for secondary infection. If environmental conditions are favourable, the zoospores are formed repeatedly and secondary infections may take place as well causing serious damage to the potato crop.

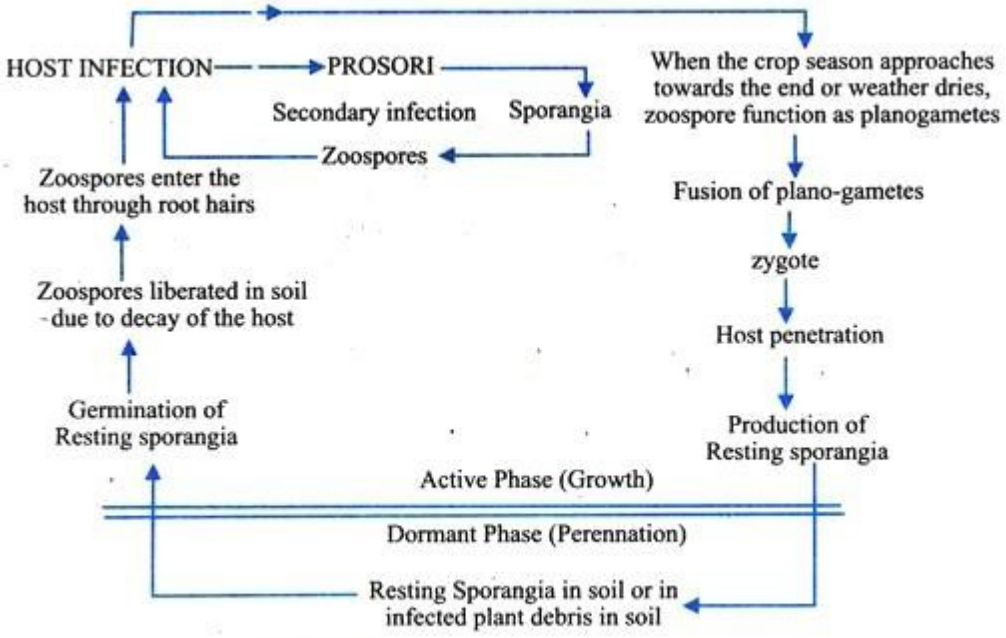



Fig. 22.2. Disease cycle of wart disease of Potato.

When the weather conditions become unfavorable and the crop season approaches towards the end, the zoospores function as plan gametes, fuse and produce resting sporangia. While the crop is harvested, the resting sporangia remain buried in the soil and perennate waiting for the favorable conditions to return back.

**Control Measures of Wart Disease:**

It is very difficult to control the disease once it has been introduced in the field.

However, some of the control measures practised are listed below:

**(i)** Entry of diseased material into healthy areas should beprevented.

**(ii)** The diseased potato tubers should bediscarded.

**(iii)** Soil treatment may control the disease to a large extent. These include steam sterilisation and application of mercuric chloride—copper sulphate and 5 percent formaline. But these are very costly.

**(iv)** Cultivation of disease resistant varieties continuously for 8-10 years is the only effective controlmeasure.

**Early blight of Potato**

Early blight is a common foliage disease of potato and tomato. According to Mathur, Singh and Nagarkote (1971), there have been outbreaks of early blight of potato during the past-5 or 6 years in the plains of Uttar Pradesh. The pathogen causes injury to the leaves as a leaf spot disease and instigates permature defoliation. Finally it incites tuber rot of potato and fruit rot of tomato.

**Symptoms of Early Blight Disease:**

The disease appears on the leaflets, 3-4 weeks after the crop is sown as small, isolated, scattered pale brown to dark spots, oval or angular in shape mostly up to 3 or 4 mm in diameter. Each spot is usually delimited by a narrow chlorotic marginal zone which fades into the normal green.

The chlorotic zone increases with the increase in size of the spot. As a rule the oldest (lowest) leaves are affected first and the disease progresses upwards.

The necrotic tissue of the spot often shows a series of concentric ridges which produce a target-board effect, a symptom characteristic of this disease. The number of spots on the leaflets may be a few but if the conditions are favourable the spots increase in number and size involving the entire leaf surface.

Under humid conditions, the diseased areas coalesce and big rotting patches appear on the leaf surface. In severe cases of infection the leaves dry up, shrivel and drop off. Falling of leaves starts with the older (lower) ones until a few remain at the top.

Sometimes there is complete defoliation. Stems and petioles may also develop brown to dark lesions which may finally lead to either worthless plants or collapse of the entire over-ground portion of the plant.

Folsom and Bonds (1925) reported infection and rotting of potato tubers. The surface lesions on the potato tubers are a little darker than the healthy skin and slightly sunken.

**Causal Organism:**

The pathogen causing early blight of potato and tomato is a form-species *Altemana solani* (Ell. and Martin) Jones and Grout of form class Deuteromycetes.

The mycelium consists of light brown, slender, septate sparsely branched hyphae which become dark-coloured with age.

The conidiophores which are relatively short (50- 90 μ long and 9 μ broad) and dark-coloured arise from the older diseased tissue of the host and emerge through the stomata. There are 5-10 transverse septa and a few longitudinal ones. Each conidium develops from a bud formed on the terminal cell of the conidiophore.

**Disease Cycle**:

The mycelium and conidia of the pathogen remain viable for a considerable time, the former for about a year or more in the infected dry leaves and the latter for 17 months at room temperature.

The source of primary inoculum is the infected plant debris such as the dried leaves, stems, potato tubers and contaminated tomato seeds.

Primary infection may be brought about byconidia(**A**) or mycelium from the infected debris in the soil.

According to Walker (1969), the conidia germinate (**B**) at the optimum temperature of 28° to 30°C within 35 to 45 minutes. The germ tubes gain entry into the lower leaves of the host plant through stomata.

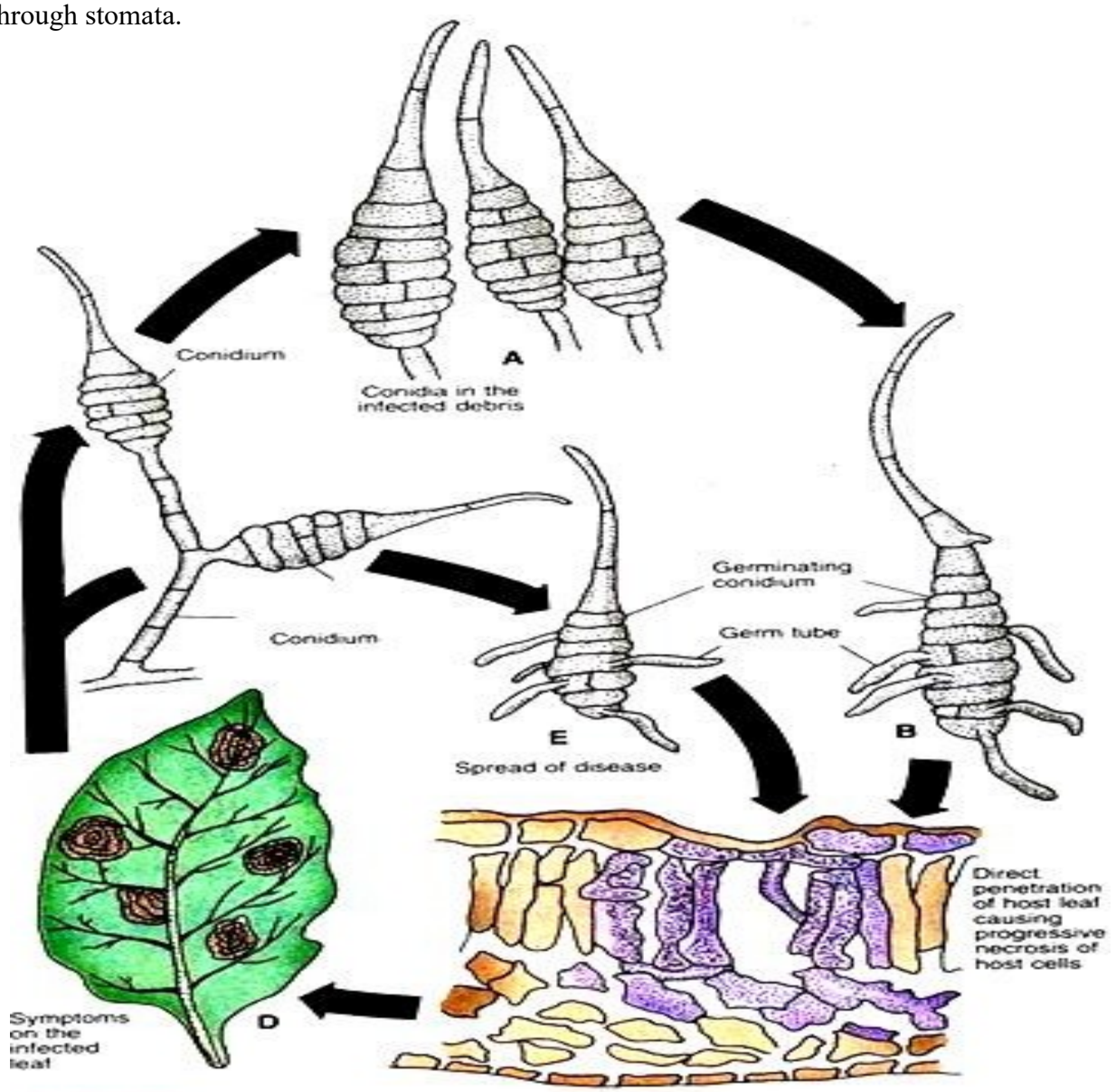



Fig. 22.23 (A–B). Early blight of potato—Pictorial disease cycle of *Alternaria solani*.

Walker (1969) reported that the fungus penetrates the host leaf and stem directly through the epidermis (**C**). The incubation period varies from 48 to 72 hours. The primary infection usuallyappears on the foliage as leaf spots within 2 or 3 days under favourable conditions of temperature and moisture (**D**).

Secondary spread immediately follows through conidia produced on the primary spots within 5-7 days after infection. Conidia start forming when the spots are about 3 mm in diameter. Rand (1917) reported that heavy dew with rains now and then promote abundant sporulation.

The mature conidia are readily detached and dispersed through the agency of air currents, water and insects. Flees, beetles and Colorado bettles are reported to assist infection by carrying conidia on their bodies and facilitate penetration of germ tubes by inflicting wounds on the host surface.

**Control Measures of Early Blight Disease:**

As in the case of other soil borne diseases, crop rotation and field sanitation are the two rational measures which provide effective check against primary infection from spores in the infected plant debris. Infected dry leaves and dead haulms should be raked together and burned immediately after harvest.

Application of regular spray schedule is another effective measure. When the plants are 15- 20 cm high, weekly sprays (with Bordeaux mixture or other suitable fungicides) throughout the period of plant growth effectively control the early blight. Mathur, Singh and Nagarkote (1971) found that spraying potato crop with Dithane M-45 was most effective against early blight disease.

**Downy Mildew of Grape:**

The best information available indicates that the downy mildew of grape is endemic in North America, where it was for the first time reported by Schweinitz in 1837.

The disease is confined largely to species and varieties of grapes (*Vitis*) although it has been reported also on the five-leaved ivy (*Parthertocissus quinquefolia* Planch.), and on English ivy (*P. tricuspidata* Planch.).

**2. Symptoms of Downy Mildew ofGrape:**

The disease attacks all green parts of the plant leaf blades, petioles, tendrils, green shoots, and fruits at different stages of development.

Early symptoms of the disease on the leaves consist of round light-green spots of an oily appearance on the upper surface, which enlarge even 1 /2 cm or more in diameter. In moist weather it persists; in extremely dry weather it may disappear. Later the spots become yellow, or variegated with tints of yellow and yellowish-brown forming patches of irregular shape.

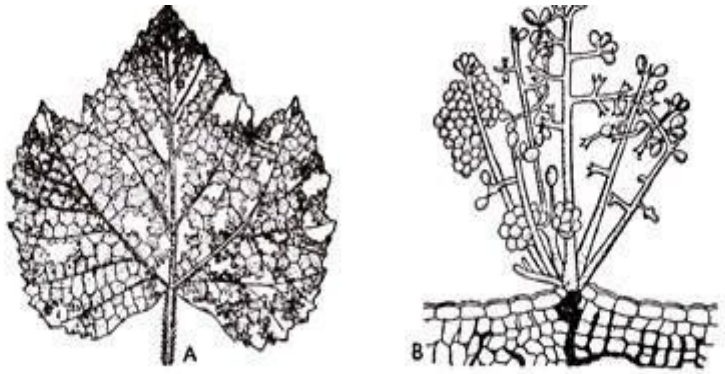



Fig. 361. Downy mildew of grape A. A diseased leaf. B. Section of a diseased leaf with sporangiophores emerging from a stoma.

On the stems, the lesions are brown and sunken, and along with the death of the affected parts, portions of the wine become brittle and break off easily.

Flowers may be completely blighted by early attacks of the disease. Fruits may be attacked when young or when approaching maturity. The young fruits show brownish spots and later become covered with downy mildew, their growth is checked. They may also assume a reddish- brown colour and failing to ripen, develop a soft root.

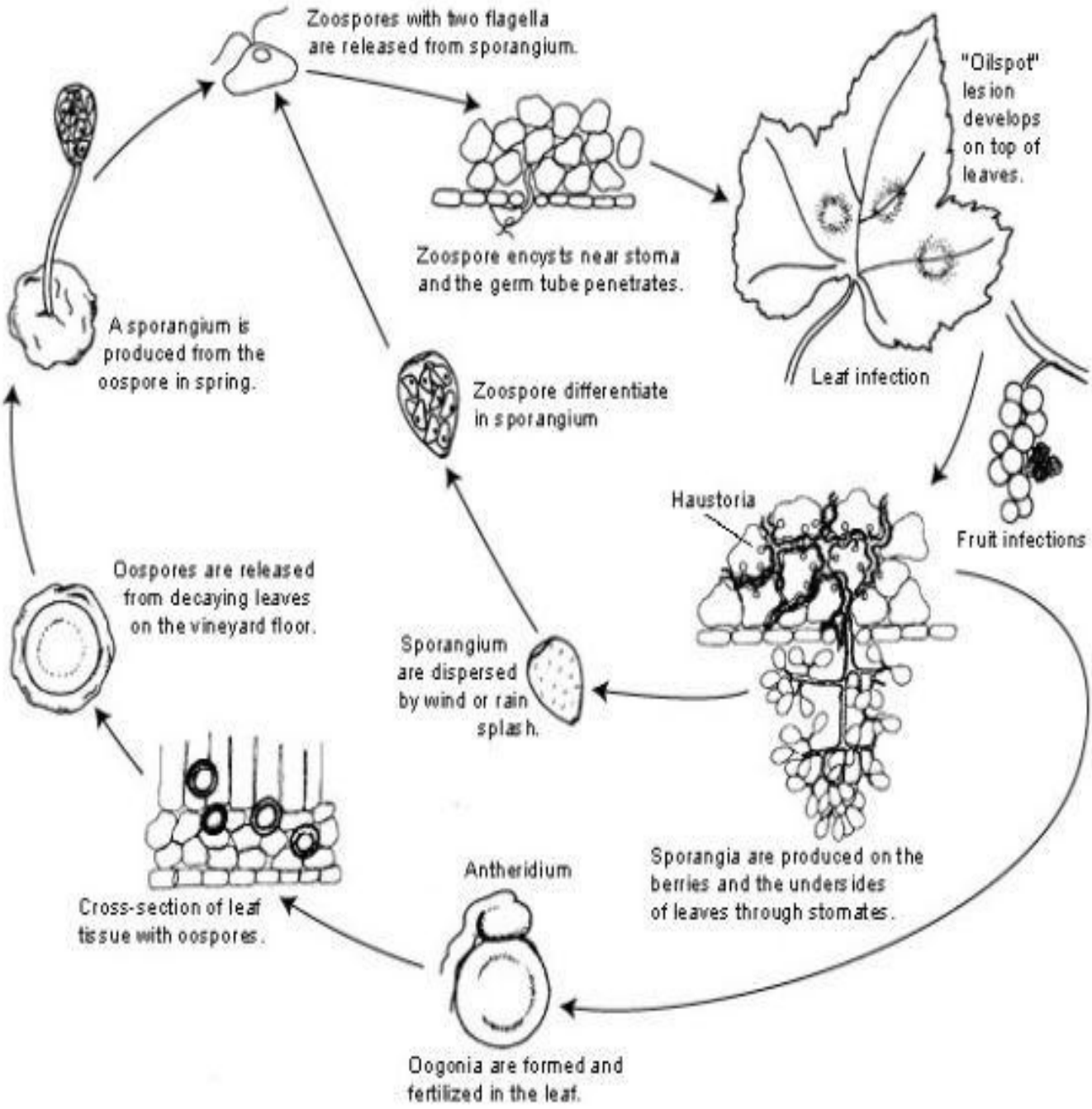
**3. Causal Organism of Downy Mildew ofGrape:**

*Plasmopara viticola* (Berk, and Curt.) Berl. The characteristic aseptate, intercellular mycelium produces knob-like haustoria to absorb food from the host cells. Fasciculate- sporangiophores arise from the hyphae in the intercellular spaces just beneath the lower epidermis and emerge through the stomata.

The oospores are produced from the mycelium embedded within the leaf tissue. The oospore consists of a thick endospore wall surrounded by a thinner rough exposure wall.Disease Cycle of Downy Mildew ofGrape:

In general, the sporangia are not adopted for long survival but serve to spread the disease in localities where the leaves may remain on the vines all the year round. In such cases infection by sporangia may be continuous from season to season. On the other hand, the oospores are capable of survival in the soil for at least a year.

Oospores which remain in the fallen leaves, vine debris, or shrivelled fruits on the ground are probably responsible for the primary infections which break out in the spring. Moreover the fungus may, in some localities, survive in the form of mycelium perennating in the winter buds.
The oospores germinate at a temperature range of 13°C to 33 °C. Primary infections are believed to occur when the zoospores formed during oospore germination, are conveyed by splashing raindrops from the soils on to the lowermost leaves of thevines.


Zoospores with two flagella are released from sporangium.
"Oilspot" lesion develops on top of leaves.
Zoospore encysts near stoma and the germ tube penetrates.
A sporangium is produced from the oospore in spring.
Zoospore differentiate in sporangium
Leaf infection
Haustoria
Fruit infections
Oospores are released from decaying leaves on the vineyard floor.
Sporangium are dispersed by wind or rain splash.
Sporangia are produced on the berries and the undersides of leaves through stomates.
Antheridium
Cross-section of leaf tissue with oospores.
Oogonia are formed and fertilized in the leaf.

It may so happen that the sporangia produced during oospore germination may themselves be carried on the leaves near the ground and then zoospores are produced there.

Host petetration of the pathogen takes place through the stomata. The mycelium invades the intercellular spaces, ramifies in the host tissue deriving its nutrients in a large measure through haustoria which penetrate the cell lumen. In almost all cases, infection of the leaves takes place through the lower surface.

The optimum temperature for the development of the disease is 18°C to 24°C. Prolonged warm wet weather is conducive to an epidemic of the disease.

They also serve as the source of secondary inocula of the disease. Through the secondary inocula, secondary infections are induced resulting in secondarycycles.

Disease cycle of Downy mildew of grape is very similar to that of Downy mildew of pea, except that the sporangia germinate by secondary zoospores.

**4. Control of Downy Mildew ofGrape:**

The chief methods of control of the disease are as follows:

**(i) Sanitation:**

Destruction of old leaves is recommended to eradicate the source of inoculum since the causal organism of the disease usually hibernates as oospores in the fallen leaves.

**(ii) Spraying of BordeauxMixture:**

Bordeaux mixture, 5-5-50 strength is usually recommended as a protective spray to the susceptible parts; but even up to 8-8-50 is often recommended for epiphytotic conditions. Sporangia or zoospores absorb copper until a toxic limit is reached, the more epiphytotic the disease, the stronger must be the spray to be effective.

**(iii) Spraying of OtherFungicides:**

The spread of the disease can be effec•tively controlled by spraying the vines with 0.3 per cent. Blitox-50, Dithane Z-78, Ferbam or Captan first when the shoots are 6 to 8 inches high, again when they are about to flower, and finally when the fruits are just about to change their colour.

**(iv) Improved Cultivation Practices:**

Certain improved methods of cultivation are helpful to control the disease incidence. Vineyards should be sufficiently open to allow the foliage to dry quickly after rains. This method not only ensures free access of air but facilitates spraying as well. The risk of infection from resting oospores is reduced.

**(v) Use of ResistantVarieties:**

The use of resistant varieties is of course a very useful practice. But even with resistant vines, spraying with Bordeaux mixture is advisable.

**Powdery mildew of rose:**

**Causal Agent:** The fungus, *Sphaerotheca pannosa* var. rosae

**Hosts:** Rose

**Symptoms:** Powdery mildew produces a powdery, whitish coating on the leaves and other plant parts. In case of severe attack, plants may become stunted, leaves may curl, become dried, and drop. Unopened buds, young stems, and thorns may be entirely overgrown with the powdery coating. Only in very rare cases does death of the plant result from infection. Generally, the most favorable conditions for powdery mildew infection are as follows: daytime temperature near 80F (27C) and a relative humidity of 97-100%. These conditions prevail during most of the early part of the growing season, thus control must be made on a regular basis.

**Control:** During seasons of low rainfall in the spring and very early summer when the daytime temperatures are below 80F, control may not be needed until later. Thoroughly applied fungicides can effectively control powdery mildew. To avoid having to spray for powdery mildew control, resistant cultivars can be grown. Please contact your local county extension office for currentinformation.

**White Rust of Crucifers:**

White rust or white blisters disease is one of the common diseases of crucifer crops. It is worldwide in distribution occurring in all the areas wherever crop is cultivated. Both wild and cultivated varieties are attacked.

**Symptoms of White Rust Disease:**

The disease affects all the aerial parts of the plant, the roots are not attacked. Symptoms may appear as a result of two types of infection: Local and Systemic.

In case of local infection, isolated spots or pustules appear on leaves or stems or inflorescence. The pustules are of variable size, measuring 1 -2 mm in diameter and are raised shiny white areas.These may arise in close proximity and coalesce to form large irregular patches.

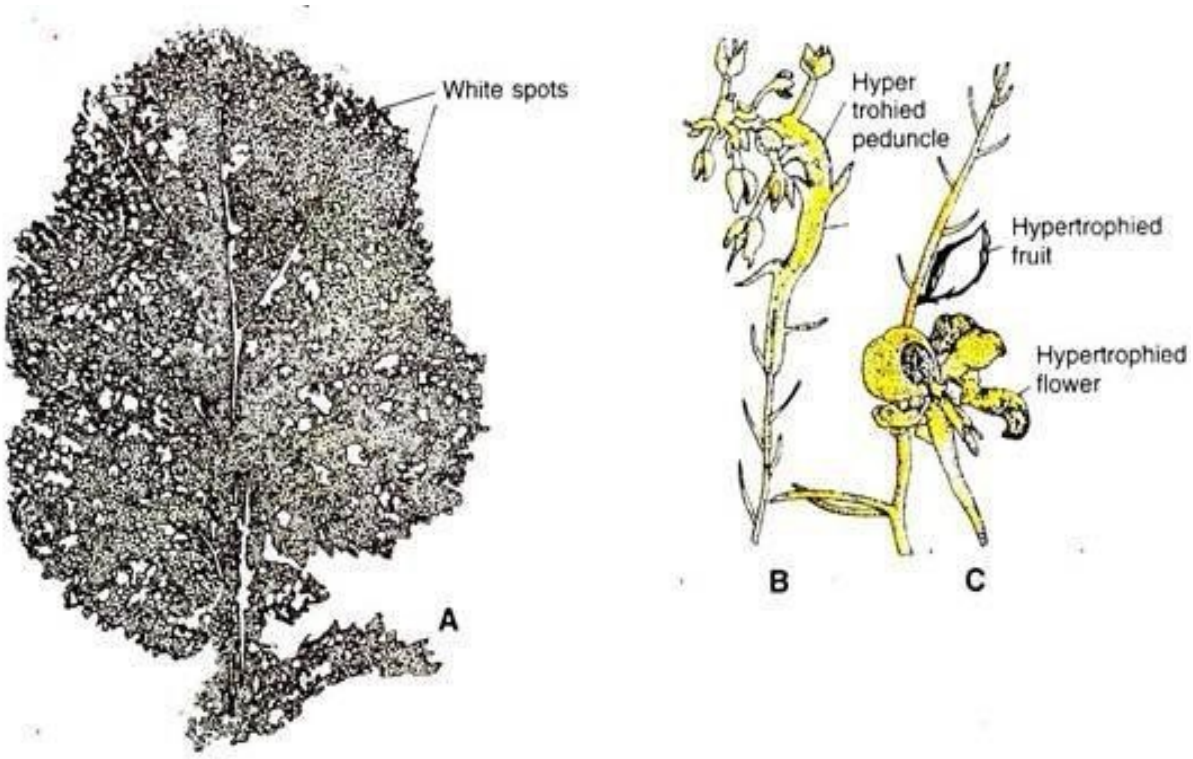



Fig. 22.3. Symptoms of white rust disease on leaves (A) and inflorescence (B and C).

In systemic infections, young stems and inflorescence are infected. The fungus becomes systemic in these parts and the affected tissues are stimulated to various types ofdeformities.

The peduncle and pedicel may become enormously thickened upto 12-15 times, the normal

diameter. Floral parts become fleshy, swollen, green or violet in colour, the stamens falling off early.

**Causal Organism:**

The causal organism *Albugo Candida* (Lev.) Kunze or Cystopus candidus Lev. is an obligate parasite.

**Disease Cycle:**

The primary infection occurs due to oospores perennating in the soil or due to mycelium perennating on perennial hosts. These serve as primary inoculum when the environmental conditions are favourable.

Oospores germinate in presence of water to form a vesicle in which a large number of zoospores are formed. These zoospores swim in a film of water and land on the suitable host, germinate by germ tubes, enter the host and establish infection. The mycelium in the host is intercellular with globose haustoria. Conidiophores, which are clavate, and formed at the tip of hyphae, begin to produce conidiosporangia in basipetal succession.

The conidiosporangia produced during early phase of the growing season cause secondary infection in the host. These are blown away by wind or any other agency, land on the host surface and germinate to formzoospores.

The zoospores germinate by formation of germ tubes which enter the host and cause secondary infection. If the conditions are favourable, this is repeated.

**Control Measures of White Rust Disease:**

The disease may be controlled by the following methods:

**(i)** Clean cultivation and destruction of weed should bepractised.

**(ii)** Crop rotation will avoid the soil borne primaryinoculum.

**(iii)** Spraying with 0.8 percent Bordeaux mixture or Dithane M-45 (0.2%) may be undertaken to check the spread of thedisease.

**(iv)** Disease resistant varieties be preferred.

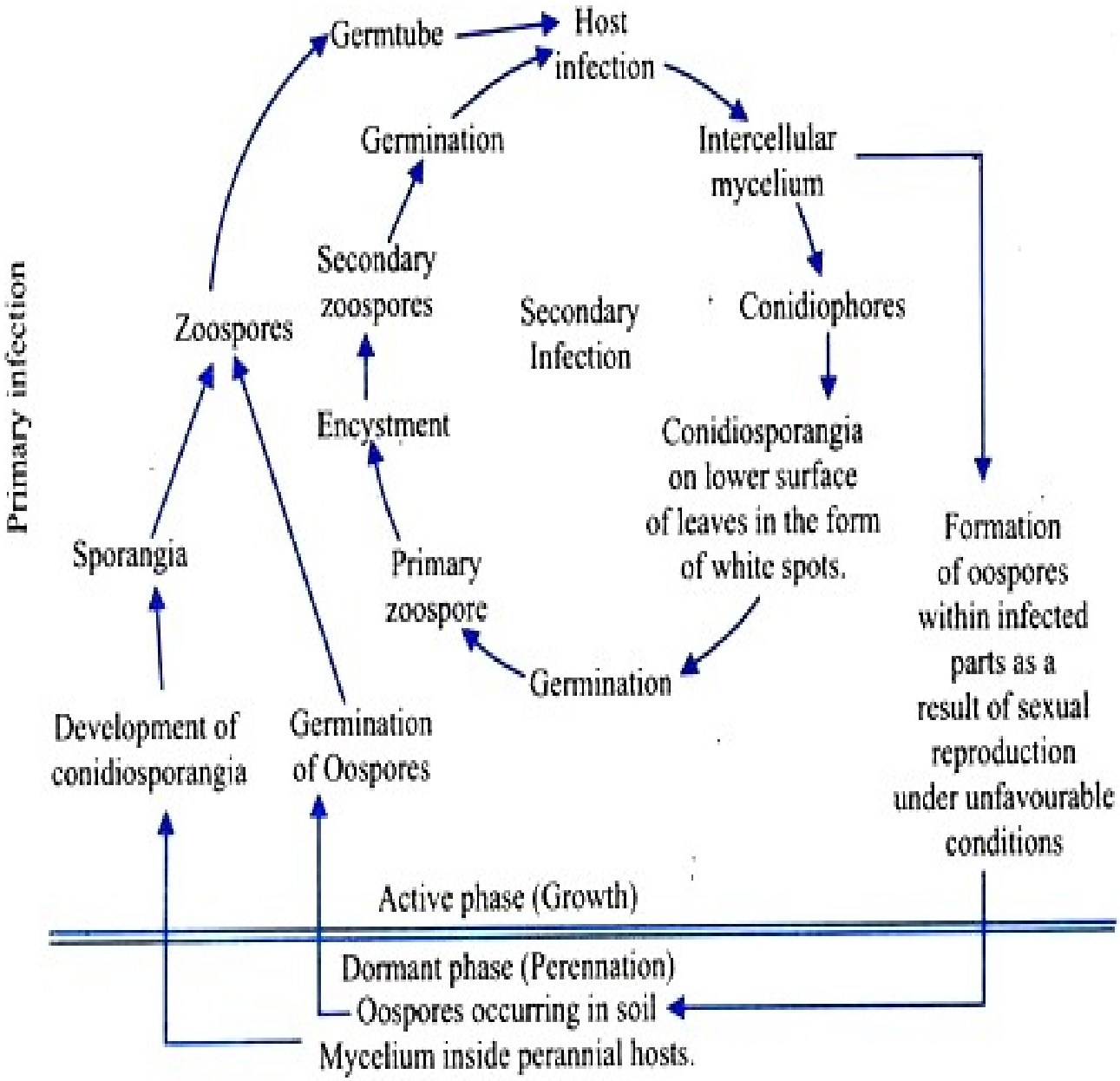



Fig. 22.4. White rust of crucifers–Disease cycle.

**Leaf Curl of Peaches**

**Symptoms of the Disease:**

Symptoms first appear in early spring when the leaves come out of the bud. Some of the leaves appear twisted, puckered, thickened, curled downwards and often greatly distorted. Affected leaves are reddish or purplish in the beginning, later turning to reddish yellow or yellowish grey and finally to yellow to brown.

Affected diseased leaves fall off prematurely and in serious infections, tree become naked due to premature defoliation of leaves in the early spring itself. Blossoms, young fruits and current year's twigs also get affected and diseased and fall off prematurely.

**Causal Organism:**

This disease is caused by *Taphrina deformans* (Berk) Tulasne. The fungus is characterised by intercellular sub-cuticular mycelium, absence of fruit body and the asci produced naked on the host surface.

**Disease Cycle:**

Primary infection takes place through ascospores or blastospores or conidia which are present in the soil of the orchards. Due to rains, these ascospores or blastospores or conidia are washed, splashed or blown onto young tissues where they germinate and cause infection.

At the time of penetration the, germ tube becomes binucleate due to mitotic nuclear division. The binucleate nucleus upon entering the host tissue forms the binucleate mycelium. After the establishment of the infection inside the host, disease develops and symptoms become apparent.

Soon, the asci and ascospores are formed on host surface. Ascospores upon release usually germinate to produce blastospores or conidia.

Secondary infection has not been reported as the fungus can infect only the younger tissues. Older tissues generally become resistant to infection.

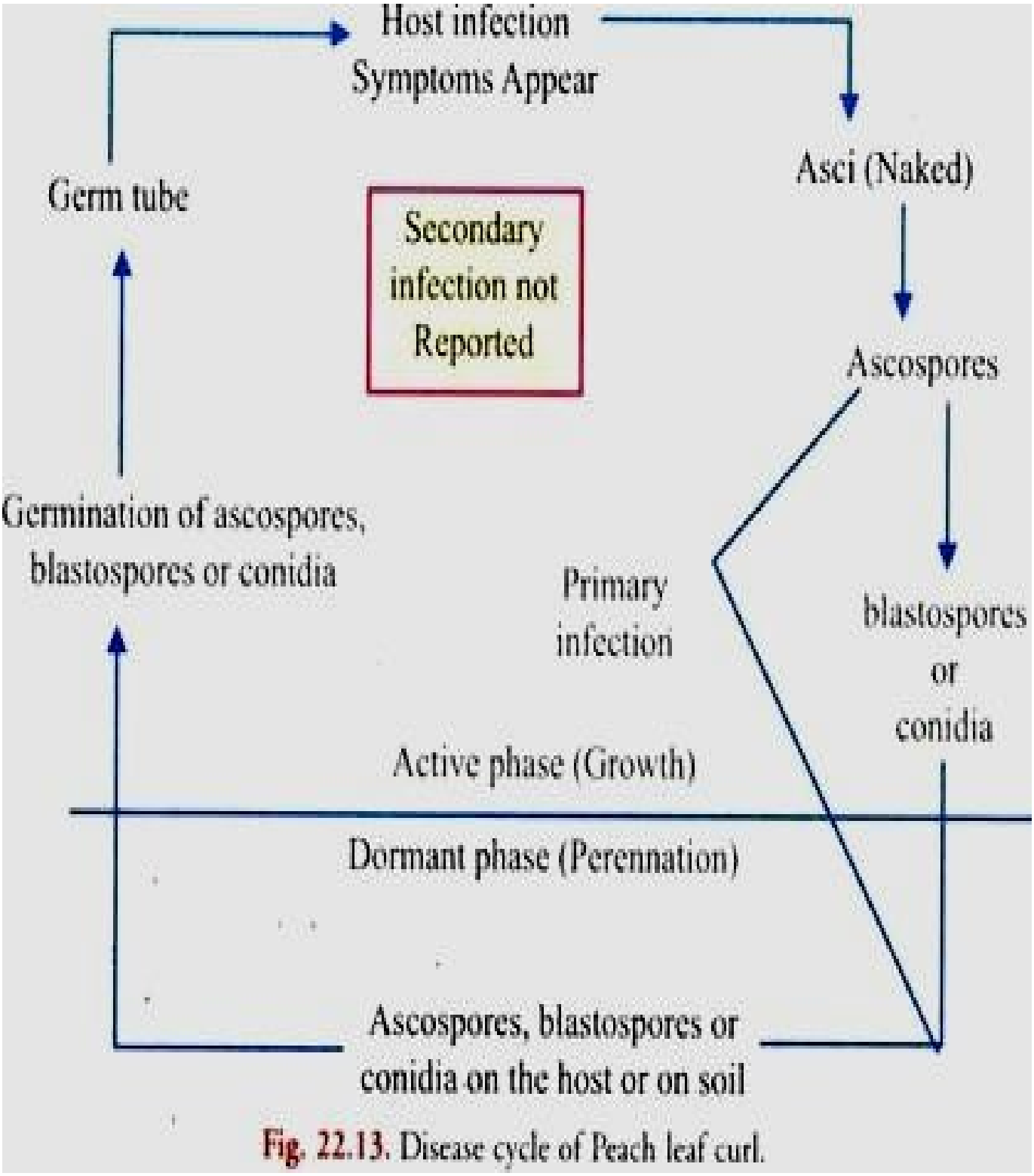



Fig. 22.13. Disease cycle of Peach leaf curl.

**Control Measures of the Disease:**

**(i)** Sanitation of the trees and orchards is important in controlling thedisease.

**(ii)** The tree must not have diseased leaves. Therefore, as soon as the disease leaf is noticed, it should be plucked andburnt.

**(iii)** Fallen infected leaves should be collected andburnt.

**(iv)** Spraying with lime sulphur or copper fungicides helps in controlling the disease. Among the fungicides, Bordeaux mixture (4: 4: 50), Perenox or Fytolan (0.3%) Blitox-50 (0.25%)arecommonly used. One spraying in autumn and another just before opening of the buds considerably reduce the incidence of the disease.

**(v)** Spraying with Cuman five times a year brings about total control of thedisease.

**Rust of Linseed:**

Rust of linseed is a common disease of flax growing areas of the world. In India flax is a major oilseed crop cultivated in almost all the states. The disease generally appears in February or later but Butler (1918) has observed the disease in central India in early November. Linsed Rust

**Symptoms of the Rust of Linseed:**

The leaves are the first to show the symptoms and gradually all the aerial parts of the plant get infected. Large, orange coloured pustules generally appear on the leaves. Small pustules are initially surrounded by chlorotic areas. Little necrosis of the leaves is at first observed but it grows, becomes more general and the leaves prematurelydie.

The pustules on the leaves are uredopustules containing uredospores. Uredopustules may also appear on stems. While the uredopustules on leaves are round and small, those on stems are elongated andirregular.

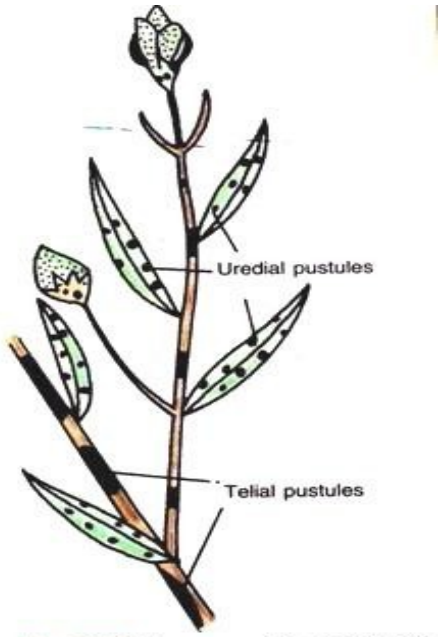


Fig. 22.18. Symptoms of Rust of Linseed.

**Causal Organism:**

The causal organism is *Melampsora lini* (Pers.) Lev.-an autoecious rust.

**Disease Cycle:**

In temperate countries, primary infection takes place through basidiospores which are produced as a result of germination of teleutospores perennating in the soil. But in tropical countries, the teleutospores, produced at the end of growing season i.e. April-May, lose their viability due to excessive heat of summers.

Uredospores too are killed due to excessive temperatures. It is presumed that the uredospores produced on linseed at hills come down to plains to cause infection. Thus the primary inoculum, windblown, fall on the host, germinate and cause infection.

Because the transportation of uredospores from hills takes some time, the outbreak of the disease in plains takes place only after 2-3 months of the sowing of the crop.

Uredospores produced as a result of primary infection cause secondary infection. Teleutospores produced at the end of the growing season appear to have no role to play in the continuance of the life cycle of therust.

**Control Measures of the Rust of Linseed:**

**(i)** Use of disease resistant vars. like NP (RR) 9, 10, 56, 95, 218, 279B 279K3, 368, 381, 389, 415 and 501 is the only effective method to control thedisease.

**(ii)** Seed treatment to kill the teleutospores in hills has also beensuggested.

**(iii)** Avoidance of excessive nitrogenous manures is recommended.

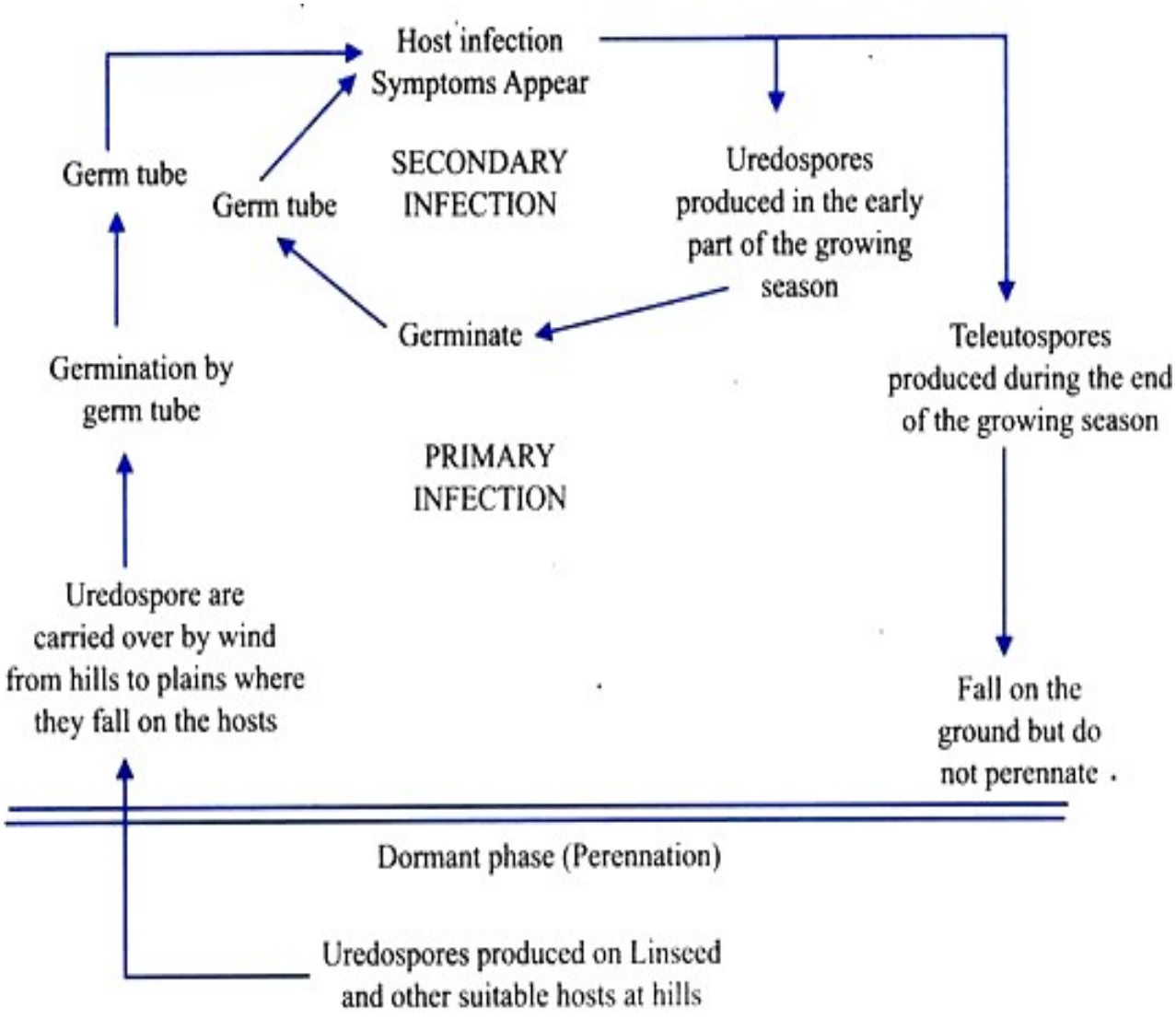



**Fig. 22.19.** Disease cycle of Rust of Linseed.

Sugarcane smut is a fungal disease of sugarcane caused by the fungus ***Sporisorium scitamineum***. The disease is known as culmicolous, which describes the outgrowth of fungus of the stalk on the cane. It attacks several sugarcane species and has been reported to occur on a few other grass species as well, but not to a critical amount. The most recognizable characteristic of this disease is a black or gray growth that is referred to as a "smutwhip"

**Symptoms:**

Two to four months after the fungus has infected the plant, black whip-like structures, instead of a spindle leaf, emerge from the meristem, or growing point, of the plant. The developing whip is a mixture of plant tissue and fungal tissue. The whip reaches maturity between the sixth and the seventh month. When spores that are contained inside the whip are released, the core of the whip remains behind and is a straw-like color.

Plants infected with the fungus usually appear to have thin stalks and are often stunted. They end up tillering much more than normal and this result in leaves that are more slender and much weaker. They sometimes appear more grass-like than non-infected plants. Less common symptoms of the disease are stem or leaf galls and proliferating buds.

**Disease cycle:**

Sugarcane smut is disseminated via teliospores that are produced in the smut whip. These teliospores located either in the soil or on the plant, germinate in the presence of water. After germination they produce promycelium and undergo meiosis to create four haploid sporidia. Sugarcane smut is bipolar and therefore produces two different mating types of sporida. For infection to occur, two sporida from different mating types must come together and form a dikaryon. This dikaryon then produces hyphae that penetrate the bud scales of the sugarcane plant and infect the meristematic tissue. The fungus grows within the meristematic tissue and induces formation of flowering structures which it colonises to produce its teliopores.The flowering structures, usually typical grass arrows, are transformed into a whip like sorus that grows out between the leaf sheaths. At first it is covered by a thin silvery peridium (this is the host tissue) which easily peels back when desiccated to expose the sooty black-brown teliospores. These teliospores are then dispersed via wind and the cyclecontinues.

**Control:**

The management of sugarcane smut is done through the use of resistant cultivars, fungicide and using disease free planting stock. Control is mainly accomplished through the use of resistant cultivars in areas where the disease is present. Fungicides also are used in the control of this disease, but typically resistant cultivars are preferred due to the cost of fungicides. In areas where this disease is not yet found it is important to use disease-free planting stock so as not to introduce the pathogen. Important regulations are sometimes implemented by governments to help prevent the spread of the disease. Quarantines are also implemented in areas that are infected.

**Red Rot of Sugarcane:**

It is a serious and destructive disease of sugarcane. It occurs in tropical and subtropical regions of the world where sugarcane is cultivated extensively. In India, it occurs in most of the sugarcane growing states particularly Bihar, Uttar Pradesh, Madhya Pradesh, Haryana and Punjab.

**Symptoms of Red Rot Disease:**

The symptoms of the disease become apparent after the rainy season on the stems (B) and leaves (C). The earliest symptoms are the yellowing and drooping of the upper leaves (near the tip).

In the later stages the stems shrivel, the rind loses its bright colour, becomes dull in appearance and longitudinally wrinkled. If the diseased stems are split open lengthwise the white pith will be found reddened with patches of white running transversely across.

At the advanced stage of disease blood red lesions with dark margins develop on the mid ribs of the host leaves (B). With the formation of asexual fruit bodies (acervuli), these lesions become covered with powdery masses of conidia which serve to spread the disease.

**Causal Organism:**

The causal agent of the red rot disease of sugarcane is the form-species *Colletotrichum falcatum* Went, of form-class Fungi Imperfecti. The perfect or sexual stage of the fungus has also been found by Arx and Muller. It is described in the Ascomycetcs as *Glomerella tucumanensis*.

**Disease Cycle:**

The incitant lives from one growing season to the next on the debris of the diseased plants. The conidia are short-lived and thus play no role in the perennation of the pathogen, the thick-walled chlamydospores and perithecia are considered as probable means of survival.

Chona (1950) and later Chona and Nariani (1952) reported that the fungus is capable of growing and producing acervuli in the soil. It thus survives in the active stage for 3 or 4 months. The survival of the mycelium for this limited period is sufficient to provide easy catching of the succeeding crop because sugarcane practically has no dead season.

The conidia serve as a secondary means of infection and spread of the disease. They get detached and are dispersed through the agency of wind, water and insects. On reaching the surface of the healthy sugarcane plants, they germinate immediately in the moisture retained in the enclosing sheaths.

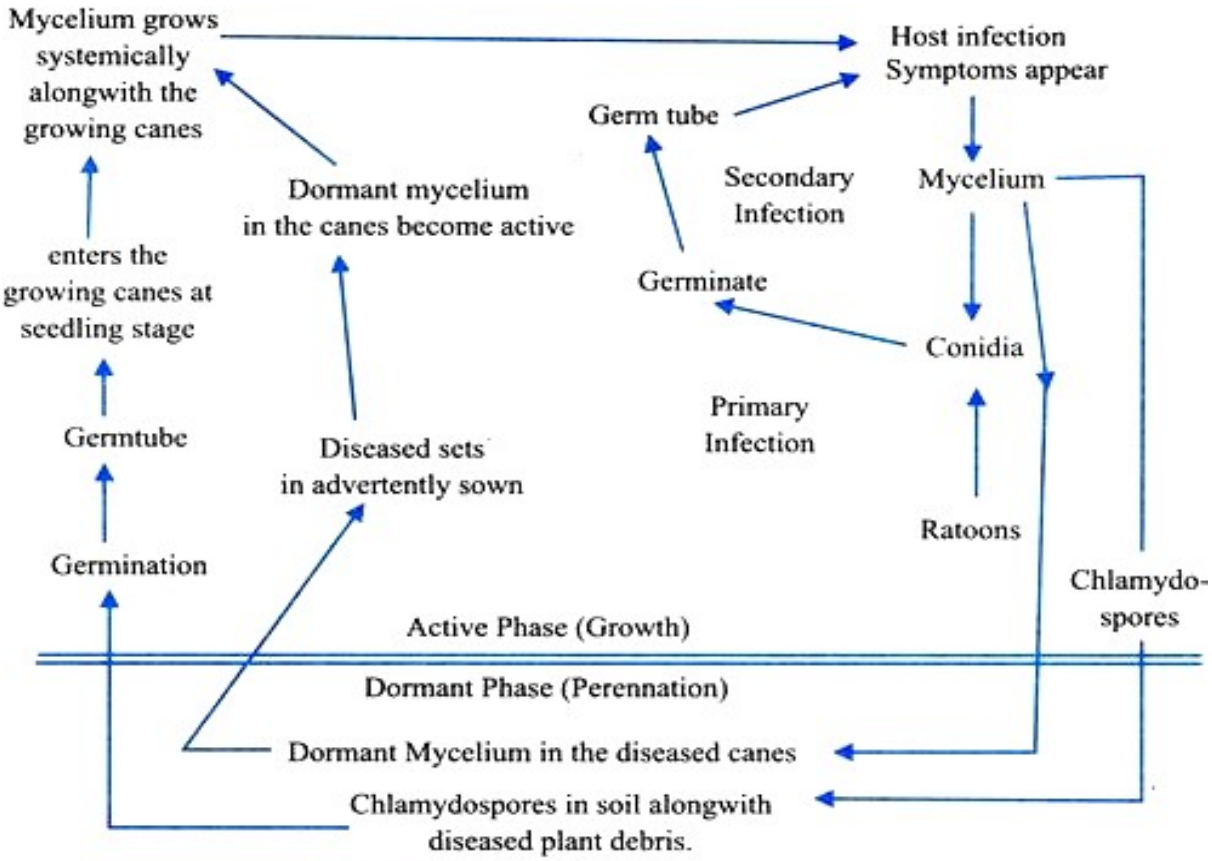



Fig. 22.27. Disease cycle of Red Rot of Sugarcane.

**Control Measures of Red Rot Disease:**

Field sanitation is an important measure to prevent the build source of primary inoculum. It consists in the collection and burning of sugarcane trash in the field. The other equally important preventive measure is the use of sound and healthy seed sets.

Long rotation of crops minimizes soil borne infection. The use of resistant varieties is the most effective method of controlling the disease. The following CO and BO varieties have been recommended to be resistant to red rot disease, Co. 846, Co. 951, Co. 1148, Co. 561, B.O.3, B.O. 7 and B.O. 32.

**Tikka Diseases of Groundnut:**

One of the best known leaf spot diseases is that of *Arachis hypogea* L. (groundnut). Popularly it is called the tikka disease. The tikka disease is a serious disease occurring in areas where the groundnut crop is grown inIndia.

**Causal Organism:**

Tikka disease of groundnuts is caused by two species of *Cercospora: Cercospora personata* (Berk. & Curt.) Elle and Eve., now known as *Cercosporidium personatum* (Berk. & Curt.) Deighton and *Cercospora arachidicola* Hori. The two form-species differ from each other with respect to the size, shape and colour of necrotic lesions they produce, conidia formation and the nature of the mycelium.

**Symptoms of Tikka Disease:**

***C. personatum***

**1.** Leaf spots are small, more circular and about 1-6 mm in diameter(A).

**2.** The necrotic lesions appear on both the leaf surfaces and change from dark brown to dark in colour.

**3.** Young spots lack bright yellow halo which, however, develops around the olderones.

**4.** The mycelium consists of hyphae which are entirelyinternal.

**5.** The septate hyphae ramify in the intercellular spaces and obtain nutrition by sending branched haustoria into the mesophyll (both spongy and palisade)cells.

**6.** The unbranched conidiophores arise in tufts from a dense, globular, and brown to black stroma (23-30 μ in dia.) and emerge by rupturing the host epidermis(B).

**7.** They are geniculate, olivaceous brown, 24-54 p long and 5-8 μ broad and are either aseptate or septate with 1-2septa.

**8.** The conidiophores are confined to the lower surface of the host leaf and are seen arranged in concentric circles in thetuft.

***C. arachidicola:***

**1.** The leaf spots are comparatively larger in size, irregularly circular in outline and 4-10 mm in diameter.

**2.** The leaf spots are often confluent and necrotic lesions occur on both the surfaces. Those on the upper surface are reddish brown to black and on the lower surface are lightbrown.

**3.** There is a yellow halo around each spot but halos on the lower surface are lessdistinct.

**Disease Cycle:**

Primary infection takes place by soil borne conidia, which in the soil and on seeds in the shells. The spread of the disease during the season is by means of wind disseminated conidia.

Penetration is direct through the epidermis and also by way of stomata. Chiefly it occurs through the upper epidermis. However, there is possibility of infection through both the leaf surfaces.

**Control Measures of Tikka Disease:**

Rotation of crops, seed treatment and disposal of infected host debris by burning or burying in deep pits eliminate chances of primary infection from the soil borne inoculum. The seeds within the shells are disinfected with sulphuric acid.

Without shells, they are soaked for half an hour in 0.5% copper sulphate solution. Agrosan GN dressing of naked seeds is equally effective.

To check secondary spread of disease in the field, spraying with suitable fungicides is the only remedy. Among the fungicides the use of Bordeaux mixture has given good results.

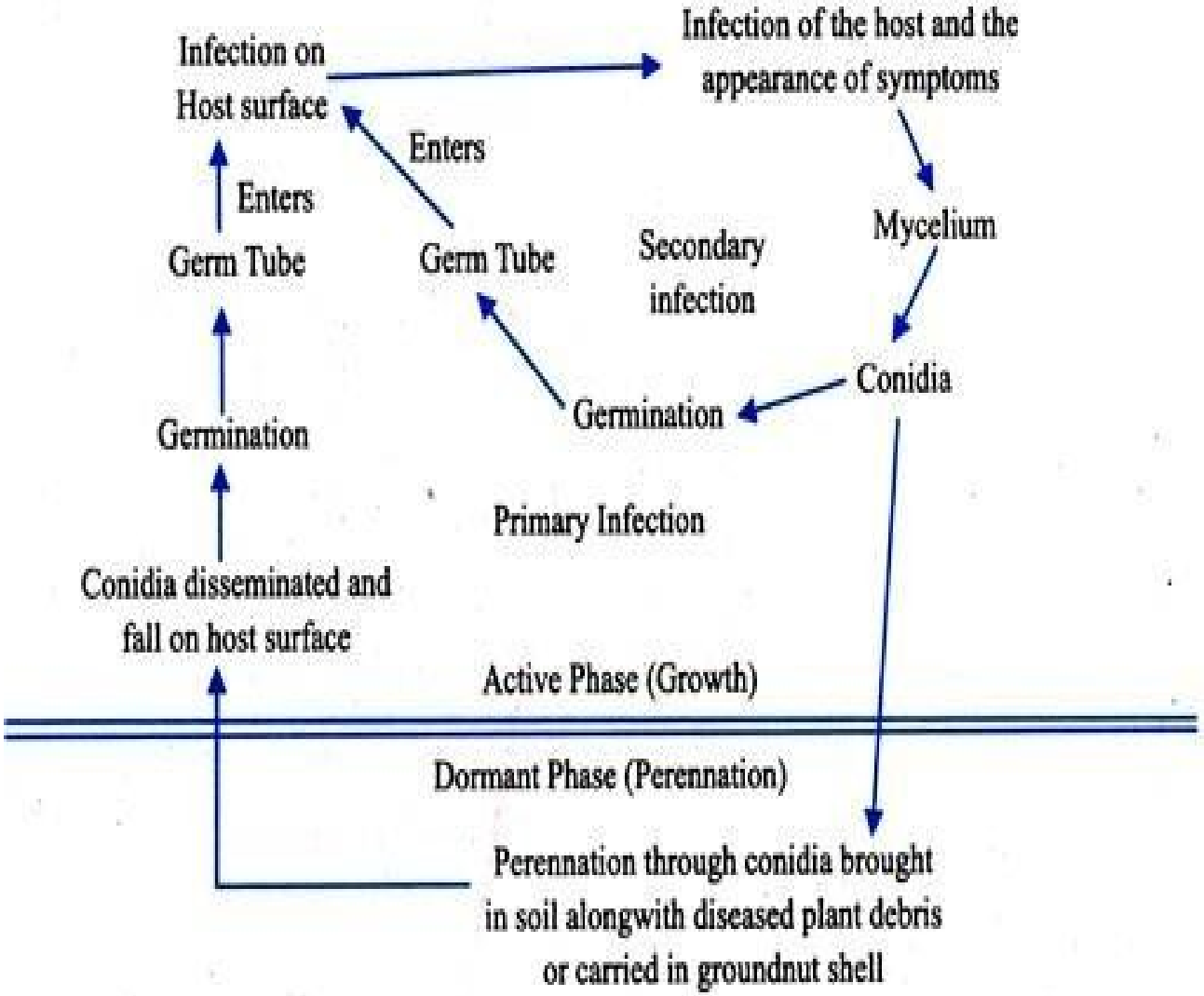



Fig. 22.25. Disease cycle of Tikka disease of Groundnuts.

**Panama disease of banana**

Panama disease is a plant disease of the roots of banana plants. It is a type of *fusarium* wilt, caused by the fungal pathogen ***Fusarium oxysporum*** f. sp. cubense (Foc). The pathogen is resistant to fungicide and cannot be controlled chemically. During the 1950s, Panama disease wiped out most commercial Gros Michel banana production.

**Symptoms**

Two external symptoms help characterize Panama disease of banana:

- Yellow leaf syndrome, the yellowing of the border of the leaves which eventually leads to bending of thepetiole.
- Green leaf syndrome, which occurs in certain cultivars, marked by the persistence of the green color of the leaves followed by the bending of the petiole as in yellow leaf syndrome. Internally, the disease is characterized by vascular discoloration. This begins in the roots and rhizomes with a yellowing that proceeds to a red or brown color in the pseudostem.

*Fusarium* wilt has no symptoms on the growing buds or suckers, no exudates visible within the plant, and no symptoms in the fruit. Bacterial wilt can be characterized by distorted or necrotic buds, bacterial ooze within the plant, and fruit rot and necrosis.

Once a banana plant is infected, it continues to grow and any new leaves will be pale in color. Recovery is rare, but if it does occur, any new emerging suckers will already be infected and can propagate disease if planted.

**Disease cycle:**

Modern banana plants are reproduced asexually, by replanting the plant's basal shoot that grows after the original plant has been cut down.

The disease is dispersed by spores or infected material that travel in surface water. One of the biggest issues in spreading the disease is the means by which new banana plants are planted. Suckers are taken from one plant and clonally propagated to grow new trees. About 30 to 40% of suckers from a diseased plant are infected and not all show symptoms, so the chance of growing a new, already infected plant is fairly high. Finally, the disease is known to infect certain weeds

without showing symptoms, meaning it can survive in the absence of banana plants and remain undetected in a place where bananas are plantedlater.

The disease survives in chlamydospores which are produced as the plant dies and can survive in the soil for upwards of 30 years. When the environment is ideal and there are hostroots available, these chlamydospores will germinate and hyphae will penetrate the roots, initiating infection. There is in increase in the number of symptomatic plants when inflorescences emerge and the highest disease incidence occurs right before harvest. Once infected, microconidia are produced and present within the vessels of the plant's vascular system. Macroconidia are another asexual spore that tends to be found on the surface of plants killed by Panama disease. Infection is systemic, moving through the vascular system and causing yellowing and buckling that starts in older leaves and progresses to younger leaves until the entire plantdies.

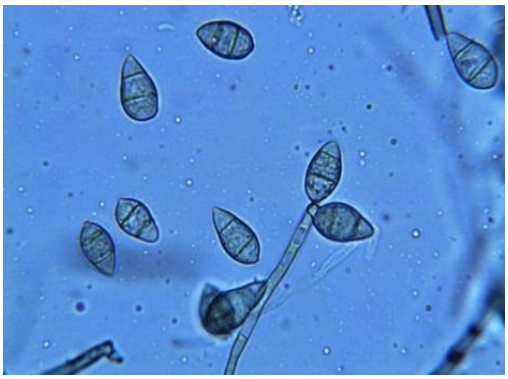
**Controls:**

Currently, fungicides and other chemical and biological control agents have proven fairly unsuccessful, or only successful in vitro or in greenhouses, in the face of Panama disease of bananas. The most commonly used practices include mostly sanitation and quarantine practices to prevent the spread of Panama disease out of infected fields. However, the most effective tool against Panama disease is the development of banana trees resistant to *Fusarium oxysporum* f. sp.Cubense.Unfortunately,theclonalreproductionofbananahasledtoaconsequentiallackofother varieties. Efforts are being made to produce resistant varieties, but with bananas being triploids which do not produce seeds, this is not an easy task. Creating clones from tissue cultures, rather than suckers, has proven somewhat successful in breeding resistant varieties, however, these tend to have decreased success in stress-tolerance, yield, or other beneficial traits necessary for commercial varieties. Nevertheless, these efforts are leading to the best control measure for Panama disease of banana.

Recently, an R gene (RGA2) was transformed into Cavendish bananas which confers disease resistance to Fusarium wilt tropical race 4. This is the first case of successful resistance in the field and is a promising step towards preventing the loss of the Cavendish cultivars that are a huge portion of banana export production and subsistence of many communities.

**Rice Blast:**

Rice (Oryza sativa L.) is the major staple food for nearly one half of the world's population. The ricecropsufferfromanumberofdiseasesamongthem rice blast caused by one of the most devastating agricultural pathogens in the world, a fungus called ***Magnaporthe grisea*** (Hebert) Barr [anomorph: ***Pyricularia grisea*** (Cooke) Sacc.], is one of the most important, causing significant losses in yield. Rice blast was probably first recorded as rice fever disease in China in 1637.



**Symptoms of Rice Blast:**

Rice blast is caused by *Magnaporthe grisea* (Hebert) Barr. All the above ground parts of the plant can be attacked by the fungus at any growth stages. However, Seedling stage, rapid tillering stage after transplanting and flower emergence stage were identified as the most susceptible ones to blast.

The disease can be described based on the part of the plant infected as follow:

**i. LeafBlast:**

On the leaves the lesion/ spots first appear as minute brown specks, and then grow to become spindle-shaped, pointed at both ends. The center of the spots is usually gray or whitish with brown or reddish-brown margin. Fully developed lesions reach 1-1.5 cm long, 0.3-0.5 broad. Under favorable conditions, lesions enlarge and coalesce; eventually kill the leaves.

**ii. CollarRot:**

Infection at the junction of the leaf blade and sheath in the typical brown "collar rot" symptom. A severe collar rot can cause the leaf to die completely. When collar rot kill the flag or penultimate leaf it may have a significant impact onyield.

**iii. NeckBlast:**

This occur when the pathogen infect the neck of the panicle to cause a typical “neck rot” or rotten neck blast symptom. The infected neck is griddled by a grayish brown lesion and the panicle falls down if the infection is severe. If the neck blast occurs before the milk stage, the entire panicle may die prematurely, leaving it white and completelyunfilled.

**iv. PanicleBlast:**

The pathogen also causes brown lesions on the branches on the panicles and on the spikelets pedicles, resulting in “panicle blast”. Infection of the neck, panicle branches, and spikelets pedicles may occur together or may occur separately.

**v. Node Blast:**

The fungus may also attack the stem at nodes, node blast in which the stem bend and break at the node causing spikelets sterility.

Sheath blight of paddy caused by *Rhizoctonia solani* AG1, banded sclerotial disease, rhizoctonia decline.

**Disease cycle:**

The pathogen infects as a spore that produces lesions or spots on parts of the rice plant such as the leaf, leaf collar, panicle, culm and culm nodes. Using a structure called an appressorium, the pathogenpenetratestheplant.*M.grisea*thensporulatesfromthediseasedricetissuetobe

dispersed as conidiospores. After overwintering in sources such as rice straw and stubble, the cycle repeats.

A single cycle can be completed in about a week under favorable conditions where one lesion can generate up to thousands of spores in a single night. With the ability to continue to produce the spores for over 20 days, rice blast lesions can be devastating to susceptible ricecrops.

**Controls:**

The fungus has been able to establish resistance to both chemical treatments and genetic resistance in some types of rice developed by plant breeders. It is thought that the fungus can achieve this by genetic change through mutation. In order to most effectively control infection by *M. grisea*, an integrated management program should be implemented to avoid overuse of a

single control method and fight against genetic resistance. For example, eliminating crop residue could reduce the occurrence of overwintering and discourage inoculation in subsequent seasons. Another strategy would be to plant resistant rice varieties that are not as susceptible to infection by *M. grisea*. Knowledge of the pathogenicity of *M. grisea* and its need for free moisture suggest other control strategies such as regulated irrigation and a combination of chemical treatments with different modes of action. Managing the amount of water supplied to the crops limits spore mobility thus dampening the opportunity for infection. Chemical controls such as Carpropamid have been shown to prevent penetration of the appressoria into rice epidermal cells, leaving the grain unaffected.

**Rice Sheath Blight**

**Causal organism:** *Rhizoctonia solani* AG1-1A Kühn (Teleomorph: Thanatephorus cucumeris (A. B. Frank) Donk.)

**Symptoms**

Early symptoms usually develop on the leaf sheaths at or just above the water line as circular, oval or ellipsoid, water-soaked spots which are greenish-gray in color. As the disease progresses, they enlarge and tend to coalesce forming larger lesions with grayish white centers surrounded by tan to dark brown irregular borders or outlines. Infection can spread to leaf blades and cause irregularlesionswithdarkgreen,brown,oryellow-orangemargins.Thelesionscandevelopextensively and coalesce on partial or whole leaf blades, which may produce a rattlesnake skin pattern. These damaged tissues interrupt the normal flow of water and nutrients to the plant tissues above (leaves and panicles).

**Controls:**

**Field sanitation:** Levels of inoculum can be reduced by destroying weed hosts and other collateral hosts that could harbor sclerotia. However, this approach is not very effective or not feasible for management of sheath blight.

**Management practices to avoid dense canopy**: High seeding rate and overuse of nitrogen fertilizer usually increase stand and induce excessive vegetative growth and canopy density, creating a moist microclimate favorable for disease development. Therefore, avoiding high seeding rates and excessive application of fertilizers, especially nitrogen, can reduce the damage

caused by sheath blight.

**Crop rotation:** Continuous rice or rotation with alternate hosts of the fungus such as soybeans increases inoculum in field soils. Fallow periods, along with efforts at reducing the inoculum by destroying collateral and weed hosts that could harbor sclerotia are viable management practices.

**Chemical Control**

Fungicides can be one of the most effective tools for control of sheath blight. In the United States, several fungicides, including azoxystrobin, provide excellent control of this disease. A single fungicide application is currently recommended to reduce production costs and maximize production returns. The timing of the application is critical for effective control of sheath blight. The disease should be scouted and monitored periodically during the development of the rice crop through heading. The application should be made during the growth stage between panicle differentiation plus five days and heading when the disease level reaches the economic threshold level.

**Bacterial diseases:**

**1. Leaf blight of paddy caused by *Xanthomonas oryzae* pv *oryzae***

*Xanthomonas oryzae:* Pseudomonaceae

Order: Pseudomonales

A distinct yellowish discoloration and drying of the leaf marginsare the typical symptoms of the rice bacterial blight. The leafappears green with broad pale margins from a distance.

A bacterial disease on rice was first reported 75 years ago fromJapan and Philippines. It was described in 1922 as *Pseudomonasoryzae.* In 1949, it was renamed as *Xanthomonas oryzae.* Itremained a minor disease till 1950. In India, the bacterial blightwas first reported in 1959 from Pune. The bacterium wasisolated in 1961 from Tanjore. The disease was however takenseriously only after 1962 when a major outbreak occurred inBihar and parts of north India.

***Morphological Characters:***The earliest symptom of the diseaseis the appearance of dull greenish water soaked or yellow spots,5-10mm in length on the leaf towards the tip or along themargins. This leads to drying and discoloration of the tips ormargins of rice leaves. As the disease progresses, several of theselesions coalesce to form straw-brown large lesions or blightedportions. The inner margin of the blighted patch in contactwith the adjoining green part of the leat is ragged or

wavy.Occasionally, the lesions may extend tip downward along themidrib itself. Small droplets of bacterial ooze may be found in the affected portions. These are pale amber in colour. Whendried, these droplets form minute crusts and impart a roughtouch to the leaves when passed between the fingers. In severecases all the leaves are affected. If such leaves are cut and put inclear water, a turbid ooze of the bacterium streaming out of theleaves can be seen.The bacterium is rod shaped with a single polar flagellum.

***Diversity:***Several strains with different levels of virulence areknown from India. The reaction of rice varieties to the differentstrains under varied ecological conditions is poorly understood.

***Distribution:***Widely distributed in Asia. It is known from allover the country affecting both indigenous and exotic varietiesof rice.

***Habitat Preference:***Plants in well watered shaded areas and innitrogen rich soils are the most affected by the bacteria.

***Life Cycle:***Primary infection is through seeds, the bacteriaremaining in the husk or endosperm. It may also remain inplant debris infecting the nursery. The bacterium also infectscertain grasses, which in turn serve as source for further infection.After infection, the bacterium becomes systemic and vascularnot showing out till weeks after transplanting becoming mostsevere at the time of flowering. A combination of rainy weather,dull windy days and temperature of 22-26°C are most conducivefor the bacterium.

**Human Significance:** The disease affects most varieties of riceall over the country causing severe crop losses (up to 60%).

Survey Method: Standing rice crops with severe infection showthe typical blast symptoms even at a distance. Under less severeconditions small quadrats, say Imxlm, can be randomly placedin selected rice fields and the number of infected tillers of ricecan be counted directly.

**Management:**

Management of bacterial leaf blight is most commonly done by planting disease resistant rice plants. PSB Rc82 is the standard variety of rice used in Southeast Asia, and the use of this cultivar enables the harvest of an estimated 0.8 million metric tons of rice per cropping season that would have otherwise been lost to bacterial leaf blight. Macassane, a new variety released in 2011, has been shown to have improved resistance to bacterial leaf blight and is being used currently in Mozambique.

Traditional treatments, such as the applications of copper compounds or antibiotics, are largely ineffective in the control of bacterial leaf blight. Increasingly, rice is being genetically engineered

for resistance to the disease, as treatment proves difficult. More than 30 genes have been identified as being associated with resistance to bacterial leaf blight, and have been given names Xa1 to Xa33. Biological control methods are relatively recent developments which are not currently in common use. They may be used in the future to reduce damage done by bacterial leaf blight, with experimental data showing up to a 64% reduction in damage.

**2. Moko disease of banana caused by *Ralstonia solanacearum* race 2,**

**Causal organism:***Ralstonia solanacearum* (Yubucchi et al.)

It was first recorded in Guyana in 1840. In India, the disease was first reported from West Bengal. Susceptible varieties are the Robusta and Poovan from Tamilnadu.

**Symptoms:**

Yellowing of younger leaves is the primary symptom. Yellowing progresses downward gradually leading to drooping and drying of leaves Fruit bunch size gets reduced with immature and irregular ripening of fruits. Infected fruits show cracking with bacterial ooze Vascular browning of the fruit along with light coloured vascular discolouration is common.

**Etiology:**

Pathogen is gram negative, lophotrichus bacteria. (More than one polar flagellum), multiplication occurs by bacterial fission.

**Mode of Survival and spread:**

Primary source of inoculum: The bacterium is soil/water borne and also spreads through infected suckers/rhizomes. It survives in susceptible host like banana and heliconia.

Secondary source of inoculum: Bacterial cells spread through irrigation water and also through suckers used for planting.


FIGURE 12-26 Bacterial wilt (Moko disease) of banana caused by *Ralstonia solanacearum*. Banana plants showing different stages of bacteria wilt, including wilted foliage only (A), infection of stalk and early infection of banana fruit (B), and thorough invasion and destruction of banana fruit (C). (D) Invasion and discoloration of several vascular bundles in the banana pseudostem. (E) Early (right) and later invasion and destruction of the contents of infected bananas (left). (F) Colonies of *R. solanacearum* growing on a specialized nutrient medium. [Photographs courtesy of H. D. Thurston, Cornell University.]

**Epidemiology:**

Soil temp 28-320c, relative humidity 87-92%, PH slightly acidic to neutral, clay loam and sandy loam soil and susceptible variety.

**Life Cycle:**

The bacteria survive through infected rhizomes and also in soil for 6 months to 2 years. The spread is through use of infected rhizomes, cutting machetes at the time of planting, and through insects which carry bacteria from oozing suckers and male flowers and bracts to healthy inflorescence and other parts of the plant. Entry into the host is mainly through injuries such as those caused during various cultural operations and attack of insects and nematodes. The bacteria multiply rapidly in the xylem. Auxin balance of the plant is disturbed. IAA is synthesized by the bacterium and by the host and accumulates due to inhibition of the auxin degrading system. Loss of virulence in the bacterium is generally accompanied

**Management:**

Use disease free planting material

Use resistant varieties like Robusta and Grand naine.

Infected plants should be collected and burnt.

Give proper drainage and avoid movement of water from infected to healthy plants

Drip irrigation method reduces the spread of bacteria.

Chemicals: Apply Copper Oxychloride @ 3gm per lit and Sreptocycline @ 0.5 gm per lit as a soil drench

Use bio- agent like Pseudomonas fluorescens.

### ii. Viral diseases:

Tungro is the most serious virus disease of rice in southand southeast Asia from Pakistan to the Philippines.Tungro (yellow-orange) is the result of concurrent infectionby two viruses: the single-stranded RNA virus *ricetungro spherical virus* (RTSV) and the double-strandedDNA virus *rice tungro bacilliform virus* (RTBV)(Figs. 14-44A and 14-44C). Both viruses are transmittedby several leafhoppers (Fig. 14-44B), particularly*Nephotettix virescens*, in the semipersistent manner. TheRTSV RNA consists of about 12.4 kilobases, whichencodes a 393-kilodalton polyprotein that is cleavedinto several smaller proteins. The protein coat is madeof two types of protein molecules. Tungro-infected rice plants are stunted and showmottling and yellow-orange discoloration of the leaves(Fig. 14-44A). Typical tungro symptoms can becaused by RTBV, but they are intensified by the presenceof RTSV. RTSV often occurs alone but causesonly very mild symptoms. The disease caused by RTSValone was earlier known as rice waika disease andthe virus as *rice waika virus*. Also, although bothviruses are transmitted by leafhoppers in the semipersistentmanner, only RTSV can be transmitted alone byleafhoppers, whereas RTBV transmission by leafhoppersis possible only when RTSV is also present in the donorplant.

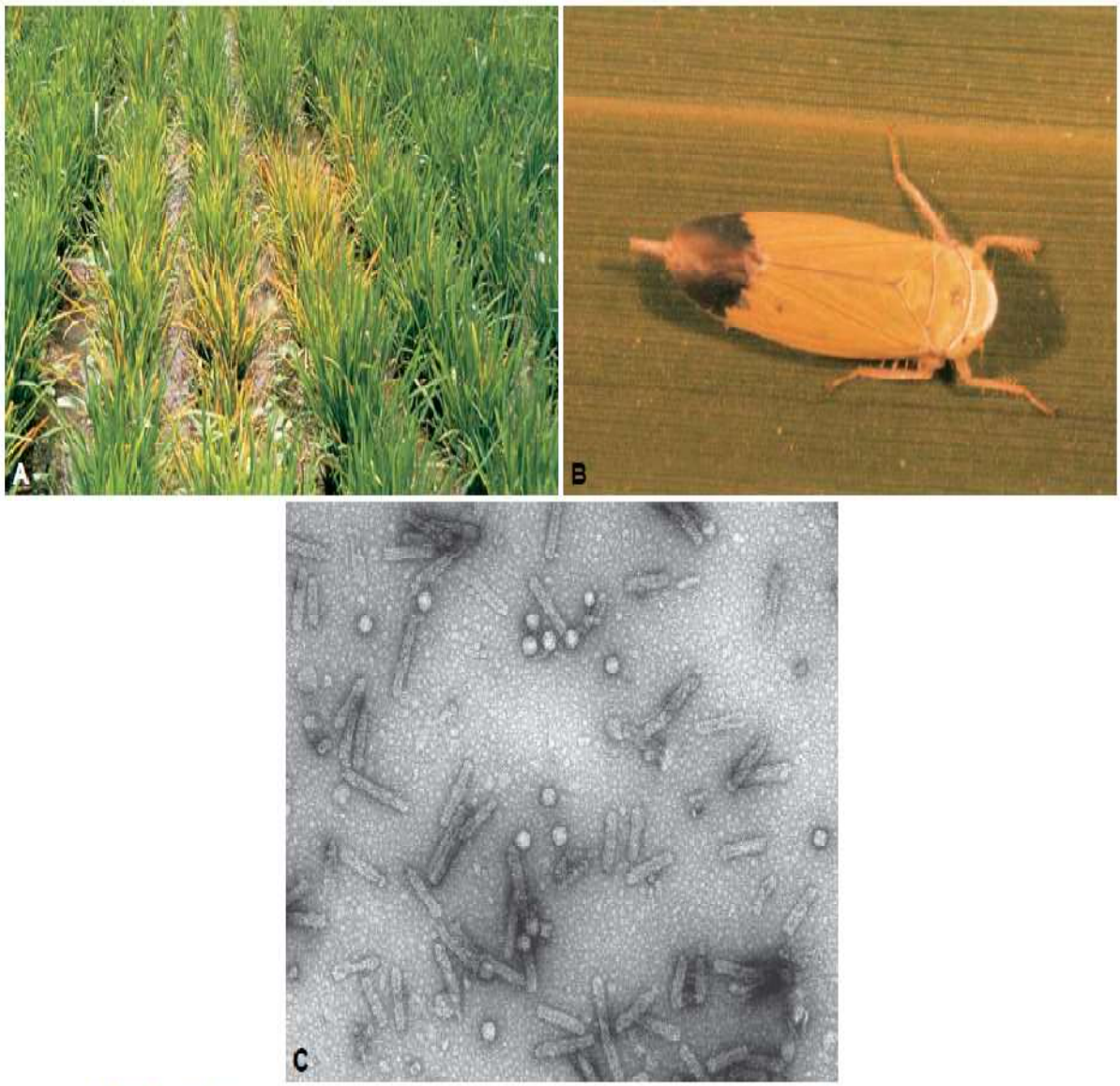



**FIGURE 14-44** (A) Rice tungro-infected rice plants in the field showing stunting and yellow-orange coloration. (B) Female of the leafhopper vector of the tungro viruses. (C) Purified particles of the spherical (waikavirus) and bacilliform (badnavirus) viruses that together cause the rice tungro disease. (Photographs courtesy of H. Hibino.)

**iii. Disease caused bynematode**:

**Root knot of tomato a nematode (*Meloidogyne* spp.):**

Root-knot nematodes occur throughout the world, especiallyin areas with warm or hot climates and short ormild winters, and in greenhouses everywhere. Theyattack more than 2,000 species of plants, includingalmost all cultivated plants, and reduce world crop productionby about 5%. Losses in individual fields,however, may be much higher.

Root-knot nematodes damage plants by devitalizingroot tips and causing the formation of swellings of theroots. These effects not only deprive plants of nutrients,but also disfigure and reduce the market value of manyroot crops. When susceptible plants are infected at theseedling stage, losses are heavy and may result in completedestruction of the crop. Infections of older plantsmay have only slight effects on yield or may reduceyields considerably.

**Symptoms**

Aboveground symptoms are reduced growth and fewer,small, pale green, or yellowish leaves that tend to wiltin warm weather. Blossoms and fruits are few and ofpoor quality. Affected plants usually linger through thegrowing season and are seldom killed prematurely.

Characteristic symptoms of the disease appear on theunderground parts of the plants. Infected roots developthe typical root-knot galls that are two to several timesas large in diameter as the healthy root. Several infections along the root give the roota rough, clubbed appearance. Roots infected by certain

species of the nematode also develop a bushy root

***The Pathogen:*** **Meloidogyne *spp.***

The male and female root-knot nematodes are easilydistinguishable morphologically. The males are wormlike and about 1.2 to 1.5millimeters long by 30 to 36 micrometers in diameter. The females are pear shaped and about 0.40 to 1.30 millimeterslong by 0.27 to 0.75 millimeters wide.

Each female lays approximately 500 eggs in a gelatinoussubstance. The first- and second-stage juvenilesare wormlike and develop inside each egg.

The second-stage juvenile emerges from the egg into thesoil. This is the only infective stage of the nematode. Ifit reaches a susceptible host, the juvenile enters the root, becomes sedentary, and grows thick likea sausage. The nematode feeds on the cells around itshead by inserting its stylet and secreting saliva into thecells. The saliva stimulates cell enlargement and also liquefies part of the contents ofthe cells, which are then withdrawn by the nematodethrough its stylet.

A life cycle is completed in 25 days at 27°C, but it takeslonger at lower or higher temperatures. When the eggshatch, the infective second-stage juveniles migrate toadjacent parts of the root and cause new infections in thesame root or infect other roots of the same plants or rootsof other plants. Most root-knot nematodes are found inthe root zone from 5 to 25 centimeters below the surface.

Root-knot nematodes are spread primarily by water orby soil clinging to farm equipment or on infected propagatingstock transported into uninfested areas.

***Development of Disease***

Second-stage juveniles enter roots behind the root tipand keep moving until they reach positions behind thegrowing point. There, they settle with their head in the developing vascular cylinder. Inolder roots the head is usually in the pericycle. Cells nearthe path of the juveniles begin to enlarge. Two or 3 days

after the juvenile has become established, some of thecells around its head begin to enlarge. Their nucleidivide, but no cell walls are laid down. The existingwalls between some of the cells break down and disappear,giving rise to giant cells. Enlargement and coalescing of cells continuesfor 2 to 3 weeks, and the giant cells invade the surroundingtissues irregularly.

Each gall usually containsthree to six giant cells, which are due to substances containedin the saliva secreted by the nematode in the giantcells during feeding.

The giant cells attract nutrients from surroundingcells and serve as feeder cells for the nematode. Thegiant cells crush xylem elements already present butdegenerate when nematodes cease to feed or die. In theearly stages of gall development the cortical cells enlargein size and, later, they also divide rapidly. Swelling of theroot results from excessive enlargement and division ofall types of cells surrounding the giant cells and fromenlargement of the nematode.

As the females enlargeand produce their egg sacs, they push outward, split thecortex, and may become exposed on the surface of theroot or remain completely covered,depending on the position of the nematode in relationto the root surface.

In addition to the disturbance caused to plants by thenematode galls themselves, damage to infected plants isfrequently increased by certain parasitic fungi, whichcan easily attack the weakened root tissues and thehypertrophied, undifferentiated cells of the galls.

Moreover, some fungi, e.g., *Fusarium*, *Rhizoctonia*, andthe oomycete *Pythium*, grow and reproduce much fasterin the galls than in other areas of the root, thus inducingan earlier breakdown of the root tissues.

***Control***

Root knot can be controlled effectively in the greenhousewith steam sterilization of the soil or soil fumigationwith nematicides. In the field the best control ofroot knot is obtained by fumigating the soil withapproved chemical nematicides. Each treatment usuallygives satisfactory control of root knot for one season.

In several crops, varieties resistant to root-knot nematodesare also available. Transgenic plants producinginhibitors to certain nematode proteinases have shownpromising resistance to the nematode and their use mayprove practical in the future. Several cultural practices,such as crop rotation, fallow soil, soil solarization, andcertain soil amendments, are also helpful in reducingroot-knot losses. Biological control of root knot hasbeen obtained experimentally by treating nematodeinfestedsoil with endospores of the bacterium *Pasteuriapenetrans*, which is an obligate parasite of some plantparasitic nematodes, or with preparations of the fungus*Trichoderma harzianum*; by treating transplants orinfested soils with spores of the fungus *Dactylellaoviparasitica*, which parasitizes the eggs of *Meloidogyne*nematodes; and in some experiments by treatingtransplants or infested soils with spores of the vesiculararbuscularmycorrhizal fungi *Gigaspora* and *Glomus*.

Fairly good experimental control of root knot has alsobeen obtained by mixing essential oils from plant spicesinto nematode-infested soil before planting and throughan increase in plants of their local and systemic-inducedresistance to root knot nematodes by mixing in the soilor spraying the plants with amino-butyric acid and otheramino acids.

**iv. Disease caused by mycoplasma-likeorganism:**

**Symptoms of Little Leaf Disease:**

The main symptom of the disease is the production of very short leaves by affected plant. The petioles are so much reduced in size that leaves appear sticking to the stem. Such leaves are narrow, soft, smooth and yellowish in colour.

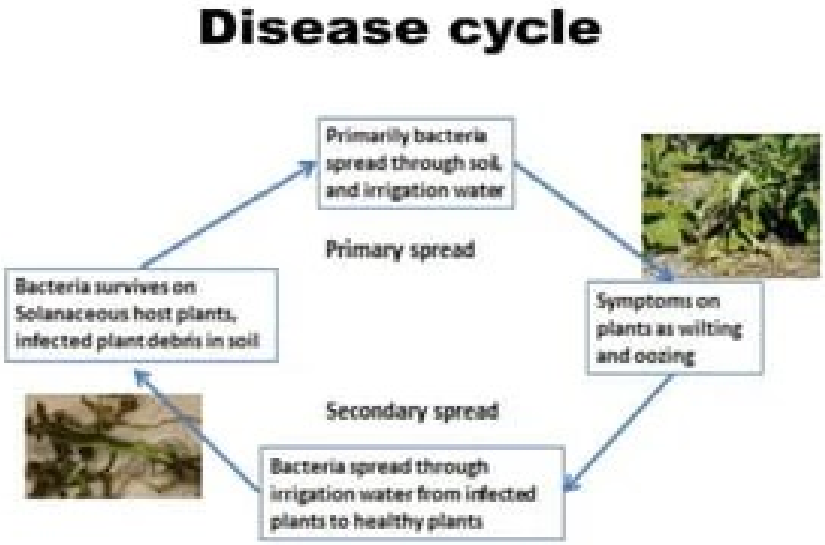
Newly formed leaves are further reduced in size. The internodes are shortened and at the same time large number of axillary buds are stimulated to grow into short branches with small leaves. This gives whole plant a bushy appearance. Usually such plant unable to form flowers. Fruiting is very rare.

**Causal Organism:**

Mycoplasma like organism (MLO).

**Disease Cycle:**

The disease is transmitted through by the vector *Cestius phycitis*. Artificially the disease has been transmitted successfully to tomato, potato and tobacco. Probably during the season of Brinjal crop, the causal agent survives on weed hosts and from there it is transmitted to main crop by its insect vector.



**Control Measures of Little Leaf Disease:**

Since no effective control measure is found it is better to eradicate the weed host and remove the diseased Brinjal plants. Tetra-cycline has been reported to control the disease.

Field sanitation Eradication of volunteer crops/plants previous season and Solanaceous weeds that can carry the disease. Plant barrier crop (Maize) In northern province, plant brinjal in late June or early July in order to avoid high populations of the vectors Plant tolerant variety (Padagoda) Keep plant healthy and vigorous Use good pest management practices to conserve the natural enemies such as lace wing, damsel bug, minute pirate bug Avoid cultivating alternative host plants (e.g. Chilli) to prevent build-up of the vectors Plant in correct spacing (90 X 75 cm)

Observe for small leaves and short internodes at top Witches broom or rosetting Visit daily and observe the mobility of green hoppers Observe under surface of the leaves for vectors

Early destruction of infected plants. Uproot and burn infected plants. Control vector

**Suggested reading**


1. Agrios, George N. (1972). Plant Pathology (5th ed.). AcademicPress.
2. http://www.biologydiscussion.com/
3. https://en.wikipedia.org/wiki/
4. Martinelli, F., Scalenghe, R., Davino, S., Panno, S., Scuderi, G., Ruisi, P., Villa, P., Stroppiana, D., Boschetti, M., Goulart, L.R., Davis, C.E., Dandekar, A.M. (2014). "Advanced methods of plant disease detection. A review". Agronomy forSustainable Development. **35**: 1–25.doi:10.1007/s13593-014-0246-1.
5. Creamer, Rebecca; H. Hubble; A. Lewis (May 2005). "Curtovirus Infection of Chile Pepper in New Mexico". Plant Diseases. **89** (5): 480–486.doi:10.1094/PD-89-0480.
6. https://www.apsnet.org/edcenter/intropp/lessons/fungi/ascomycetes/.../RiceSheath.aspx

**Assignments:**

1. Define inoculum potential
2. What are the major host specific toxins?
3. Explain the role of cell wall degrading enzymes in host pathogen interaction. Discuss different physiological and chemical methods of crop protection Give a short note on structural defense mechanism.
4. What are Pr proteins?
5. Mention two systematic Fungicides and its role
6. Resistant of fungicides
7. Describe symptoms, causal organism, disease cycle and control of red rot of sugarcane disease. What is IPM?
8. Describe about the plant quarantine methods
9. Describe about the disease triangle
10. Different types of diseases cycle caused by deferent pathogens
11. Different stages about disease epidemic
12. Discuss about the different types of fungal toxins
13. Details recognition mechanism and signal transduction during plant-pathogen interaction
14. Describe with schematic diagram of any bacterial disease and its life cycle
15. Describe with schematic diagram of any fungal disease and its life cycle
16. Describe with schematic diagram of any viral disease and its life cycle
17. Mention about the different physical and biochemical mechanism of host defense system
18. Mention about different factors related to disease triangle
19. Mention about the molecular changes happened in host plant after pathogem onfections
20. Describe about the Gene for gene hypothesis; concept of horizontal and vertical resistance
21. Mention about the factors affecting inoculums potential
22. Discuss about the systemic acquired resistance, induced systemic resistance

**All the materials are self written and collected from eBooks, journals and websites.**