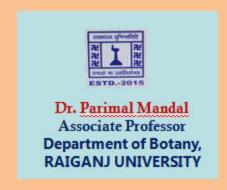
Elective/Special Course: MYCOLOGY and PLANT PATHOLOGY

4103 [Special paper – II (Theoretical)]



1. Biology of Mycorrhizae

 Contents: Diversity of Mycorrhizae and its development, Ectophytic and endophytic mycorrhiza (VAM), mycorrhiza in plant growth promotion, mycorrhizal interactions with soil microorganisms, mycorrhiza in plant disease control.

Mycorrhizae

Plant root symbioses with fungi occur in several different forms and are referred to as mycorrhiza.

(the Greek 'mycos', meaning fungus and 'rhiza', meaning root).

The fungal partner, which cannot photosynthesize benefited by a steady supply of sugar donated by the host plant.

In return, the fungus provides increased surface area to plant for water and nutrient uptake. It also secretes a growth hormone that stimulates roots and branch to grow. The fungus also produces antibiotics that help protect the plant from disease.

Mycorrhizae

- •98% of all plant species have symbiotic fungal partners associated with their root systems.
- •Fungal partners belongs to
 - >Zygomycota hyphae invade root cells
 - ➤ Ascomycota & Basidiomycota hyphae invade root but don't penetrate cells

Extremely important ecological role of fungi

The Two Main Types of Mycorrhizae

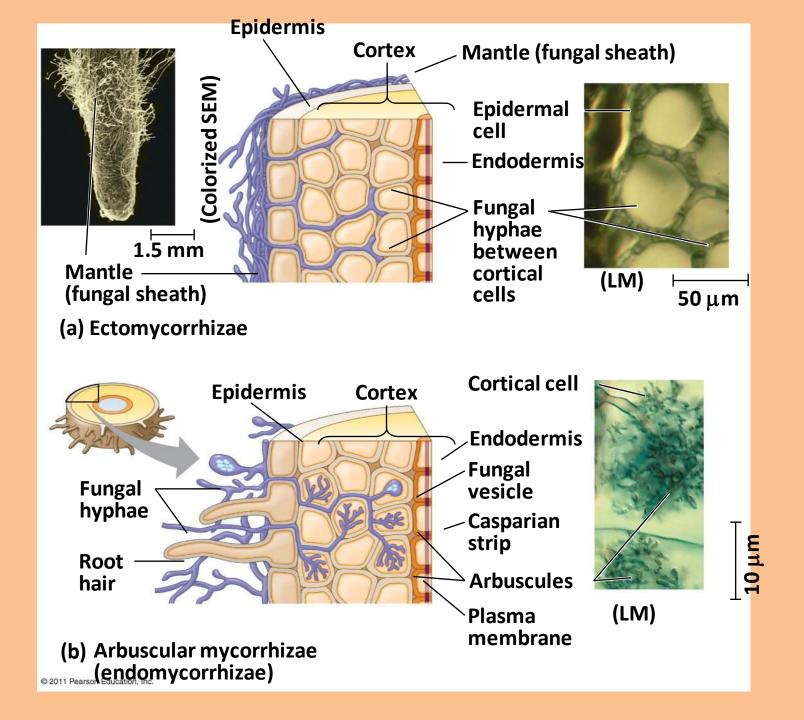
- Mycorrhizal associations consist of two major types
 - Ectomycorrhizae
 - Endomycorrhiza also called as Arbuscular mycorrhizae (AM)/ Vasicular Arbuscular Mycorrhiza (VAM)

Ectomycorrhiza

In ectomycorrhiza, which is predominant on trees in temperate forests, the fungal partner remains outside of plant cells,

whereas in endomycorrhiza, including arbuscular mycorrhiza (AM), part of the fungal hyphae is inside the host cell.

AM is probably the most widespread terrestrial symbiosis and is formed by 70–90% of land plant species with fungithat belong to a monophyletic phylum, the **Glomeromycota**.



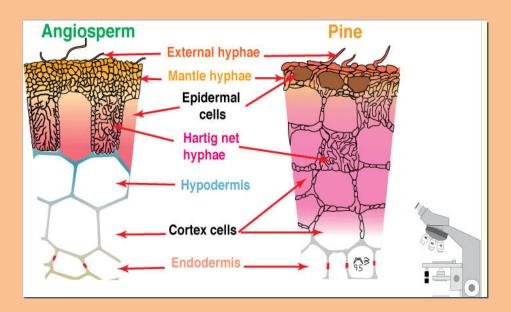
What special is for ectomycorrhiza?

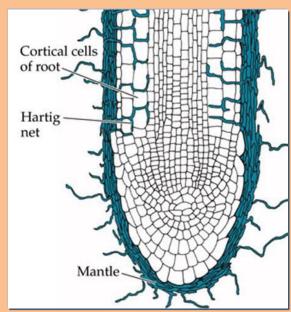
- In ectomycorrhizae, the mycelium of the fungus forms a dense sheath over the surface of the root called fungal Mantle.
- These hyphae form a network in the apoplast, but do not penetrate the root cells
- Ectomycorrhizae occur in about 10% of plant families including pine, spruce, oak, walnut, birch, willow, and eucalyptus
- There is anatomical changes in root. Fungus hyphae form sheath on root tip (called as Mantle) and resulting root hairs die off.
- Nutrient supplies perform by external fungal hyphae,

What special is for ectomycorrhiza?

Hyphae penetrate also cortex cells of the host plant and form complex intercellular system, what call **Hartig's net**.

Function of Hartig net is **nutrient exchange between fungi and host plants**. This structure is characteristic feature only for ectomycorrhiza. This structure is named in honor **of Robert Hartig**, the nineteenth century German mycologist, plant pathologist and forestry scientist, who studied the anatomy of ectomycorrhiza fungi.





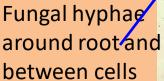
"Ecto" my corrhizae

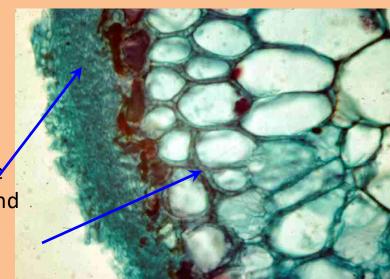


mushroom mycorrhizas on Western Hemlock root



Mycorrhiza cross sections





Ectomycorrhizae

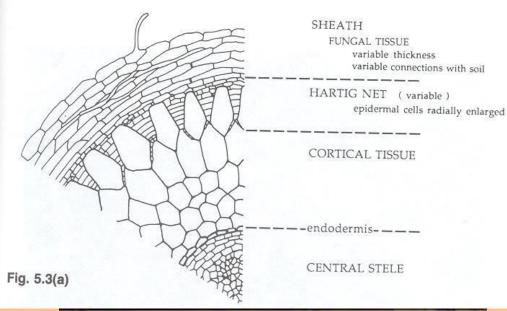


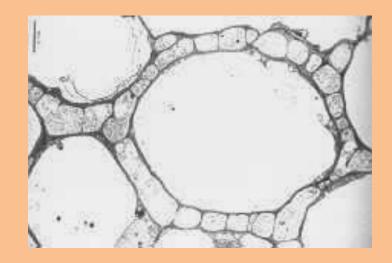


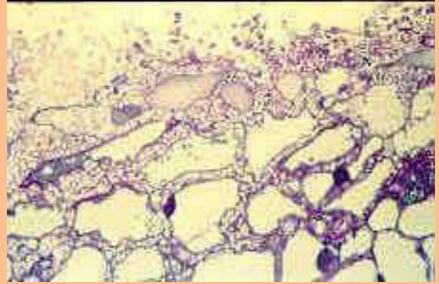


Ectomycorrhizae

- Contains a fungal sheath
- Parenchyma of root cortex is surrounded by hyphae – Hartig net







Ectomycorrhizal fungi



Ascomycete

A = Xylaria sp.

B = Geoglossum glutinosum

C = Aleria rhenana

D = Peziza sp.

E = Helvella sp.*

F = Morchella elata

G = Cordyceps sp.

H = Elaphomyces sp.*

*I = Labyrinthomyces varius**

Ectomycorrhizal fungi













Basidiomycete: Agaricaceae

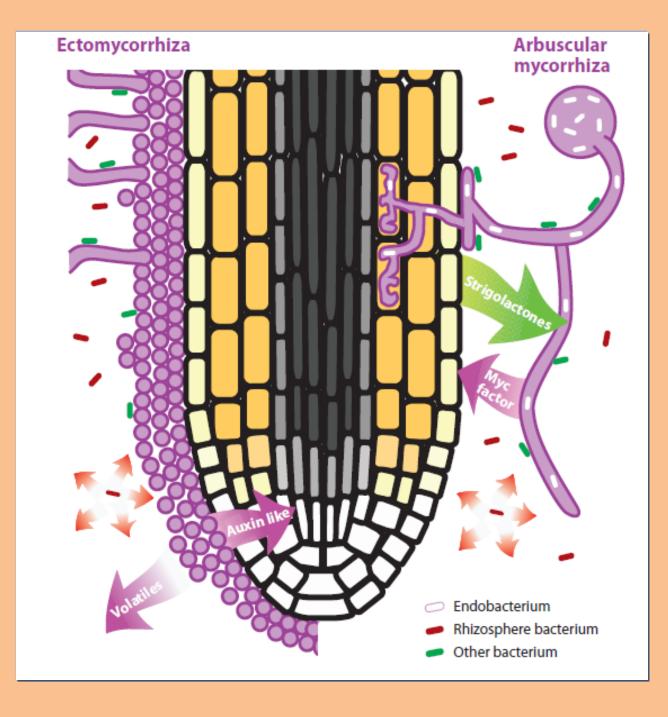
- A. Amanita sp. cf hemibapha*
- B. Amanita sp.*
- C. Amanita sp. * (vaginatae g.)
- D. Amanita muscaria *
- E. Amanita brunneibulbosa *
- F. Limmacella cf. illinata **

Endomycorrhiza / Arbuscular mycorrhizae (AM)/ Vasicular Arbuscular Mycorrhiza (VAM)

Arbuscular mycorrhizal (AM) development and its signaling molecules

TWO signaling molecules for AM development...

- 1. Plant derived Strigolactones
- 2. Fungal derived- 'Myc factors'



Arrows from roots and mycorrhizal fungi illustrate the release of diffusible factors (strigolactones , Myc factors, volatiles, and auxin-like molecules) that are perceived the reciprocal

1. Plant derived - Strigolactones

- ✓AM development is accompanied by an exchange of signalling molecules between the symbionts. A novel class of plant hormones known as strigolactones are exuded by the plant roots.
- ✓On the one hand, strigolactones stimulate spore germination, hyphal branching and increase physiological activity in fungal spores and hyphae.
- ✓On the other hand, they also trigger seed germination of parasitic plants..

In two landmark papers, strigolactones were found to be responsible for the induction of branching and alterations in fungal physiology and mitochondrial activity.

Strigolactones can also stimulate spore germination in some AM fungi. Strigolactones are short-lived in the rhizosphere owing to a labile ether bond that spontaneously hydrolyses in water.

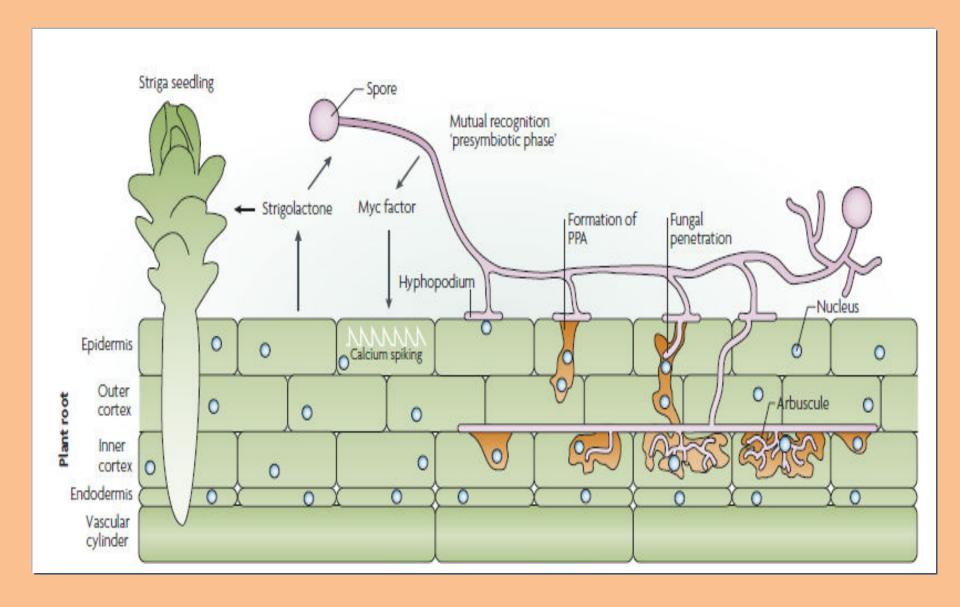
Strigolactones is an novel endogenous plant hormones in diverse angiosperms that range from *Arabidopsis thaliana to* pea and rice, which is characterized by continued by plant

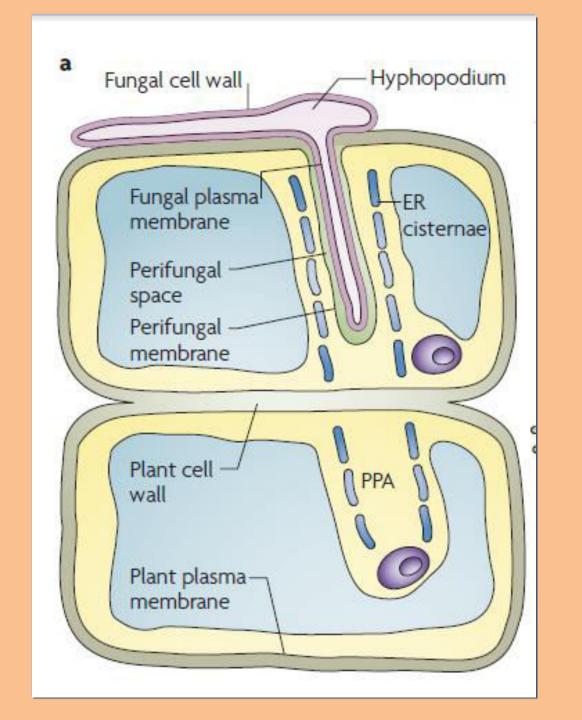
Fungal derived- 'Myc factors

Fungi release signalling molecules, in the form of 'Myc factors' that trigger symbiotic root responses

Fungi produce mycorrhiza (Myc) factors that are operationally defined through their ability to induce calcium oscillations in root epidermal cells and to activate plant symbiosis-related genes.

AM development

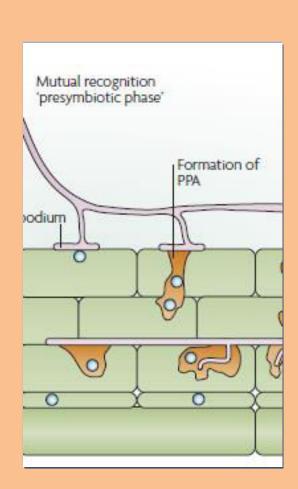




Prepenetration apparatus (PPA)

Formation of the PPA is preceded by a migration of the plant cell nucleus towards the point of anticipated fungal entry. The nucleus then moves ahead of the developing PPA.

The PPA is a thick cytoplasmic bridge across the vacuole of the host cell. It contains cytoskeletal microtubules and microfilaments, which together with dense endoplasmic reticulum cisternae form a hollow tube within the PPA



AM fungi form special types of appressoria called hyphopodia, which by definition develop from mature hyphae and not from germination tubes.

As a consequence of sequential chemical and mechanical stimulation, plant cells produce a prepenetration apparatus (PPA).

The PPA is a subcellular structure that predetermines the path of fungal growth through the plant cell and is formed 4–5 hours after the formation of a fungal appressorium, also called a hyphopodium.

Formation of the PPA is preceded by a migration of

Subsequently, a fungal hypha that extends from the hyphopodium enters the PPA, which guides the fungus through root cells towards the cortex. Here, the fungus leaves the plant cell and enters the apoplast, where it branches and grows laterally along the root axis.

These hyphae induce the development of PPA-like structures in inner cortical cells, subsequently enter these cells and branch to form arbuscules. Vesicles, which are proposed to function as storage organs of the fungus, are sometimes, but not always, formed in AM and are present in the apoplast.

Now opered are typically synthesized systeids of

Colonization by AM fungi is the most complex, leading to the development of hyphopodia (or appressoria) at the root surface, inter- and intracellular hyphae, coils, and characteristic branched structures called arbuscules (little trees) that develop inside cortical cells. These structures, which give their name to the symbiosis, are considered the main site of nutrient exchange between the partners.

Arbuscular mycorrhizal (AM)

AM fungi are unusual organisms because of their age, lifestyle and genetic make-up; they have existed for more than 400 million years morphologically unaltered and could therefore qualify as living fossils.

The hyphal network of AM fungi is usually aseptate and coenocytic, with hundreds of nuclei sharing the same cytoplasm. Likewise, individual spores contain hundreds of nuclei.

There is no confirmed report of a sexual stage in the life cycle of AM fungi, it is possible that genetic material is exchanged and recombined by

Although **spores** of AM fungi can germinate in the absence of host plants, they are **obligate biotrophs**, and therefore depend on a living photoautotrophic partner to complete their life cycle and produce the next generation of spores.

Individual fungal strains exhibit little host specificity when grown with different plants under laboratory conditions. Likewise, a single plant can be colonized by many different AM fungal species within the same root.

- In arbuscular mycorrhizae (AM), microscopic fungal hyphae extend into the root
- These mycorrhizae penetrate the cell wall to form branched arbuscules within root cells
- Hyphae can form arbuscules within cells; these are important sites of nutrient transfer
- Arbuscular mycorrhizae occur in about 85% of plant species, including grains and legumes

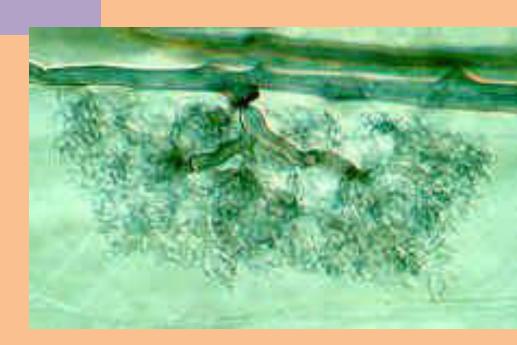
Arbuscular mycorrhiza (AM), a symbiosis between plants and members of an ancient phylum of fungi, the Glomeromycota, improves the supply of water and nutrients, such as phosphate and nitrogen, to the host plant.

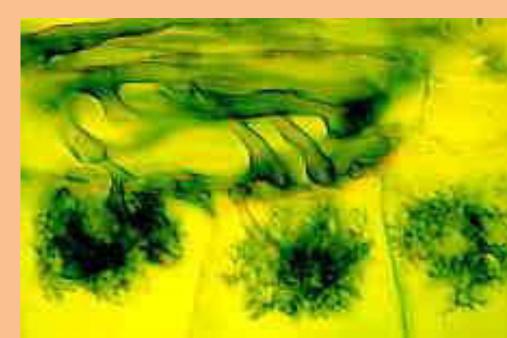
In return, up to 20% of plant-fixed carbon is transferred to the fungus.

Nutrient transport occurs through symbiotic structures inside plant root cells known as arbuscules.

Arbuscules

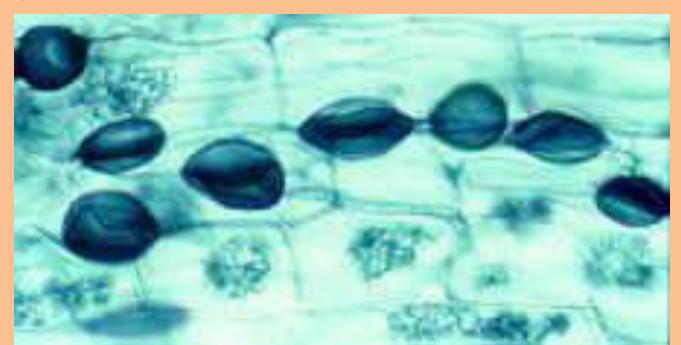
- Intra or inter-cellular
- Typically disintegrate after 2 weeks in plant cell and release nutrients
- Thought to be site of nutrient exchange

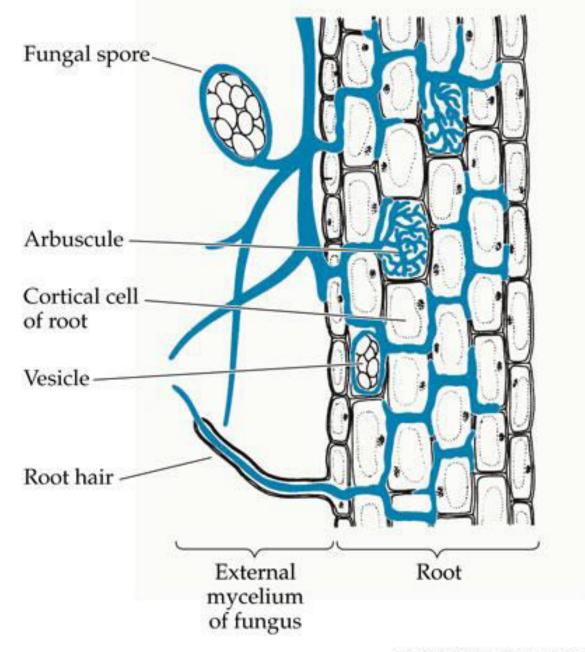




Vesicles

- Intercellular hyphae may also form large swellings – vesicles – at ends of hyphae or intercalary
- Typically rich in lipids & thought to be involved in storage



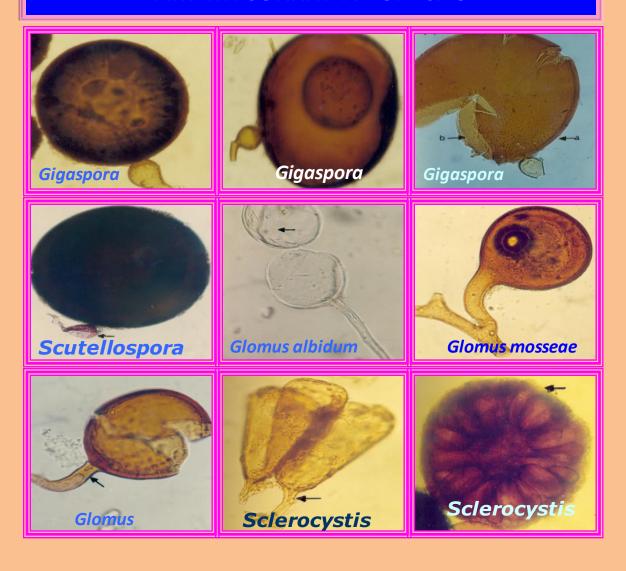


VAM Fungi Zigomycete: Glomales Obligate fungi

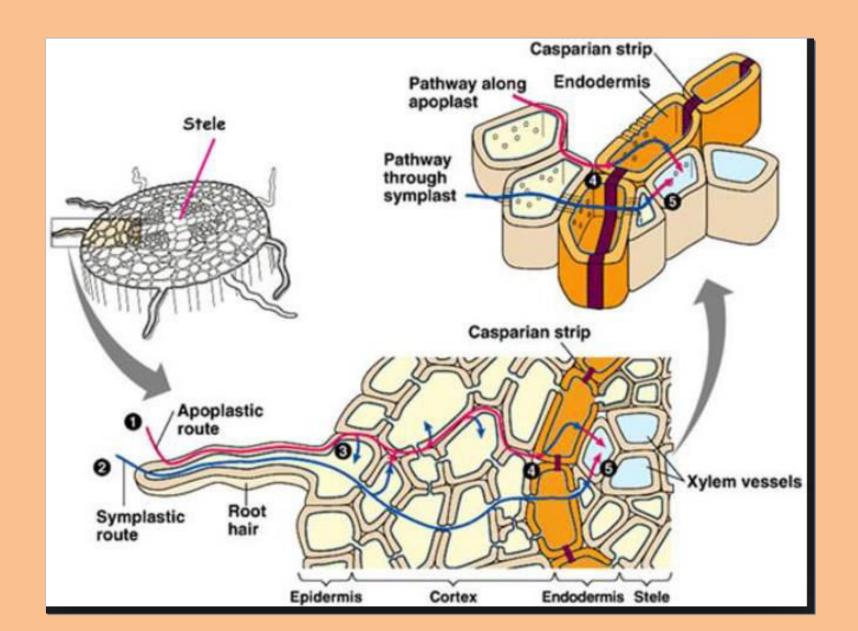
- Glomus
- Acaulospora
- Entrophospora
- Sclerocystis
- Gigaspora
- Scutellospora

Arbuscular Mycorrhizal (AM) Fungi

AM MYCORRHIZAL SPECIES



APOPLASTIC ROUTE and SYMPLASTIC ROUTE



Mycorrhizal importance

- Mycorrhizae have a greater surface area than the roots, so they are able to absorb soil nutrients more efficiently and pass them onto the roots. Almost every plant has some type of mycorrhizae to enhance nutrient uptake.
- In return, the fungi get photosynthetic products from the plants.

How do they impact crops?

Numerous studies have shown that arbuscular my-corrhizal fungi can provide direct benefits to host crops (crops that form arbuscular mycorrhizas) that lead to increased crop productivity.

These benefits include:

- ➤Increased nutrient uptake (especially phospho-rus and zinc)
- >Increased pathogen resistance

Arbuscular mycorrhizal fungi can also have a positive impact on crops by improving the soil quality.

They improve soil quality by binding particles together, resulting in:

- >Improved soil structure
- >Increased water infiltration and retention
- > Reduced risk of soil erosion

Mycorrhizae Importance

- Transfer nutrients to root cells of host plants
- Improve metal uptake
- Improve water uptake
- Break down soil proteins
- Protect roots from toxins
- Protect plant from fungal, bacterial diseases
- Fungal hyphae increase nutrient uptake from soil

Mycorrhizae Importance

- Most abundant endomycorrhizal groups is what is call the vesicular-arbuscular mycorrhizae (VAM), because most produce distinctive mycelial vesicles in the intercellular spaces and arbuscules (i.e. highly branched, finely rooted.
- ➤ Increase nutrient uptake of plant from soil e.g. P nutrition and other elements: N, K, Ca, Mg, Zn, Cu, S, B, Mo, Fe, Mn, Cl
- ➤ Increase diversity of plant
- > Produce uniform seedling
- > Significant role in nutrient recycling

Mycorrhizae Importance

- More tolerant to adverse soil chemical constraints which limit crop production
- Increase plant resistance to diseases and drought
- Stimulate the growth of beneficial microorganisms
- > Improve soil structure
 - Stable soil aggregate hyphal nolysaccharides hind and aggregate soil

Agricultural and Ecological Importance of Mycorrhizae

- Farmers and foresters often inoculate seeds with fungal spores to promote formation of mycorrhizae
- Some invasive exotic plants disrupt interactions between native plants and their mycorrhizal fungi
 - For example, garlic mustard slows growth of other plants by preventing the growth of mycorrhizal fungi

3. Plant defense mechanisms:

 Contents: Plants defense against infection: Preexisting structural and induced structural and chemical defense, hypersensitive reaction, role of phytoalexins and other phenolic compounds,

Defense or Resistance

- Always controlled by genes.
- Non-host Resistance: When a plant resist the attack of pathogenic organism which is otherwise not the host of that pathogen is termed as non host resistance.
 - -e.g. the fungus that causes powdery mildew on wheat (*Blumeria* (*Erysiphe*) graminis f. sp. tritici) does not infect barley and vice versa, the fungus that causes powdery mildew on barley (B. graminis f. sp. hordei) does not infect

Defense Mechanism in Plants

- > Constitutive or Induced
- > Structural (Morphological) defense
- > Biochemical defense

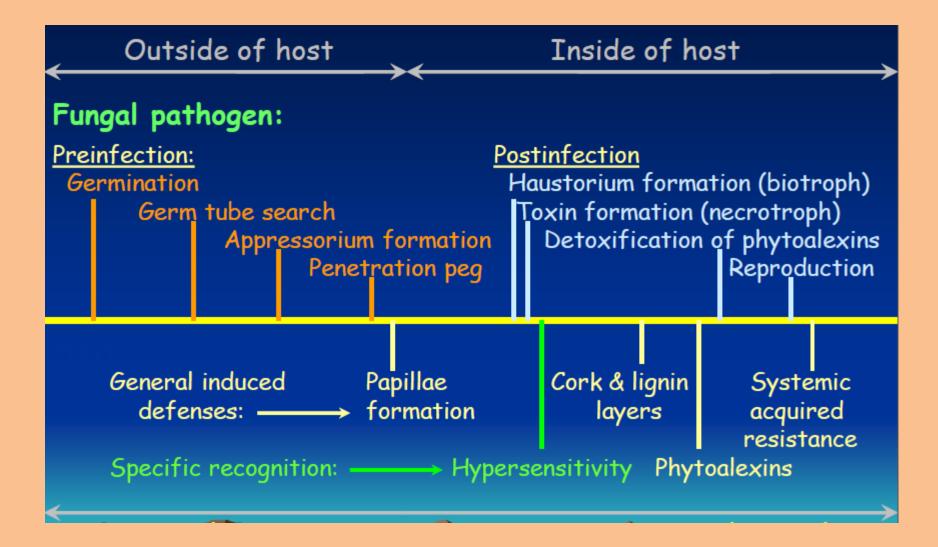
Both the defenses are affected by:

- Age of the plant
- Type of organ infected
- Nutritional status of the host
- Environmental conditions

Host defenses

	Constitutiv e	Induced	
Structural	Biochemica I	Structural	Biochemica I
Wax, cuticle thickness. Hairs. Shape and activity stomata	pre-formed inhibitors, phenolics	cork layer, abscission layer, gums, papillae, tyloses	Hypersensit ivity, Phytoalexin s, systemic acquired of resistance (SAR),

TIME-LINE OF INFECTION



structural CONSTITUTIVE DEFENSES chemical

Plant Defense

Constitutive

- –Physical barriers (Structural)
- –Phytoanticipins (Biochemical)

Inducible

- –Structural
 - Corky, absiccion layers, tylose etc.
- Biochemical
- –Phytoalexins
- –Hypersensitive Response
- Systemic Acquired Response (SAR)

Structural defense

Pre existing/ pre-infectional structural barriers:

Surface barrier

- -Presence of wax layers .
- -Hairs
- -Lignin
- -Thickness of cuticle

The thickness and toughness of the outer wall of epidermal cells are apparently important factors in the resistance of some plants to certain pathogens.

- -Size of stomata (Resistant citrus vars. have small stomata- X. campestris),
- Location and shape of stomata, opening & closing (resistant wheat vars. Rusts).

Post infectional defense structures

Histological defense

- Tylose formation e.g. wilts
- •Corky layers e.g. in potato- R. solani
 - Cellular defense altered walls of cells (changes in cell wall- thickening, deposition of callose papillae)
 - -Cytoplasmic defense
 - -Hypersensitive responses

Hypersensitive response

- Result in sudden death of the host cells in the vicinity of the pathogen
- Highest degree of resistance in both structural & biochemical in nature.
- The hypersensitive response is initiated by:
- the recognition by the plant of specific pathogen-produced signal molecules, known as elicitors.
- recognition of the elicitors by the host plant activates a cascade of biochemical reactions in the attacked and surrounding plant cells that lead to
 - new or altered cell functions and to new or greatly

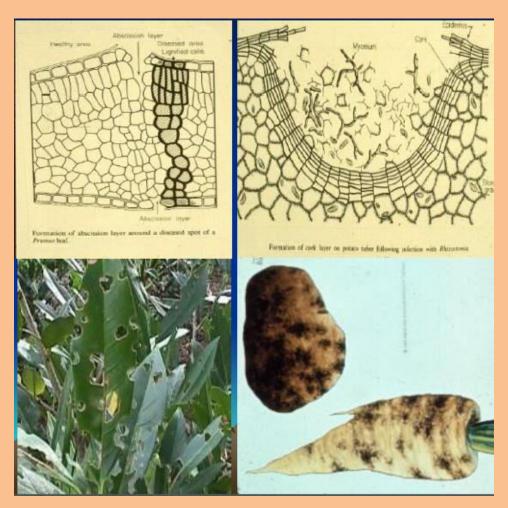
Hypersensitive response

- -the most common new cell functions and compounds include:
 - a rapid burst of reactive oxygen species (ROS), leading to a dramatic increase of oxidative reactions;
 - increased ion movement, especially of K+ and H+ through the cell membrane;
 - disruption of membranes and loss of cellular compartmentalization
- -Production of antimicrobial substances such as phenolics (phytoalexins); and formation of antimicrobial so-called pathogenesis-related proteins (PR Proteins) such as Chitinase, peroxudase, b-1,3-Glucanase etc.

·Due to HR

- The necrotic tissues isolate the obligate pathogen from living cells.
- Devoid the pathogen of nutrition, thus starved and die.

Induced structural defenses



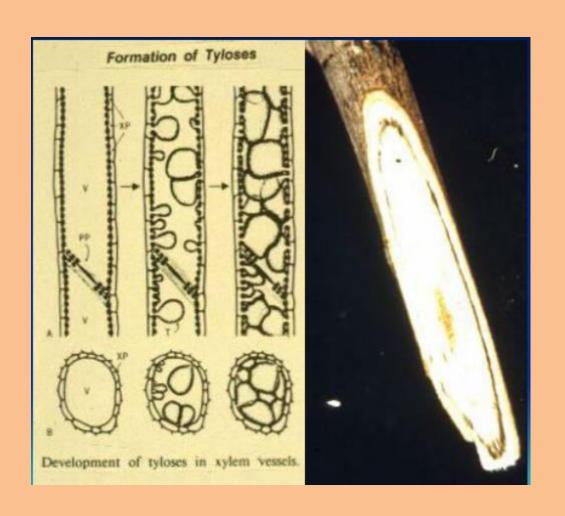
Abscission layer
X. pruni- shot hole
canker

Cork layer R. solani-

The formation of cork or abscission layers can limit the size of lesions, and consequently the extent of damage that can be caused by a single infection Provide protection by inhibiting the further spread of pathogen.

-Block the spread of toxic substances of the pathogen,

Induced structural defense



Tyloses are overgrowths of living cells that protrude via pits into xylem vessels blocking the vascular system.

If they form abundantly and quickly, they can stop the spread of vascular wilt pathogens.

Biochemical defense

Pre-existing biochemical defense

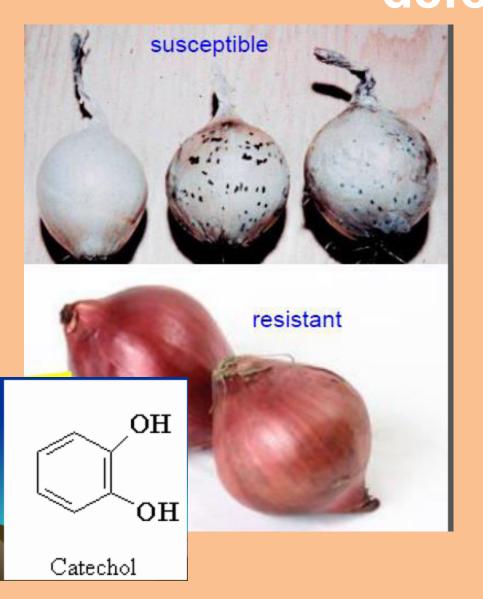
- -Inhibitors released by the plants in its environment
- Fungitoxic exudates of tomato and sugarbeet inhibit germination of Botrytis and Cercospora;
- Oil capric acid on elm seed toxic to Ceratocystis ulmi
- Phenolics: red scale onion against smudge-Colletotrichum circinans

Inhibitors present in plant cells

- Stored in vacuoles, lysogenous glands, wood periderm of plants
- Phenolics onion (catechol & portocatechoic acid)
- Saponins (tomatin in tomato, avenacin in oats (most of these compounds inhibits hydrolytic enzmes produced by pathogen e.g. pectolytic enzymes etc.

-Some plants also contains hydrolytic

Constitutive biochemical defense



In contrast to red and yellow onions, white onions do not contain significant quantities certain phenolic chemicals (one is catechol). present, these phenolics confer resistance to onion

Defense through lack of essential factors

Lack of recognitions

 Lack of host receptor and sensitive sites for toxins in the host.

Lack of essential nutrients for the pathogen

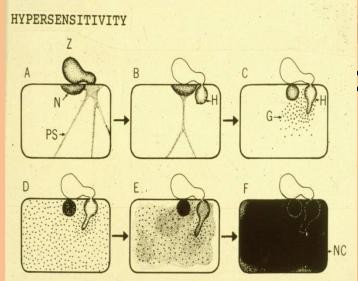
Post infectional biochemical defense

- •HR- hypersensitive response
- Biochemical inhibitors produced in response to injury by pathogen
 - -Phenolics (Chlorogenic acid, caffeic acid)
 - -Oxidation products of plants (floretin, hydroquinonesm hyroxytyromine)
 - -Phytoalexins

Induced biochemical defenses

Induced biochemical defenses hypersensitive response

The hypersensitive response (HR) is a localized death of host cells at the site of infection. It is the result of a specific recognition of a pathogen attack by the host.



de Teel HRact by a type of death.

Jeal Holation of the pathogen
-Stop flow of nutrients- thus starvation

Defense through increased level of phenolics

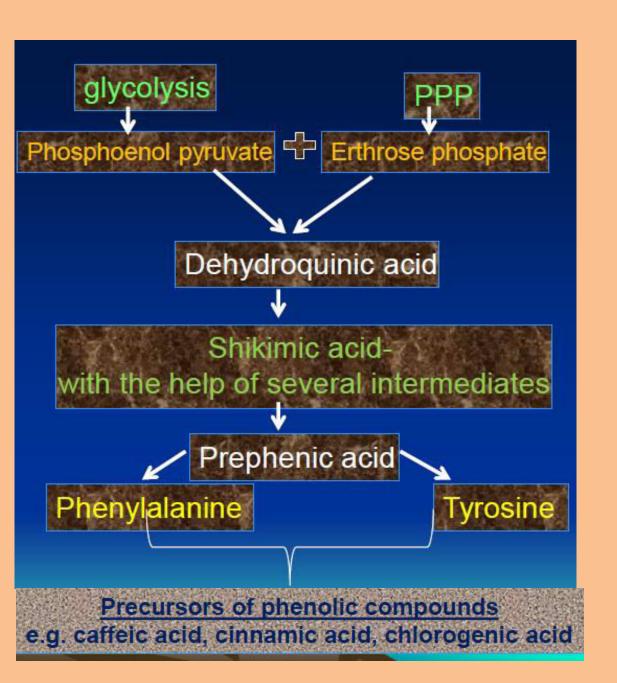
 Plants contains aromatic acids that act as precursors of phenolic compounds

Simplest compound is phenol: having a single

OH group on benzene ring



- When benzene ring has more than one OH group
 - polyphenols (lignins, phenyl glucoside, flavanoids, anthocyanin etc.
- -Quinone O Quinone



In plants, phenolic synthesis takes place through shikimic acid pathway and acetic acid pathway- PPP (PPPthis operates in diseased plants), Shikimic acid pathway is more important. These products formed Glycolysis and pentose phosphate pathway (PPP). **Enzymes** participating in these

Role of phenol oxidizing enzymes

Enzymes like

- -poplyphenol oxidases- oxidises phenols to quinones
- -Peroxidases increases the polymerization of the phenols into lignins- the complex phenols

Phenolic compounds produced by plants

- Caffeic aid: sweet potato
- Chlorogenic acid: sweet potato, potato, carrot
- Phloretin : Apple
- Hydrogunones: sweet potato

Phytoalexins

- Concept given by Borger & Muller in Potatolateblight interactions
- Defined as antibiotics produced in plantpathogen interaction or as a response to injury or physiological stimuli (Kuc, 1972)
- Paxton (1981) defined phytoalexins as low mol. Wt. antimircobial compounds which are synthesied by and accumulates in plant cells after microbial infection.
- Not produced during biotrophic infection
- Many phytoalexins have been isolated from plants (>20) families.
- -E.g. leguminosae, solanaceae, malvaceae,

Characteristics of phytoalexins

☐ Should be **fungistatic & bacteristatic** and active at very low conc. ☐ Produced by the host in response to infection or metabolic by products of micro-organisms and stimuli. ☐ Absent in healthy cells or present in very minute quantity. ☐ Usually remain close to the site of their production ☐ Produced in quantities proportionate to the size of inoculum ☐ Produced in large quantity in response to weak pathogen or non pathogen than virulent one.

☐ Produced relatively quickly in cells after infection

Best studied phytoalexins

are:

Legumes

- -Phaseollin; Common beans
- -Vestitol: Red clover
- -Phaseollidin: Common bean
- -Medicarpin; alfalfa, lucern, beans, chickpea
- -Pisatin: Pea

Solanaceae

- -Rishitin: potato, tomato
- -Capsidol: chilli/ pepper
- -Phytubrin: potato

•Graminae

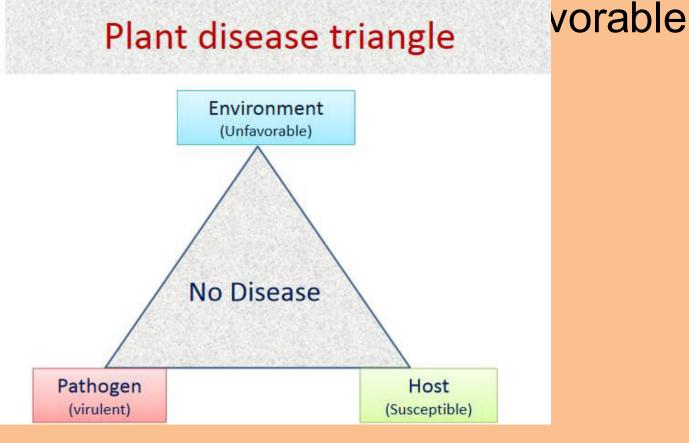
- -Avenalumin-I,II, III: Oats
- •6-mehtoxymellin: carrot
- Orchidaceae
 - -Orchinol: Orchid

7. Plant Diseases

Disease Development

 The classic disease triangle establishes the conditions for disease development, i.e. the interaction of a susceptible host, a

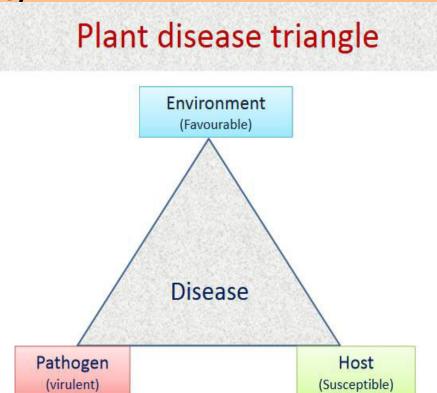
virule enviro



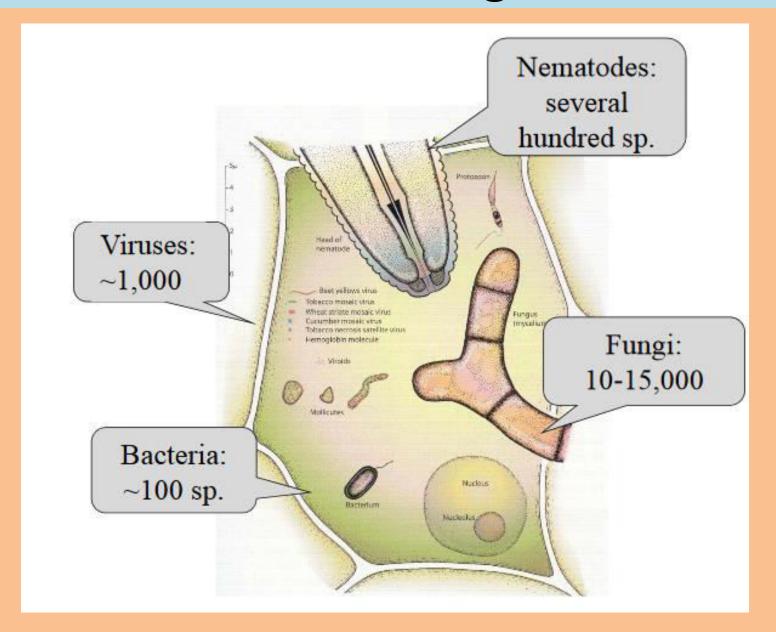
Disease Development

1. Pathogens (virulent)

- Fungi
- Viruses
- Bacteria
- Phytoplasma
- Nematodes
- Parasitic higher pla
- 2. Host –susceptible
- 3. Environment-favourable



Plant Pathogens



Assessment of losses

- At least 10-30% of global food production
- Representing a threat to food security
- Agrios (2004) estimated that annual losses by disease cost US\$ 220 billion.

Important diseases of wheat (Triticum aestivum L.

Rusts of Wheat -caused by the fungus Puccinia

- > Stem or black rust- Puccinia graminis f. sp. tritici
- Brown rust or leaf rust- Puccinia triticina (Syn. P. recondita) and
- Yellow or stripe rust-Puccinia striiformis f. sp. tritici.

Smuts of Wheat

Bunts -caused by the smut fungus Tilletia

- Hill bunt or Common bunt or stinking smut or covered smut- two very closely related fungi, <u>Tilletia tritici</u> (syn. <u>Tilletia caries</u>) and <u>T. laevis</u> (syn. <u>T. foetida</u>).
- Karnal or Partial bunt-Tilletia indica (Syn. Neovossia indica)
- Loose smut -Ustilago nuda (Syn. U. segetum)
- Flag smut-Urocystis agropyri

Powdery mildew of wheat-Blumeria graminis (DC.) Speer

Tundu disease of wheat- Calvibacter tritici associated with ear cockle disease (caused by the nematode Anguina tritici.

Puccinia Life Cycle

- Puccinia is an obligate parasite.
- It is a heteroecious parasite.
- It's life cycle on Wheat and onBarberry.
- Wheat is a primary host.
- Barberryisasecondaryoralternate host.

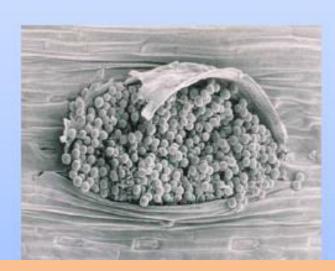
- DISEASE: Stem rust (black rust or cereal rusts)
- PATHOGEN: Puccinia graminis f. sp. tritici
- HOSTS: Wheat, barley and common
- Barberry (Berberis, Mahoberberis and Mahonia spp.)

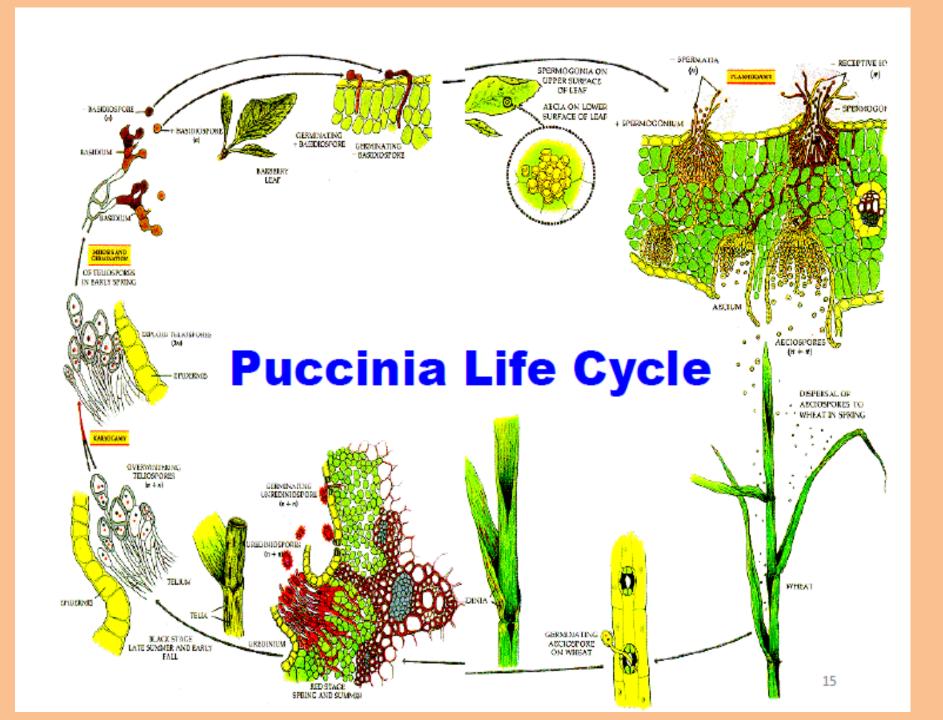
Symptoms and Signs

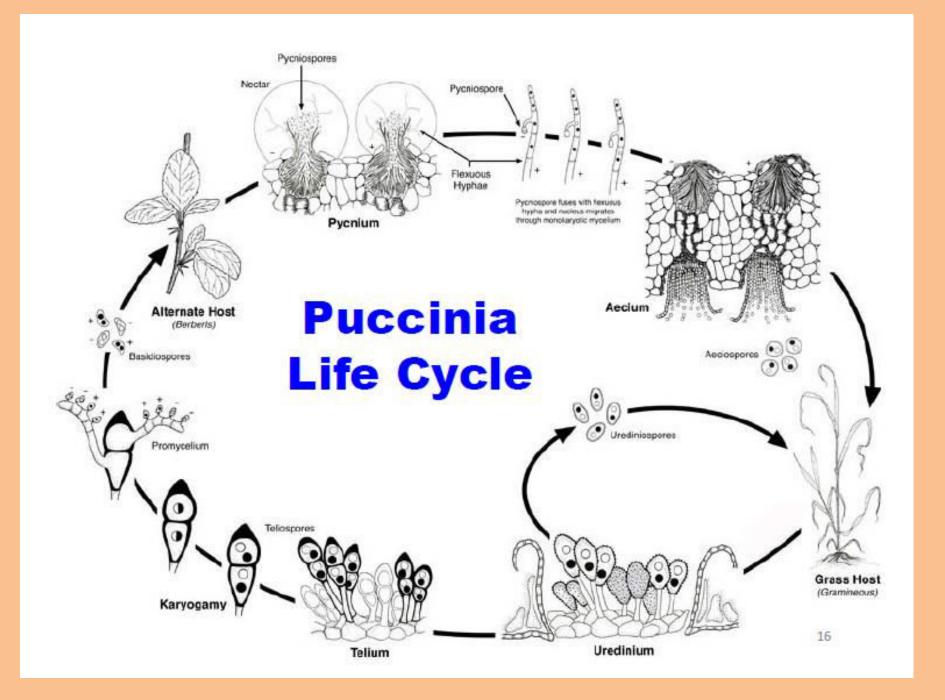
On wheat and other grass hosts:

- ➤ After 7 to 15 days infection
- Elongated to irregular shaped pustules (uredinia) of brickred urediniospores break through the epidermis and develop on the upper leaf surfaces, leaf sheaths, glumes and awns.

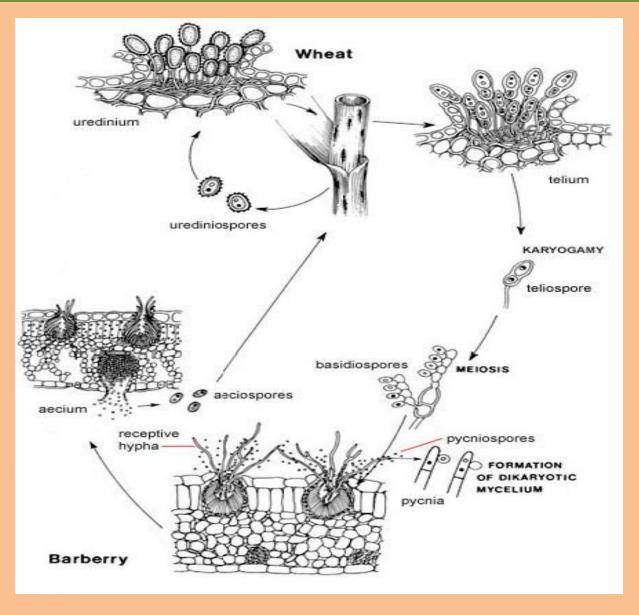


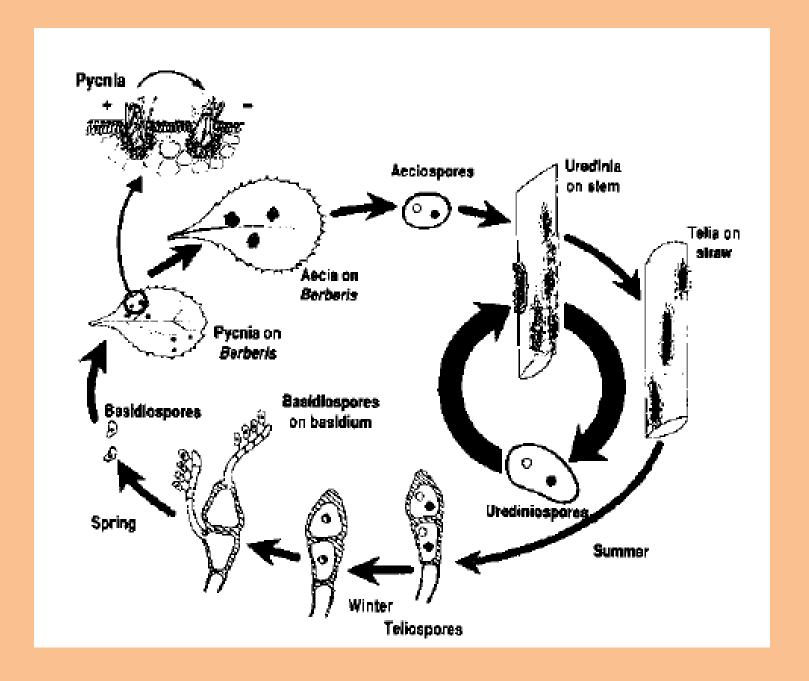




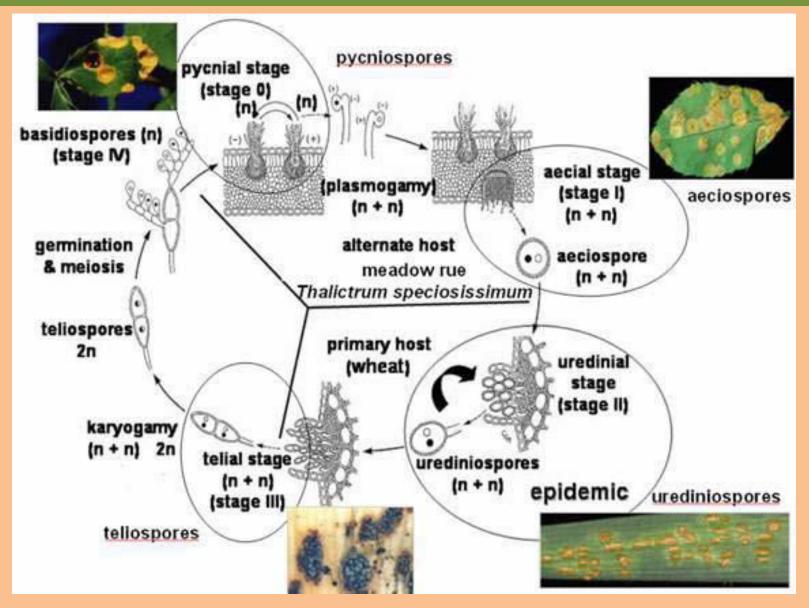


Disease Cycle and Epidemiology





Stages of rusts in wheat



- Rusts can produce up to five spores types during their life cycle
- 0-Pycniospores (Spermatia)-Haploid gametes in heterothallic rusts.
- I-Aeciospores-non-repeating dikaryotic vegetative spores
- Il-Urediniospores-repeating dikaryotic vegetative spores. These spores are referred to as the repeating stage because they can cause auto-infection (re-infect the same host from which the spores were borne). These spores are red/orange and are a characteristic sign of rust fungus infection.
- III-Teliospores-Diploid spores that produce basidia and are the survival stage of life cycle

Management of rusts of wheat

- Prevention, early warning and rapid response
- Using resistant cultivars and early intervention are the key principles of controlling wheat rust diseases.
- Ug99 is a virulent strain of stem rust which emerged in Uganda in 1998-1999
 Most of the wheat varieties are susceptible to this strain
- FAO in collaboration with CIMMYT provide policy and technical support to the

Cultural practices

- Grow early maturing resistant wheat varieties.
- Appropriate spacing and fertilizer (N) application

Chemical control

- Prophylactic sprays of Mancozeb (Dithane Z- 78) @ 0.25%
- Fungicides that inhibit the synthesis of sterols [i.e., sterol biosynthesis inhibitors-SBIs] like Tilt (Propioconazole) 25 EC @ 0.1% at 15 days intervals

Smuts of Wheat

- Loose Smut of Wheat
- Hill Bunt of Wheat
- Karnal Bunt of Wheat
- Flag Smut of Wheat

 The black powdery spores (Teliospores) are typical of smut diseases, which also includes bunt.

Loose Smut of Wheat

- DISEASE: Loose Smut
- PATHOGEN:
 Ustilago nuda var.
 tritici. (Syn. U.
 segetum var.
 tritici)
- HOSTS: Wheat, barley and common



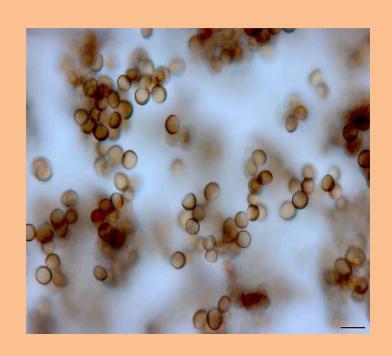
Symptoms and Signs

 Every ear of the is affected plants converted into a black mass of spores and no grains are formed. The infected heads emerge from the boot one to three days earlier than those of healthy plants. As a rule, all the glumes (chaff) and grain in a smutted head

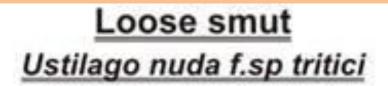


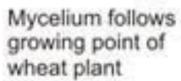
Teliospores

 This sooty mass is composed almost entirely of millions of teliospores. The spores are quickly dispersed by the wind during the flowering period of normal heads, and by harvest only an erect bare rachis remains. All of the floral parts of the head, except for the rachis and pericarp membrane, are invaded by mycelium of the fundue and converted



Disease Cycle of Loose Smut

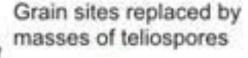


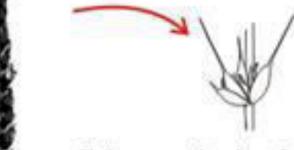


Mycelium invades the grain sites

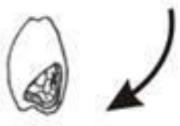


Mycelium invades young seedlings





Teliospores land on flowers of healthy plants and infect developing grain



Mycelium invades part of embryo in seed

Management of Loose Smut of Wheat

Disease control:

 The disease is internally seed borne and as such spray of fungicides is not effective in controlling the disease.

Seed treatment

Use of resistant varieties:

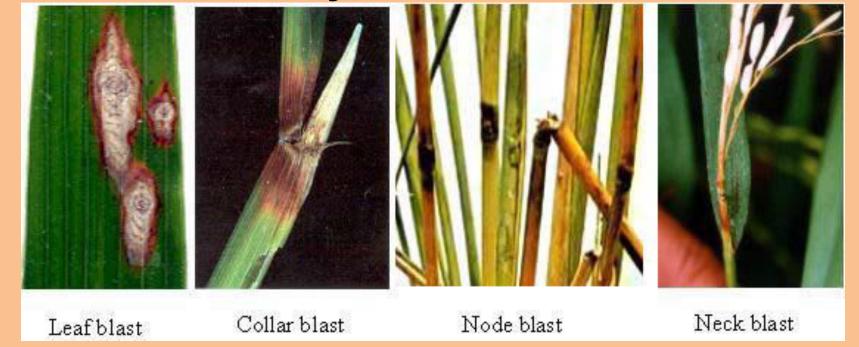
 The most successful method is the use of resistant varieties.

Crop rotation:

 Crop rotation at suitable intervals is also effective in disease control.

Blast of Paddy

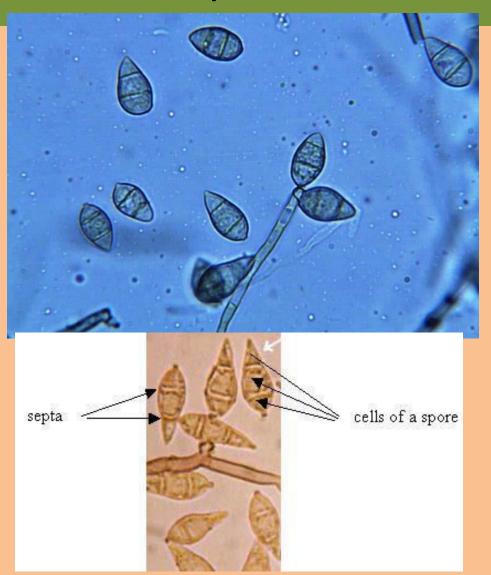
- DISEASE: Blast of Paddy
- PATHOGEN: Pyricularia grisea (Magnaporthe grisea)
- HOSTS: Paddy



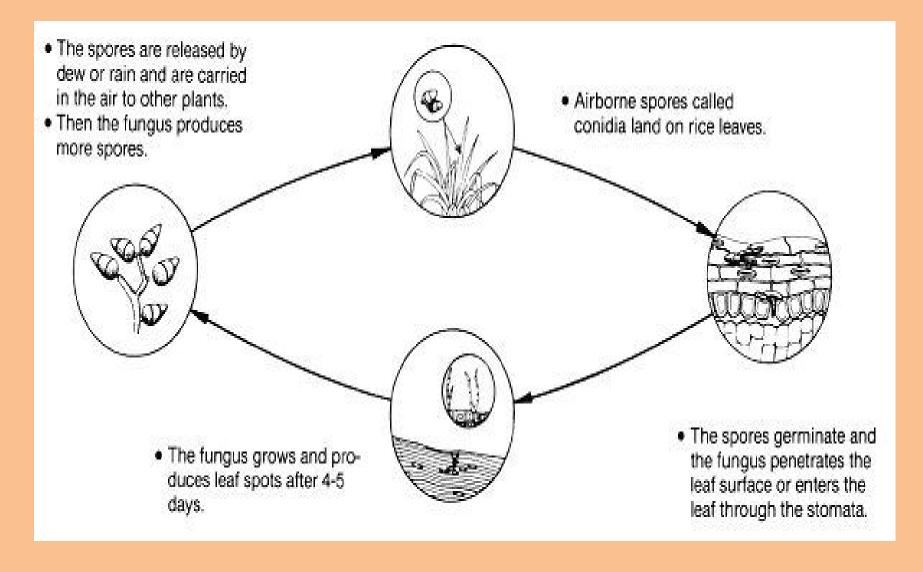
Symptoms and Signs

 Blast of Paddy: Blast symptoms can occur on leaves, leaf collars, nodes and panicles. Leaf blast, the lesions are elliptical or spindle shaped with brown borders and gray centers. Collar blast or node blast occurs when the pathogen infects the collar that can ultimately kill the entire leaf blade. Neck blast occurs when the pathogen infects the neck of the panicle. The infected neck is girdled by a grayish brown lesion and the panicle falls over if the infection is severe. If neck blast occurs before the milk stage, the entire panicle may die prematurely, leaving it white and completely unfilled. The fungus produces two toxins-upicolinic acid and piricularin

Microscopically pyriform (pear-shaped) conidia are usually three-celled or 2 septate borne on conidiophores



Life cycle of Blast of Paddy



Management of Blast of Paddy

- Use of resistant cultivars.
- Efficient use of nitrogen fertilizers.
- Soil amendment with silica.
- Spray Beam (tricyclazole) melanin biosynthesis inhibitor fungicide for effective control

Brown spot of Paddy

- DISEASE: Brown spot of Paddy
- PATHOGEN: Bipolaris oryzae (Breda de Haan) Shoemaker (Syn. Helminthosporium oryzae)

HOSTS: Paddy





 The fungus may also infect the glumes, causing dark brown to black oval spots, and may also infect the grain, causing a

At the point of attachment to a conidiophore

with a minute slightly protographic details and the second details are second details and the second details are second details and the second details and the second details are second details and the second details and the second details are second details and the second det

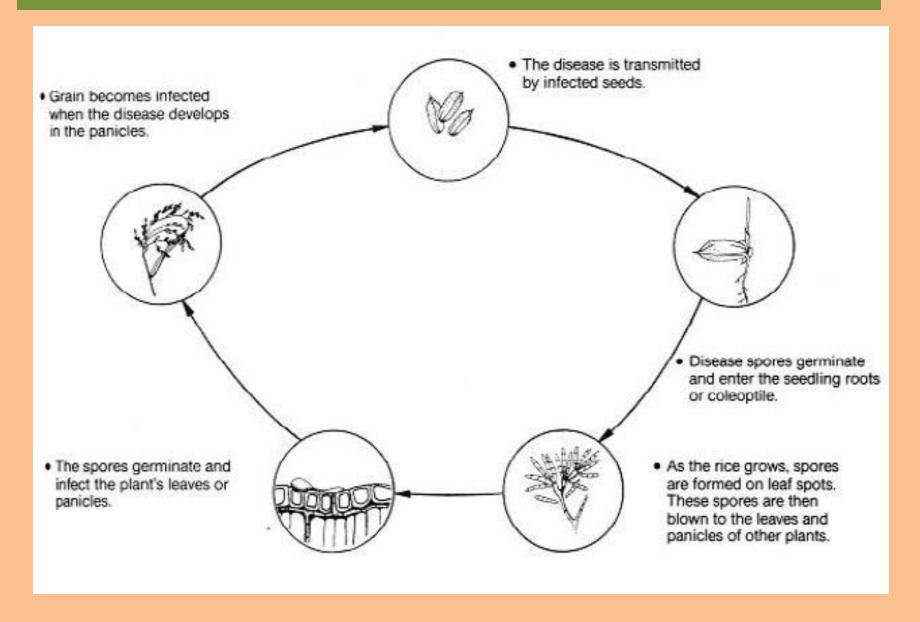
with a minute, slightly protruding hilum (dot).

Conidia are generally curved, boat, or club-shaped, with 6 to 14 transverse septa or cross walls

Historical Events

Bipolaris oryzae (Breda de Haan) Shoemaker (anamorph) **Drechslera oryzae** (Breda de Haan) Subramanian & P. C. Jain (synonym) Helminthosporium oryzae Breda de Haan (synonym) Cochliobolus miyabeanus (Ito & Kuribayashi) Drechsler ex Dastur (teleomorph) is a <u>fungus</u> that causes <u>brown spot</u> disease in <u>rice</u>. This disease was the causal agent of the Bengal famine of 1943. It was used by the <u>USA</u> as a <u>biological weapon</u> in Japan during World War II

Life cycle of Brown spot of Paddy



Management of Brown spot of Paddy

- Use disease free seed
- Use of resistant varieties.
- Avoid excesses of N -application
- Use of silicon fertilizers (e.g., calcium silicate slag)
- Since the fungus is seed transmitted, a hot water seed treatment (53-54°C) for 10-12 minutes. And controls primary infection at the seedling stage.
- Seed treatment with captan, thiram or carbendazim.
- Seed treatment with tricyclazole followed by

Sheath blight of rice

- DISEASE: Sheath blight of rice
- PATHOGEN:
 Rhizoctonia solani
 Kuhn
- HOSTS: Paddy

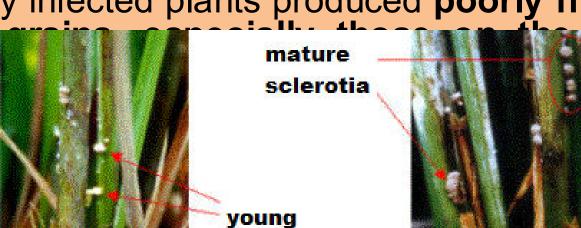


Symptoms and Signs

- Initial lesions asmall, ellipsoidal or ovoid, greenishgray band watersoaked and usually develop near the water line in lower leaves.
- Older lesions are elliptical or ovoid with a grayishv white center and light brown to dark brown margin.
- Lesions may coalesce forming bigger lesions with irregular outline and may cause the death of the whole leaf.

Severely infected plants produced poorly filled or

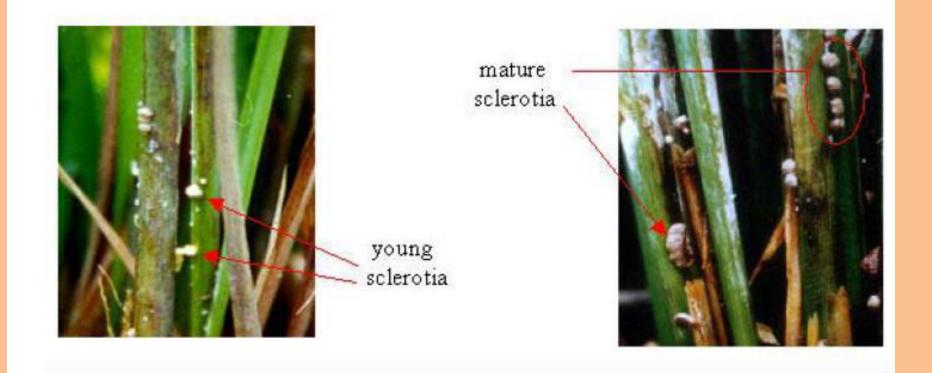
empty portion fungi ai plant.



sclerotia

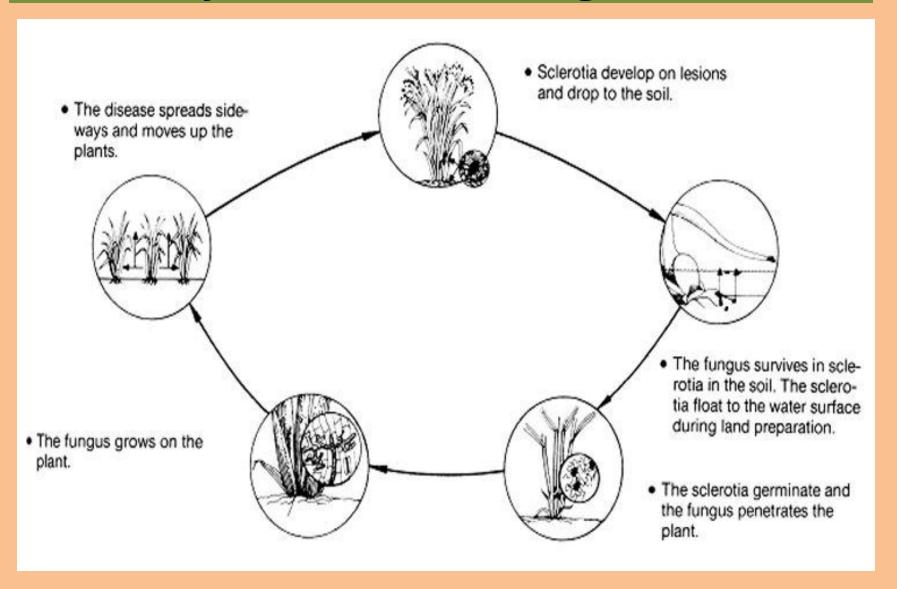
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Sclerotia on the soil or floating on the water are assumed to be the sole source of inoculum in temperate regions.

Life cycle of Sheath blight of rice



Management of Sheath blight of rice

Host resistance:

No resistant cultivars.

Cultural practices:

- General sanitation-destruction of crop debris.
- Flooding of rice fields.
- Proper spacing.
- Efficient use of N-fertilizers.

Chemical control:

Spray of Tilt (Propioconazole) ,a combination of fungicides (Azoxystrobin 18.2% +
 Difenoconazole 11.4% SC) was found highly effective against rice sheath blight.

False smut or green smut of Paddy

- DISEASE: False smut or green smut of Paddy
- PATHOGEN: Ustilaginoidea virens (Cooke)
 Takah (anamorph), Claviceps oryzae-sativae Hashioka (teleomorph)
- HOSTS: Paddy



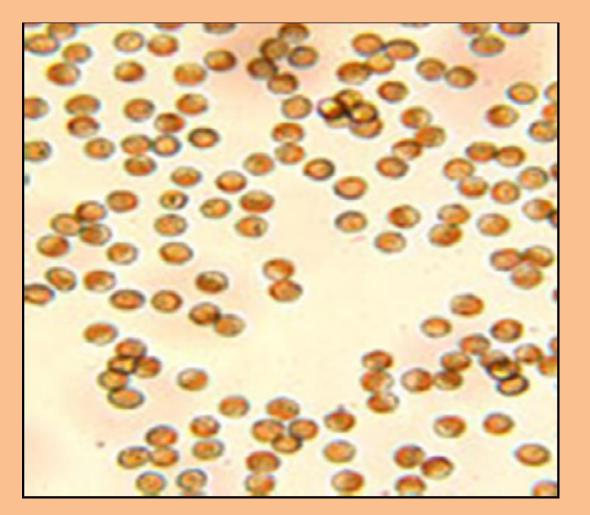


Symptoms and Signs

- Individual rice grain transformed into a mass of yellow fruiting bodies.
- Growth of velvety spores that enclose floral parts.
- Only few grains in a panicle are usuall infected



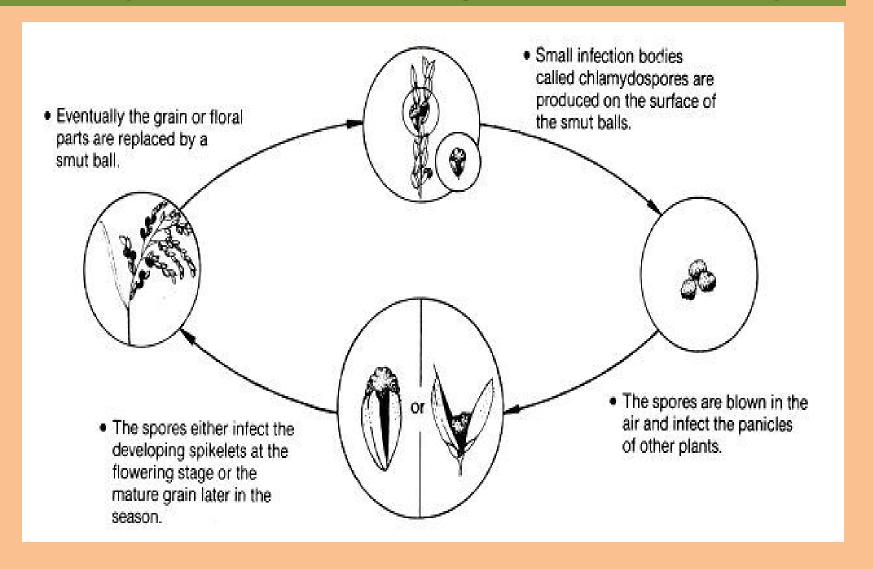




Conidia

The causal organism is a fungus. Microscopically the chlamydospores or the conidia of the fungus are spherical to elliptical. They are pale and almost smooth when young, volivaceous and warty when mature.

Life cycle of False smut or green smut of Paddy



Management of False smut or green smut of Paddy

- Removal and proper disposal of infected plant debris.
- Use of disease-free seeds that are selected from healthy crop.
- Seed treatment with carbendazim 2.0g/kg of seeds.
- Spraying of copper oxychloride at 2.5 g/litre or Propiconazole at 1.0 ml/litre at boot leaf and milky stages will be more useful to prevent the fungal infection.

Bacterial blight of rice

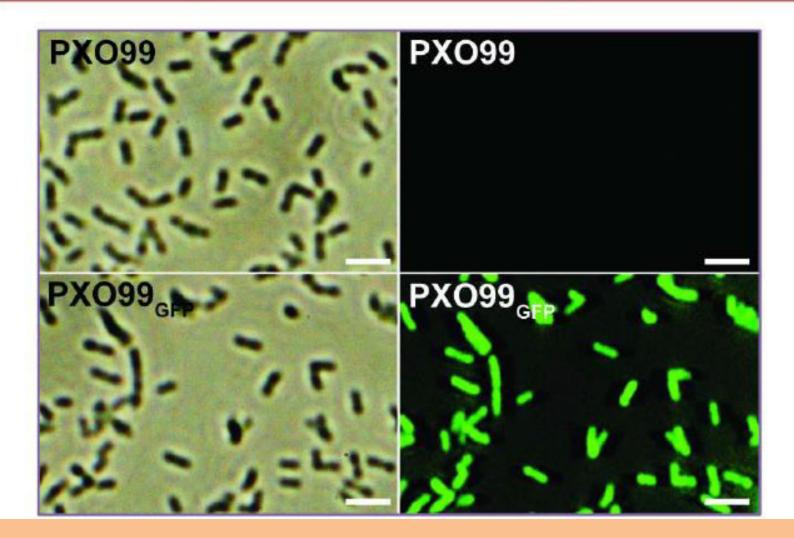
- DISEASE:Bacterial blight of rice
- PATHOGEN: Xanthomonas oryzae pv. oryzae (Is

Symptoms and Signs

- 'Kresek' occurs in early stage (Seedling wilt-Plants withers and dies up).
 - •In later stage **blightening** starts from the **tip of leaves to the base**.
 - •Straw turned yellow to white.
 - •Partially filled grains.
 - Appearance of bacterial ooze (a milky or opaque dew drop) on young lesions.



The gram negative bacteria causing the disease are rod-shaped and devoid of capsules.



Tungro disease of rice

- DISEASE:
 - Local Name: Tungro virus rog
- PATHOGEN: Rice Tungro Spherical Virus (RTSV)
- HOSTS: Rice

Symptoms and Signs

- Yellow discoloration begins from leaf tip and extends down to the blade or the lower leaf portion
- Infected leaves may also show mottled or striped appearance stunting
- Reduced tillering.
- Delayed flowering, which may delay maturity.
- Most panicles sterile or partially filled grains and covered with dark brown blotches.



Banded Leaf and Sheath blight of Maize (Zea mays)

- Disease: Banded Leaf and Sheath blight of Maize
- Pathogen:
 Rhizoctonia solani
 Kühn f. sp.sasakii
 Exner
- Host: Maize (Zea mays)



Symptoms and signs

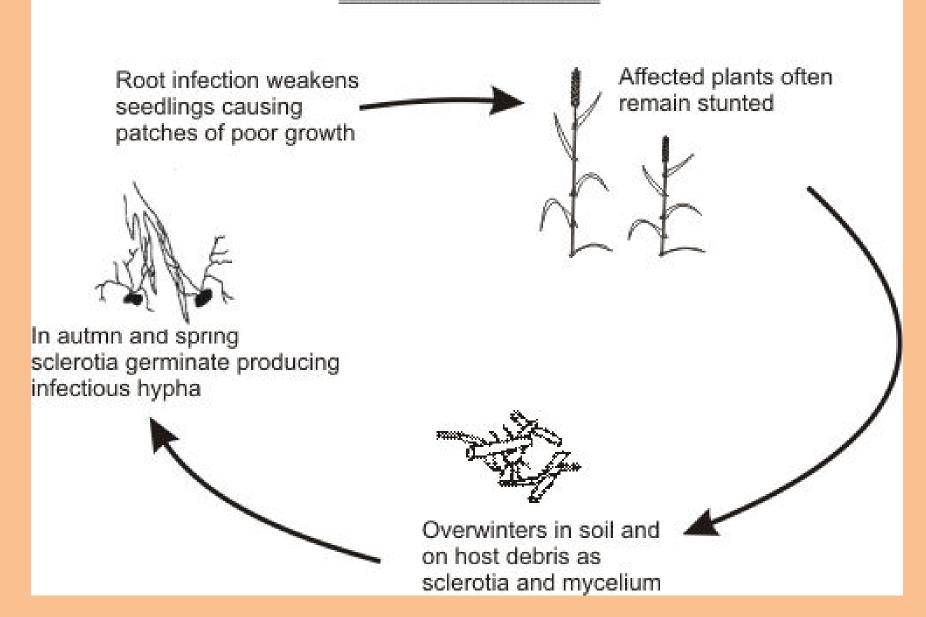
 Characteristic symptoms include concentric bands and rings on infected leaves and sheaths that are discolored, brown, tan or grey in color. The disease develops on leaves and sheaths and can spread to the ears causing ear rot.



Life cycle of Rhizoctonia solani

 Rhizoctonia solani survives in the soil and on infected crop debris as sclerotia or mycelium. Sclerotia are known to survive for several years in the soil. The fungi spread by water (flooding), irrigation, movement of contaminated soil, and plant debris. At the onset of the growing season, in response to favorable humidity and temperatures (15 to 35°C), fungal growth is attracted to freshly planted host crops by chemical stimulants released by growing plant cells. The fungi infect plants, leading to characteristic symptoms on the stem

Rhizoctonia stunt Rhizoctonia solani



Management Strategy

Host resistance

- Lack of resistant commercial varieties.
- Available tolerant germplasm should be cultivated.

Biological control

•Trichoderma bioformulations-through seed dressing and soil apllication

Cultural control

- •Stripping of 2-3 lower leaves.
- Composting of hardwood.
- •Fields should be well drained prior to planting.
- •Seeds should be planted on raised beds

Northern maize leaf blight or *Turcicum* leaf blight of Maize

- Disease: Northern leaf blight or Turcicum leaf blight of Maize
- Pathogen: Anamorph
 (asexual phase)
 Exserohilum turcicum or
 (syn. Helminthosporium
 turcicum) Teliomorph
 (sexual phase)
 Setosphaeria turcica
- Host: Maize



Symptoms

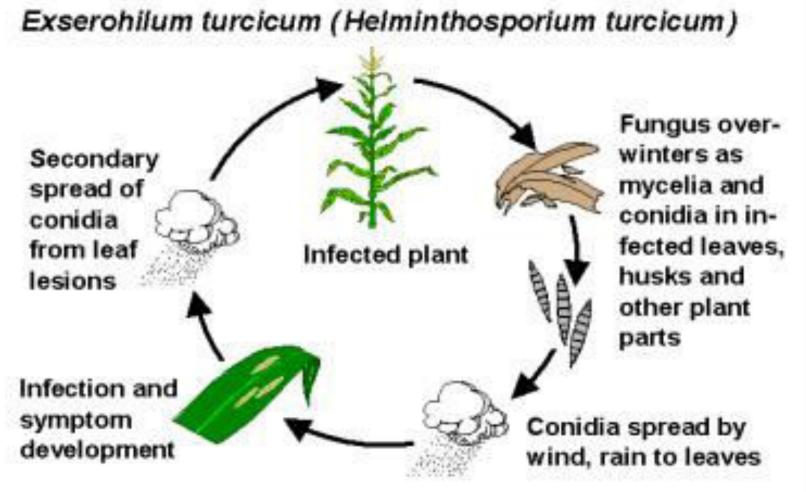
Lesions are elliptical and tan in color, developing distinct dark areas as the fungus sporulates. Lesions typically first appear on lower leaves and spread upwards. Under severe infection, lesions may coalesce, blighting the entire leaf.



Conidia are 3 - 8 septate, spindle-shaped and have a protruding hilum

Disease cycle





Southern Leaf Blight of Maize

Disease: Southern Leaf Blight of Maize

Pathogen:

Anamorph (asexual phase)

- Bipolaris maydis [Syn. Helminthosporium maydis]
- Teliomorph (sexual phase)

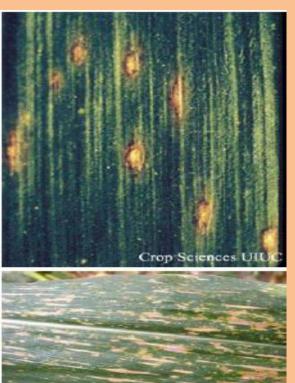
 Cochliobolus heterostrophus

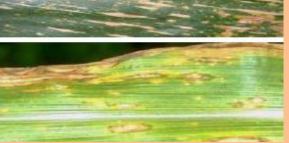
Host: Maize



Symptoms

There are 3 physiological races of Bipolaris maydis: Race T, Race O and Race C. Race O causes long, tan, lesions that have brown borders on leaves. Race T and race C are known to be specifically virulent to corn with cytoplasm male-sterile T and cytoplasm male-sterile C, respectively





Lesions on the leaves caused by Race 0 of the fungus are elongated tan coloured between the veins up to one inch long Lesions produced by Race T (which was very prevalent in the early 1970s) are tan spindle-shaped or elliptical, with yellow-green or chlorotic halos. Later, the Race T lesions often have dark, reddish-brown borders and may occur on all parts of the plants

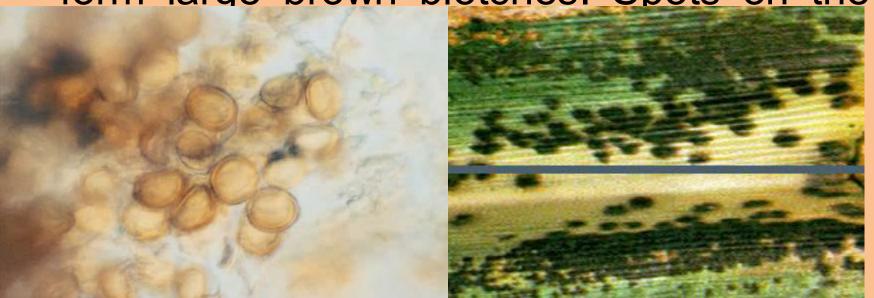
Brown spot Of Maize

- Disease: Brown spot of Maize
- Pathogen: Physoderma maydis
- Host: Maize



Symptoms and sign

 Lesions occur mainly on the leaf, but may also occur on leaf sheath, stalks, outer ear husks and tassels producing small roundto-oblong a chocolate brown to reddish brown spots which may merge together to form large brown blotches. Spots on the



Common smut of corn

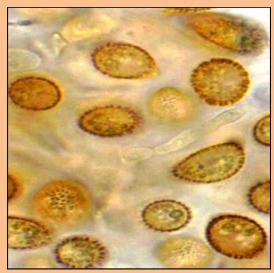
- DISEASE: Common smut (Syn. boil smut, blister smut)
- PATHOGEN: Ustilago maydis (Syn.



Symptom and Sign

 Grayish smut galls develop on the stalks, ears and tassels, while smaller galls often appear on the leaves. The galls initially have a white membrane cover that eventually breaks and releases dark-brown or black powdery spores (teliospores) which are wind borne. Common smut incidence increases





Disease Cycle

 During spring and summer, the black globose or subglobose spiny diploid (2N) teliospores, survive in the soil, germinate to form a 4-celled basidium (promycelium) that produces oval - haploid (N) sporidia or basidiospores, which are of two sexes, (+) and (-). The sporidia are blown about by air currents or are splashed by water to young developing tissues of corn plants. Infection occurs when the + and - sporidia germinate to form fine hyphae that penetrate into corn tissue through stomata, wounds, or directly through cell

Late blight of potato

Symptoms (On Potato)

- •Symptoms of late blight of Potato are small, light to dark green, circular to irregular-shaped water-soaked spots. These lesions usually appear first on the lower leaves. Lesions often begin to develop near the leaf tips or edges. The pathogen on the lower surfaces of the leaves was seen as a white downy mass. The disease is at most aggressive under damp conditions and will rapidly kill all the aerial parts of a plant. A cycle of infection to sporulation can take as little as four days. If there is a dry period, the disease pauses but will resume when the weather turns damp again.
- Infected potato tubers show surface damage only, but the damage may allow other microorganisms to enter the tuber and destroy it. The rot can be so severe that entire fields may smell of rotting vegetation.





Late blight of potato

 Causal Organism: The pathogen that causes late blight of Potato (Solanum tuberosum) is Phytophthora infestans

Control measure:

- 1. Cultural practices: certified seed of potatoes should be used for planting,
- 2. Potato waste should be burned or treated with herbicides as should volunteer plants.
- 3. Disease-resistant varieties
- 4. Avoid frequent or night-time overhead irrigation of potatoes.
- 5. The infection can be treated by repeated spraying with fungicides including: Chlorothalonil, Copper preparations such as Bordeaux mixture;, Mancozeb; Maneb; Metalaxyl; Ridomyl/ Bravo, TPTH.
- 6. Harvest should not be started until plant are completely dead
- 7. Remove infected tubers before storage
- 8. BIOLOGICAL CONTROL

With antagonistic fungi and bacteria, mycorrhizae, antibiotics

- I. Fungi: Trichoderma harzianum, Verticillium dahliae
- II. Bacteria: Pseudomonas syringae, Bacillus subtilis

List of other Important Diseases of Tea

- 1. Red rust. Cephaleuros parasiticus Karst. and C. mycoidea Karst., both are algal parasite.
- 2. Copper blight. *Guignardia camelliae* (Cooke) Butler.
- 3. Thread blight. Corticium koleroga.
- 4. Brown blight. *Glomerella cingulata* (Stonem) S. and V.S. (Syn. *Colletotrichum camelliae* Mass).
- 5. Canker. Nectria cinnabarina (Tode) fr.
- 6. Stump rot. *Ustulina deusta*.
- 7. Root rot. Rosellinia arcuata Petch and Ustulina zonata (Lev.) Sacc.
- 8. Brown rot. Hymenochaete noxia Berk.
- 9. Red root. Sphaerostilbe repens Berk, and Br.