MICROBIOLOGY AND PLANT PATHOLOGY

Diseases of crop plants

M.Sc Botany I SEM: Hard core 1.1 Microbiology and plant pathology and Soft core 1.1 Plant pathology Unit-4

CONTENTS

1. Sandal spike disease

Causal Organism: Phytoplasma

Host: *Santalum album* L. a semi root parasitic tree

Distribution:

Spike disease is major disease in sandal plants. This disease was first reported from Coorg (D), Karnataka in 1903. First detected in 1969 by electron microscopy. Verma *et al*., reported it is caused by mycoplasma like organism. Found in almost all sandal growing regions of India and Indonesia. In India Karnataka, Kerala and Tamil Nadu are the growing regions. Iyengar- 1969- estimated the annual loss of Rs. 3 million due to this disease.

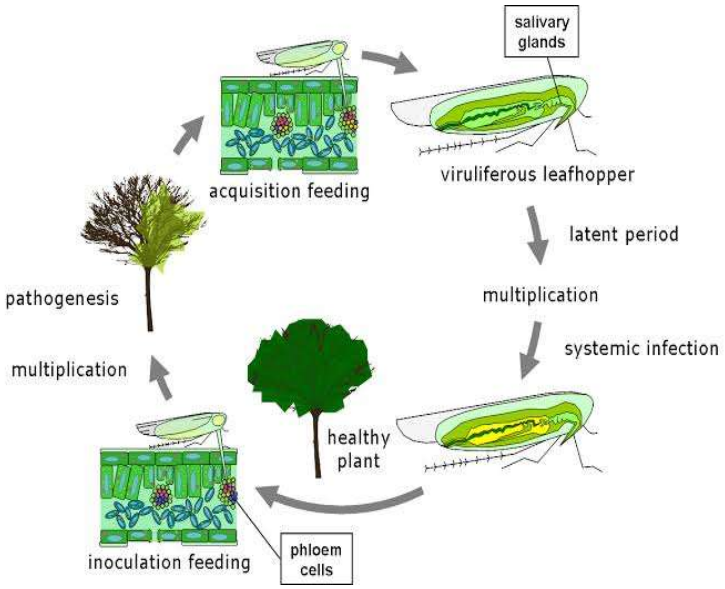
Symptoms: 2 types of symptoms

Rosette spike- severe reduction of internodes and crowding of leaves on branches, leaves become stiff and spike inflorescence like, later turns reddish/ yellowish. Flowers- become leaf like and do not bear any fruit. Root- tips/ ends die out, loss of haustorial connection with host plant. Trees die within 1-2 years after symptom appearance.

Pendulous spike- continuous apical growth of individual shoot, no thickening results in drooping. Flowers- Dormant buds do not grow- no rosettes. Roots- Roots and haustoria are not damaged.

Disease cycle:

Phytoplasma- MLOs Mycoplasma Like Organisms, were observed in sieve elements of the phloem of diseased plants. Phytoplasma cannot be cultivated *in vitro* so far, it is the smallest (0.4-1µm dia) organism that can replicate without host. Ellipsoid forms commonly seen. Transmission- is through root contacts and insect vectors- *Moonia alhimaculata* and *Nephotettix virescence.* But studies did not give any conclusive evidence of vector transmission. Natural transmission of the disease in the field usually occurs through haustorial connections.



Management:

No specific method developed so far. Planting of Mysore gum trees (*Eucalyptus tereticornis*) at 10-20 m from sandal tree keeps free from infection. Other MLOs treated with tetracycline, benlate and antibiotics, could provide temporary control. Disease reappears after varying periods of time. Though infusion of dimethyl chlorotetracycline HCl applied by girdling tree trunk, provided quick recovery from the disease, the disease reappears after 2-3 months. Foliar spray of chemicals is not practically feasible. Aqueous solutions of tetracyclines @ 500mg to 8 g in 500 ml of water were infused by trunk girdling method. Disease resistant trees through genetic engineering and tissue culture is still under research.

2. Citrus Canker

Causal Organism: *Xanthomonas compestris* p.v. *citri*

Domain: Bacteria

Phylum: Proteobacteria

Class: Gammaproteobacteria

Order: Xanthomonadales

Family: Xanthomonadaceae

Genus: *Xanthomonas*

Host: *Citrus* sp.

Distribution:

A very well-known and wide spread disease in all *Citrus* producing areas of the world. It is quite serious disease in countries like India, China, Japan and Java. According to Fawcett, bacterial canker had its origin in India. From where it spread to the rest of the world. The disease was eradicated in the USA at a cost of several million dollars by destroying millions of diseased trees to kill the bacteria. Strict quarantine regulations were then established to prevent its re-entry into the USA.

Symptoms:

The disease is characterized by brown scab by irregular spots, surrounded by dark brown glossy margin on the leaves, twigs, older branches and fruits. The spots initially develop on the lower surface of the leaf and then on both the surface. Leaf lesions first appears as small round water translucent spots usually with raised convex surface surrounded by light yellow halo and become yellowish brown. In fruits symptoms are same as in leaves except the yellow halo is absent here. In stem watery soaked appearance will later change to dark brown corky appearance.

Disease cycle:

The bacterium over seasons chiefly in leaf twig and fruit canker lesions and it spread onto young tissues and enters them through stomata or wounds. Old tissues are penetrated only through wounds/ corky lesions and several cycles of infection can occur on fruits and thus fruits have lesions of different size. Free moisture

and strong winds often favors the spread of the disease. Pathogen multiply rapidly in the intercellular space, dissolve the middle lamella and establish in the cortical region.

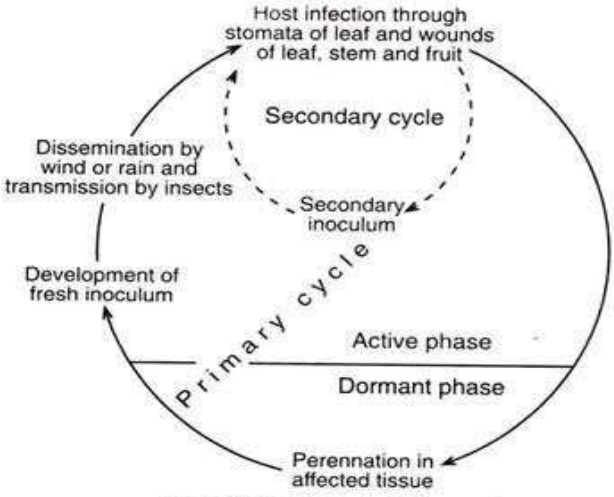


Fig. 5.50 : Disease cycle of Citrus canker

Disease management:

The only practical method of control is complete destruction of the diseased plants by burning- Phyto-sanitation. Since the disease is well established in India it is not possible to eradicate entire orchards. Disease is controlled by spraying with Bordeaux mixture for 3-4 seasons. Spraying antibiotics like streptomycin sulphates. Proper irrigation is needed along with the disease resistant varieties in the field.

3. Bacterial Blight of Paddy

Causal Organism: *Xanthomonas oryzae* pv. *oryzae*

Domain: Bacteria

Phylum: Proteobacteria

Class: Gammaproteobacteria

Order: Xanthomonadales

Family: Xanthomonadaceae

Genus: *Xanthomonas*

Host: Paddy- *Oryza sativa*

Distribution:

The disease was reported from Japan and Philippines almost 60 years ago. In India it was described by Srinivas *et al* from Pune in 1959 and Rangaswamy also isolated the organism from infections in the Tanjore delta during 1961. But the disease was not taken seriously until 1962 when epidemics broke out in Bihar and other parts of North India. Now the disease occurs all over the country in the exotic and indigenous rice varieties, and is considered a severe menace to rice production in India.

Symptoms:

Small green water soaked spots develop at the tips and margins of fully developed leaves and then expands along the veins and margins. Such spots merge and become chlorotic, then necrotic forming opaque white to grey color lesions. Bacterial ooze resembles dew drop in morning later dry on the lesion surface. During the initial stage straw color to yellow stipe with many margins appear on the leaves. Drying and bursting of the leaves can be seen, the whole leaf turns into yellowish brown and finally die.

Disease cycle:

The bacteria enter through the water pores, hydathodes, wounds of the leaf or root. It multiply in intercellular spaces of the plant, disseminate through rain water, irrigation and wind. It is both seed borne and soil borne pathogen. Bacteria that enters through root clough's xylem and cause wilt. Rice plants can become infected with bacterial blight from many sources like diseased stubbles, diseased straw, diseased seeds, paddy water etc…Alternate hosts are *Leersia, Zoysi* etc…

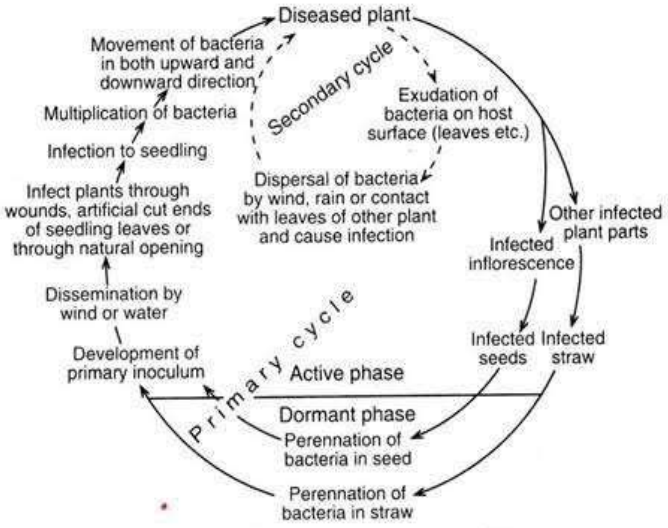


Fig. 5.48 : Disease cycle of Bacterial leaf blight of rice

Disease management:

Phytosanitation- Avoiding contaminated water. Disease is controlled by spraying with Bordeaux mixture for 3-4 seasons. Use of antibiotics- Agrimycin, Agrimycin 100, Agrimycin + Cerasan 3g/ kg. Soaking the seeds for 8 hrs in Agromycin followed by hot water treatment of seeds at 52^{o}C- 54^{o}C for 10 mins. Removal of alternative hosts like *Leersia, Zoysi* etc…Eradication of bacteria in the seeds. Growing resistant varieties like TIMN-6, IR-42 which are tolerant to disease is helpful.

4. Late Blight of Potato

Causal Organism: *Phytophthora infectans*

Host: Potato- *Solanum tuberosum*

Distribution:

In India the disease was first observed in the Nilgiri hills between 1870-1880. There after it was observed in Darjeeling district. Now it is one of the most severe of all potato diseases in the plains of Northern India.

Symptoms:

The symptoms generally appear on the above ground parts of potato but later on the underground parts of such tubers are infected. Small brown patches appear on stem and leaf, appears as water soaked spots, it enlarges if adequate humidity and temperature is available. The colour of the spots rapidly changes to black and at this time fungus produces spores on the underside of leaves hence, white cottony growth is seen. The infected tubers are shrunken on the outside and corky then rotted on the inside.

Disease cycle:

The disease is both seed and soil borne. Mycelia survives in dormant condition within the potato tubers and when such tubers/ seeds are sown plants become diseased. Secondary infection takes place from spores produced on infected parts. Spores fall to the soil or as carried by wind to healthy plants. It also disseminates through rain water, irrigation and wind.

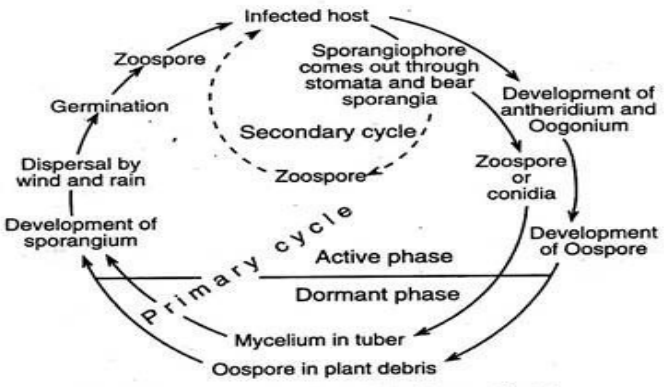


Fig. 5.20 : Disease cycle of Late blight disease of potato

Disease management:

Soaking the seeds for 8 hrs in Agromycine and antibiotic followed by hot water treatment for 10 min at 52-54^{o} C. Spray Methalaxyl formulation like Radomil N2 or MACo at 2g/ L, spraying should be repeated 10-15 days interval. Use of disease free tubers. Site selection and good drainage. Phytosanitisation- destroying/ eradication of the pathogen in the seeds and infected plant material. Growing resistant varieties like TIMN-6, IR-4d which are tolerant to disease is helpful.

5. Downy mildew of Bajra

Causal Organism: *Sclerospora graminicola*

Kingdom: Fungi

Phylum: Oomycota

Class: Oomycetes

Order: Sclerosporales

Family: Verrycalvaceae

Genus: *Sclerospora*

Host: Bajra- *Pennisetum glaucum*

Distribution:

It is most common disease in all the areas where pearl millet is grown. It occurs in many parts of Africa as well as in India. The permeable growing states of India are Rajasthan, Maharashtra, Madhya Pradesh, Gujarat and Andhra Pradesh.

Symptoms:

Infected plants are dwarf because of shortening of internodes and excessive tillering. The leaves become pale chlorotic with whitish cover the sporangia on the lower side of leaves. Leaves become distorted and wrinkled. Oospore stage of the fungus is predominant and the ear head produced becomes leafy like structure called phyllode instead of grains. Leafy structure may be covered the lower part of ear head and upper part bear normal grains. The infection starts from the seedling and if the favorable condition it spreads rapidly by the formation of sporangia and zoospores.

The symptoms can be of three types

1. whole cob is converted into leafy beard structure
2. lower half converted into leafy beard structures and the upper half bearing normal grains
3. whole inflorescence reduced and converted into leafy beard structures without bearing any grains

Disease cycle:

The disease is primarily soil borne, it can survive from 8 months to10 years. The pathogen is an obligate parasite/ biotroph, it requires a living protoplasm. The pathogen reproduces by both asexual and sexual method. The oospores which are abundantly present in the diseased leaves fall on the ground. Oospore is a sexual spore, dark brown, thick walled and hard in nature. The hyphae are inter cellular with haustoria, aseptate, sporangiophores emerge in clusters through stomata measuring 150 µ length and 12-15 µm in breadth.

Disease management:

Deep ploughing, crop rotation, rouging and phytosanitisation. Seed treatment with metaxyl, Cereson and Agrosan GN. Spray the crop with metaxyl+mancozeb at 1 kg. Growing resistant varieties viz. CO1, COHI, COH2. Seed tubers should be free from the blight obtained by certified seed. Spraying of the recommended fungicide should start when the plants are 6-8 inches tall and continuous at 10 days interval until harvest. Chemicals like Dethio carbonate, Captofol, Mongel, Chlorothianol, Bordeaux mixture.

Downy mildew of Maize

Causal organism: ***Sclerospora rayssiae* var. *zeae***

Kingdom: Fungi

Phylum: Oomycota

Class: Oomycetes

Order: Sclerosporales

Family: Verrycalvaceae

Genus: *Sclerospora*

Host: *Zea mays*

Distribution:

Downy mildews are important maize diseases in many tropical regions of the world. They are particularly destructive in many regions of tropical Asia where losses in excess of 70% have been documented. Downy mildews are caused by up to ten different species of oomycete fungi in the genera *Peronosclerospora, Sclerophthora* and *Sclerospora.* Downy mildews originated in the Old World although they have since been introduced to many regions of the New World. The disease is restricted to India and severe outbreaks have occurred in several states. The disease was first reported in Pantnager, UP in 1966.

Symptoms:

The most characteristic symptoms are the development of long, rather broad and chlorotic stripes on the leaf. When a number of such stripes fuse, the margins are lost and irregular patches are formed. 'Downy' growth is often observed on both leaf surfaces, but is more common on the lower leaf surface. Initially these strips are yellow in colour and finally browning of the tissue is seen. On the younger leaves the growth of fungus can be seen more prominently than on old leaves. The symptoms also occur on bracts of green unopened male flowers in the tassel. Infected plants have leaves that are narrower and more erect compared to healthy leaves. Infected plants are often stunted, tiller excessively and have malformed reproductive organs (tassels and ears). Infected plants may not seed, while tassels may exhibit 'bushy' growth.

Disease cycle:

The pathogen can penetrate through oospores and alternate hosts. The pathogen has many alternate hosts such as *Saccharum spontaneum, Sorghum bicolor* and *S. halepense*. Secondary infection occurs through the production of conidia or sporangia. Information is available on the seed-borne nature of downy mildew disease. Depending on the pathogen species, the initial source of disease inoculum can be oospores that overwinter in the soil or conidia produced in infected, overwintering crop debris and infected neighbouring plants. Some species that cause downy mildew can also be seedborne, although this is largely restricted to seed that is fresh and has a high moisture content.

At the onset of the growing season, at soil temperatures above 20°C, oospores in the soil germinate in response to root exudates from susceptible maize seedlings. The germ tube infects the underground sections of maize plants leading to characteristic symptoms of systemic infection including extensive chlorosis and stunted growth. When oospores initiate infection, the first leaf generally remains disease free as it is able to outgrow the fungi. However, the whole plant will show disease symptoms if the pathogen was seedborne. Oospores are reported to survive in nature for up to 10 years.

Once the fungi have colonised host tissue, sporangiophores (conidiophores) emerge from stomata and produce sporangia (conidia) which are wind and rain splash disseminated and initiate secondary infections.

Depending on the species, sporangia germinate directly or release zoospores that initiate infection. Sporangia are always produced in the night. They are fragile and cannot be disseminated more than a few hundred metres and do not remain viable for more than a few hours.

Germination of sporangia is dependent on the availability of free water on the leaf surface. If sufficient water is available, sporangia germinate and infect the plant through stomata on the leaf, sheaths, or stems in a couple of hours. Initial symptoms of disease (chlorotic specks and streaks that elongate parallel to veins) occur in 3 days. Conidia are produced profusely during the growing season. As the crop approaches senescence, oospores are produced in large numbers.

Disease management:

Diseases crops debris and collateral/alternate hosts should be destroyed. Long rotation of crops should be followed. Do not rotate or simultaneously cultivate maize with alternate hosts of downy mildew. Treat seed or crop with systemic fungicide like Dithane M-45 after sowing at 7 days interval, seed treatment with Demosan after 20 days of sowing. Other fungicides such as Brestan, Brestanol, Demosan have been effective in controlling the disease through seed treatment. Plant when soil temp is below 20°C which is unfavourable for oospore germination. Ensure seed has low moisture content (below 9%) before planting. Control weeds to increase aeration within the crop and reduce moisture levels in the soil. Use disease resistant varieties like Ph.DMR 1.

6. Tikka disease of ground nut

Causal Organism: ***Cercospora personata, Cercospora arachidicola***

Kingdom: Fungi

Division: Ascomycota

Class: Dothideomycetes

Order: Capnodiales

Family: Mycosphaerellaceae

Genus: *Cercospora*

Host: Potato- Ground nut- *Arachis hypogaea*

Distribution:

This is a worldwide disease occurring in United States, Philippines, Indonesia, India, Australia and many African countries.

Symptoms:

Characterised by dark leafspots. *C. arachidicola* cause early spot and that caused by *C. personata* as late spot. In the early spot, sub circular dark brown spots are produced on the upper leaf let surface, surrounded by yellow halo. In the late leaf spot circular, darker spots where yellow halo is seen only in later stage. As

the foliage is affected by the disease, yield will be reduced dramatically. When the plants are at least 2 months old the symptoms begin to appear.

Disease cycle:

The disease is soil borne the primary infection is caused by means of conidia found in the plant debris in the soil or by seed. The spread of disease takes place by means of conidia which are dispersed by wind. Oospore on diseased leaves give increased infection than fresh oospores. 55% of scientist detected oospores in the roots of pearl millet. Theses are formed late in season and can survive in the same plots in the left-over roots. Oospores passes through the intestine of cattle's and further claimed that their oospores could produce disease plants even after the composting of cattle dung.

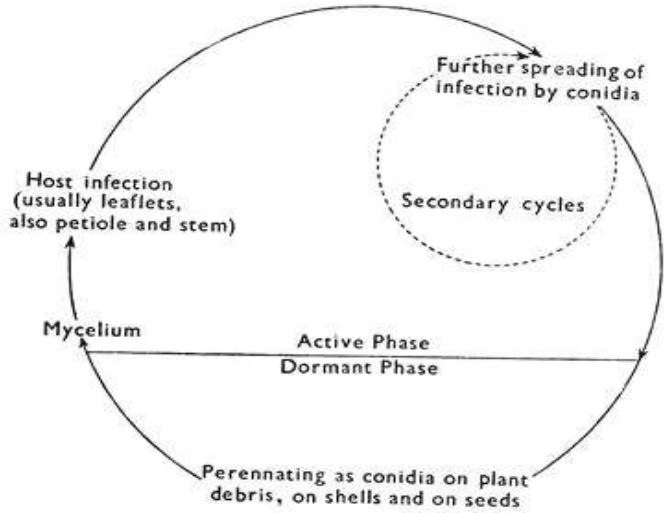


Fig. 380. Disease cycle of Tikka disease of groundnut.

Disease management:

Disease can be effectively managed by spraying crop with 7 % Bordeaux mixture or 0.2 % Zirom, Maneb, 2 or 3 times weekly intervals. Phytosanitation. Growing resistant varieties. Alternation of the date of planting, modification in planting methods, avoidance of roughing and gap filling, deep ploughing, removal of infected crop residues, crop rotation. Seed treatment may be of help in destroying the spores sticking to the surface of the grains.

7. Grain smut of Sorghum

Causal Organism: *Spacelotheca sorghi*

Kingdom: Fungi

Phylum: Basidiomycota

Class: Ustilaginomycetes

Order: Ustilaginales

Family: Ustilaginaceae

Species: *S. sorghi*

Host: Sorghum- *Sorghum bicolor*

Distribution:

It is most common in all jowar growing areas. In India it occurs in the states of Andhra Pradesh, Madhya Pradesh, Tamil Nadu and Karnataka.

Symptoms:

The disease becomes apparent only at the time of grain formation or ear head formation. In place of healthy grain bigger sized smut sori are produced. Sori are cylindrical, 5- 15 mm long and 3-5 mm broad, contains loose, dirty black coloured chlamydospores. During thawing operations, the spores burst open and contaminate the grains. The disease thus spreads to next crop.

Disease cycle:

The spores are air borne or seed borne. Spores germinate with the seed and infect the seedling by penetrating through radicle or mesopotile and establish systemic infection. At the time of flowering, the fungal hyphae get converted into spores, replacing the ovary with sori. The smut spores remain viable indefinitely. In a peridium of fungus cells forming around a central columella of host tissue.

Disease management:

Seed treatment with Sulphur organomercuric compound at the rate of 2 g/ sec. Spraying linelo at 0.3 %. Cut and burn the smutty ear head. The disease can be controlled by treating the seeds with 0.5- 3 % copper sulphate solution for 10 min reduce the fungal infection. Treating seeds with Thiram and by growing resistant varieties.

8. Phloem necrosis of coffee

Causal Organism: *Phytomonas leptovasorum*

Domain: Eukaryota

Phylum: Euglenozoa

Class: Kinetoplastida

Order: Trypanosomatida

Family: Trypanosomatidae

Genus: *Phytomonas*

Host: *Coffee arabica*

Distribution:

The disease has been reported from Surinam, Gayana, Brazil, Sansalvandar, Colombia, and probably Brazil. It has not been reported from India so far.

Symptoms:

As a result of infection trees show sparse, yellowing and dropping of leaves. Symptoms gradually advances and young top leaves remain on the otherwise bare branches. In the beginning of the dry season, trees wilt

of die within 3 to 6 weeks. Internally the roots and trunks of the trees show multiple division of cambial cells and shortened phloem vessels of disorderly structure next to woody cylinder.

Disease cycle:

The disease is transmitted through root grafts but not through grafts of the aerial parts. The disease spreads in the field from one tree to another and healthy trees often become infected if transplanted into areas where diseased tree have been removed. In nature the insect vector is the genus *Lincus.*

Disease management:

Nurseries should be free from pathogen flagellate protozoan. Healthy plants should be planted far away from the infected plants.

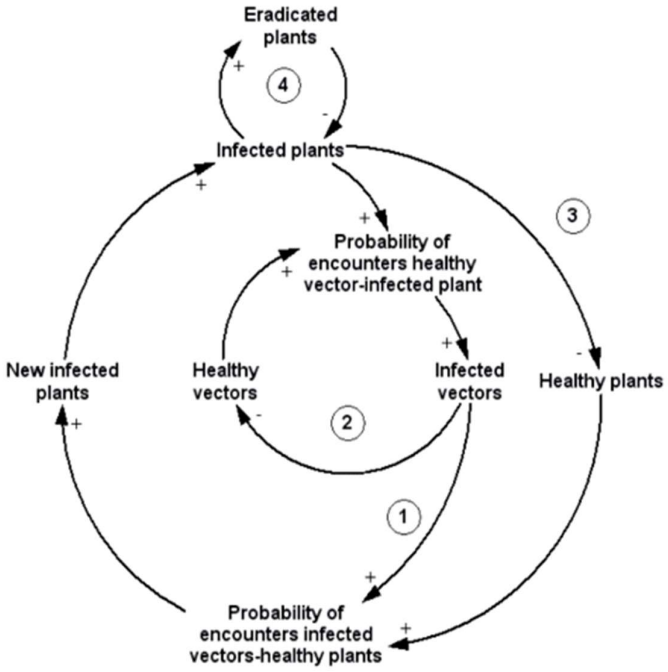


Figure 4. Diagram of causes of the system plant-vector-disease (see text for explanation).

9. Root knot of Mulberry

Causal Organism: ***Meloidogyne incognata***

Kingdom: Animalia

Phylum: Nematode

Class: Secernentea

Order: Tylenchida

Family: Heteroderdae

Genus: *Meloidogyne*

Host: *Morus alba*

Distribution:

Geographically the disease is distributed all over the world

Symptoms:

Stunted growth, poor and delayed sprouting after processing. Reduced leaf yield and leaf size. Root appears knotted due to the presence of nematodes. Chlorosis of leaf particularly followed by necrosis along the margin of leaf. Finally withering of leaves and plant may die.

Disease cycle:

Pathogen is endoparasite, may be male or female. It is pear shaped and lays 400-500 eggs in a gelatinous sac exterior to the host root surface. Juveniles grow within the egg itself and released the young juvenile to the soil which are small and easily move in soil. It gains entry to the cortical region of root and remain for 3 months and inside the host undergo excessive multiplication and enlarges, resulting in the formation of galls.

Disease management:

Nematode free saplings should be used. Other weed known to harbor nematodes must be removed from in and around the field. Keeping the land free of vegetation particularly during season like summer helps to kill the nematodes. Heavily infected soil shall be deep plough to expose the eggs, nematodes hidden in soil.

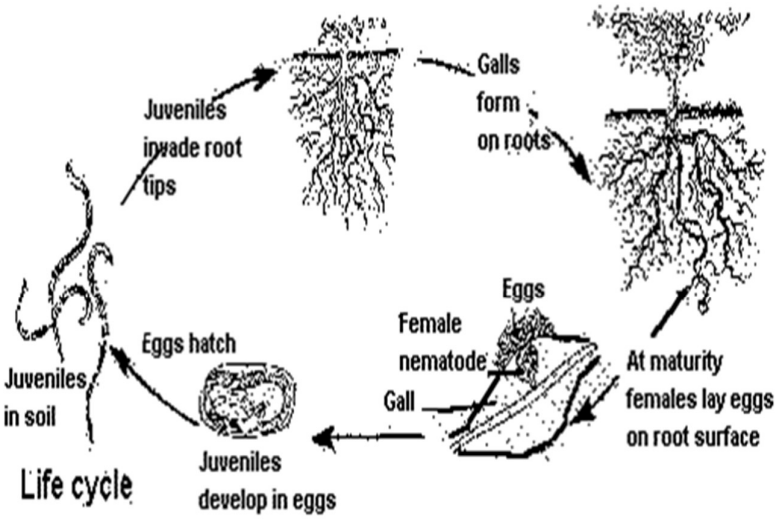


Figure 1. The life cycle of the root knot nematode

Root knot of Vegetables

Causal organism:

Nematodes–especially root-knot nematodes–cause major losses in vegetables crops in commercial farms, greenhouses, and home gardens in North Carolina. Root-knot nematodes cause root galls to develop as these microscopic parasites feed in the roots of the plants. These pests occur in about two-thirds of the fields used for crops in the state.

Many spring-planted vegetables such as beets, carrot, English pea, lettuce, potato, radish, and others, although susceptible to root-knot nematodes, often can be grown in infested soil and suffer only minor damage. This limited damage is due to the fact that nematodes are inactive at low soil temperatures (60°F). However, these same vegetables can suffer extensive damage when planted in the late spring, summer, or fall when soil temperatures are more suitable for nematode activity (70-85°F).

Symptoms:

Symptoms may include stunting, wilting in the hottest part of the day, yellowing, loss of vigor, and uneven growth. Root crops such as carrots typically become forked. Symptoms are typically unevenly distributed in the field as nematodes do not tend to move far; symptoms may begin as small patches, but these may enlarge as the nematode population increases.

NRKN feeding induces formation of characteristic root galls, which interfere with proper root function. Feeding activity also robs plants of nutrients and causes wounds through which other pathogens may enter. Plants seldom die from nematode infestation alone, but nematodes can have a serious impact on plant health and crop yield. NRKN damage is also known to increase the severity of disease caused by some plant pathogenic fungi, particularly *Verticillium* and *Fusarium*.

If nematode damage is suspected, examine plant roots for galls. The number of root galls is dependent upon the nematode population density and the species and cultivar of the host. NRKN galls are about 1/8th inch in diameter but may be larger. Don't mistake the nodules on legumes for root-knot galls- the roots of these plants develop these nodules as they form beneficial associations with soil bacteria. Club root of brassicas also resembles root-knot nematode galls but club root galls are larger.

Disease cycle:

NRKN can be introduced by infested transplants that were grown in field soil, but the nematodes also overwinter as eggs in the soil. The eggs hatch when soil temperatures reach approximately 65°F (18°C), but embryo development may begin at temperatures <50°F (10°C). The juveniles molt four times, once inside the egg and then thrice after hatching; the last molt results in an adult. Juveniles are less than 1 mm long and enter into the root where they establish a feeding site next to the vascular system. The nematode remains sedentary, feeding in one site while undergoing successive molts until the adult stage. The adult is almost always a female and sac-like in shape. Their feeding induces formation of the characteristic root galls. A single female can lay a thousand eggs which are extruded to the surface of the root in a gelatinous matrix. At optimum temperatures (80°F, 27°C), the life cycle is completed in 3-4 weeks, so the population of NRKN in the soil can increase significantly in a short time.

Disease management:

The best way to manage NRKN in vegetable fields involves developing an integrated pest management (IPM) plan that may include growing resistant cultivars when they are available, crop rotation, cultural controls, and chemical applications.

- **Crop rotation**: Asparagus and small grains are relatively resistant to NRKN. Corn is also not considered to be a host in the Northeast, although it has been reported as a host in the Pacific Northwest. *Brassicas* are generally less susceptible to NRKN than other vegetable families. Two studies have shown that a three-year rotation with onions and barley can significantly reduce NRKN damage on carrots in organic soils. Consider growing early crops such as peas, spinach, or radishes, as they grow mainly in the cooler weather of spring before NRKN activity reaches its peak.
- **Resistance**: Cultivars of several vegetables (most notably tomatoes) are marketed as nematode resistant; however, this designation refers to nematode species other than *M. hapla*. Currently, there are no NRKN resistant cultivars available for common vegetable crops.
- **Cultural control**: Destroy infested plants at harvest. Do not compost them. Control weeds as some common species (including dandelion, purslane, and plantain) are also hosts for NRKN. Add organic matter: in addition to boosting plant nutrition, this increases microbial competition and populations of natural enemies in the soil. Plants growing in fertile soils are able to withstand nematode infestation better than those grown in poor soils. Do not move infested soil to an uninfested area- clean tools, tractors, and boots before moving from one area to another.
- **Conventional control products**: Telone and Vapam are available for pre-plant soil fumigation. Vydate is labelled for use on Solanaceous crops in Massachusetts. Choices are limited as many conventional nematicides have been removed from the market due to their toxicity. Fluensulfone, a new nematicide chemistry, has recently been released under the name Nimitz. Nimitz is not a fumigant, and is labelled for cucurbits and solanaceous crops.
- **Organic control products**: Several OMRI approved products are available for nematode control; however, evidence of effectiveness is often lacking.
- **Biofumigation**: Certain *Brassica* species can be used as biofumigants to control NRKN. Research has shown that biofumigation can be effective; however, it also impairs the activity of *Steinernema* species that are used for biological control of insects.
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10. Head smut of Sorghum

Causal organism: *Sporisorium reilianum/ Sphacelotheca reiliana*

Kingdom: Fungi

Division: Basidiomycota

Class: Microbotrymycetes

Order: Microbotryales

Genus: *Sphacelotheca*

Host: *Sorghum bicolor*

Distribution:

It is found in Africa, Asia, Australia, Europe and North, Central and South America. In India, it is prevalent in Andhra Pradesh, Karnataka, Maharashtra, Madhya Pradesh, Uttar Pradesh, Punjab, Tamil Nadu. In Kashmir, it occurs significant damage to the sorghum crop. The disease causes significant damage to sorghum as it affects the entire ear, transforming it into smutted head.

Symptoms:

The disease is not noticeable in either the size or the growth of the plant. Disease becomes apparent only at the time of flowering. The entire inflorescence is converted into a big sorus, about 10-13 cm in length and 4.6 cm in breadth. When it has fully emerged, the fungal wall ruptures, exposing large mass of black, powdery spores. The large smut sore replaces the tassel and ear in sorghum. The sorus consists of the conductive tissue elements of the susceptible, surrounded by the spore mass and the fragile exterior fungal members. If the wind is blowing at the time of sorus emergence, the air-borne spores resemble a smoky cloud around the head. When the spores are blown off, a network structure of dark, filamentous vascular tissues of the host is exposed.

Disease cycle:

The fungus in soil infects young seedlings when the soil temperature is about 28^{o} C and moisture content between 15-25%. The fungus produces sorus which is composed of loosely arranged spores and the conductive tissue of the suscept which is initially enclosed by a fragile fungal membrane. The chlamydospores are reddish brown to black. The spore mass is powdery and dark brown and is quickly dispersed to expose a tangled mass of vascular strands of the host, only young plants are susceptible, the fungus becomes systemic. In dry soil, the spores remain viable for a considerable period, at least until the following crop season. With rain or irrigation water they germinate, and if young seedlings are present in the vicinity and other conditions are favourable, they infect the host plant, maintaining the disease cycle.

Disease management:

Crop sanitation to reduce the inoculum potential. Crop rotation of primary importance. Using disease Resistant varieties. Application of urea, ammonium sulphate and tri super phosphate reduces the disease considerably. Collecting smutted heads in drill cloth bags and dipping in boiling water to kill the pathogen will reduce the inoculum potential for the crops.

11. Angular leaf spot of Cotton

Causal organism: *Xanthomonas axonopodis* pv *malvacearum*

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Gammaproteobacteria

Order: Xanthomonadales

Family: Xanthomonadaceae

Genus: *Xanthomonas* sp.

Host: *Gossypium* Spp.

Distribution:

The disease was first reported in Alabama, USA in 1891 and now known to occur in all major cotton-growing regions of the world. From India, the disease was first reported from Madras in 1918 and now occurs in Maharashtra, Madhya Pradesh, Andhra Pradesh, Tamil Nadu and Uttar Pradesh. Annual losses due to the disease in India range from 5- 25%.

Symptoms:

The disease appears on different parts of cotton plant, both in seedling and mature plant stage. The disease first appears on leaves, which appear water soaked, turn black and dry up often leaving the young seedling green with a black tip. In less severely affected plants, points on leaves and stem become water soaked and enlarge into angular reddish spots about 1 mm in diameter. The spots often coalesce and the leaf gradually yellows and drops. Yellowish bacterial exudate is common on lesions, in moist weather. On bolls, water soaked lesions appear, which coalesce to form irregular, large, brown, sunken areas. Bolls infected when young, may drop prematurely. Older bolls when infected may become distorted and the lint may be discoloured.

Disease Cycle:

The bacteria enter the mature seed through the basal end of the chalaza. They over winter in this manner and as contaminants on the surface of the seeds or in the lint attacked to it. Volunteer seedlings are the chief source of primary inoculum when cotton is planted after cotton. Wind-blown soil, rain and irregular water are the means of dissemination. Insects have little importance. High humidity and moderate temperature (28 °C) favours the development of the disease. Primary infection is favoured by 30 °C and secondary infection is better at 35 °C. presence of moisture is very important for the first 48 hours. Dry and hot weather retards disease development.

Disease management:

Use of healthy seed from healthy plants. Delinting seeds with concentrated sulphuric acid then floating the delinted seeds in water and removal of the floating seeds. Disinfections of seeds with 1000 ppm streptomycin sulphate solution overnight. Destruction of diseased plant debris, and Killing of volunteer seedlings.

12. Leaf Rust of Coffee

Causal organism: *Hemileia vastatrix*

Kingdom – Fungi

Division – Basidiomycota

Class – Pucciniomycetes

Order – Pucciniales

Genus – *Hemileia*

Species – *H. Vastatrix*

Host: *Coffea* sp.

Distribution:

First described by Berkley and Broom in 1869 from Sri Lanka and was seen in Southern India and over most of South east Asia. Thought to have co-evolved alongside *Coffea* at coffee centre of origin: Ethiopia, Africa. Now endemic to all economically important coffee growing regions. It is one of the most devastating and widespread disease of coffee worldwide.

Symptoms:

The symptoms of coffee rust include small, yellowish, oily spots on the upper leaf surface that expand into larger round spots that turn bright orange to red and finally brown with a yellow border. The rust pustules are powdery and orange-yellow on the under-leaf surface. Later the pustules turn black. Rusted leaves drop so that affected trees are virtually denuded; such trees have significantly lower coffee yields and usually die within a few years.

Disease cycle:

Pathogen needs suitable temperatures to develop (not less than 10 °C and not greater than 35 °C). The presence of free water is required for infection to be completed. Loss of moisture after germination starts inhibits the whole infection process. The colonization process is not dependent on leaf wetness but is influenced greatly by temperature and by plant resistance. Fungus begins with the germination of uredospores through germ pores in the spore. It mainly attacks the leaves and is only rarely found on young stems and fruit. Appressoria are produced, which in turn produce vesicles, from which entry into the substomatal cavity is gained. Within 24–48 hours, infection is completed. After successful infection, the leaf blade is colonized and sporulation will occur through the stomata. Sexual reproduction (cryptosexuality) has been found within the generally asexual urediniospores.

Disease management:

Field sanitation, sufficient tree pruning and good site selection. Use of fungicides like copper compounds, Bordeaux mixture (40-50%), copper oxychloride and cuprous oxide. Synthetic fungicide like oxycarboxin (Plantvax) and Triadimefon found to be good eradicant of fungus. Resistant variety *Coffea canephora* crossed with *C. arabica* showed high level of resistance against all races of the rust.

13. Leaf spot of paddy

Causal organism: *Helminthosporium oryzae*

Kingdom: Fungi

Division: Ascomycota

Class: Dothideomycetes

Order: Pleosporales

Family: Pleosporaceae

Genus: *Helminthosporium*

Host: *Oryza sativa*

Distribution:

It occurs in most of the rice growing countries of the world. The disease is also referred to as fungal blight. Though the disease is believed to have been present for many years, the description of the causal organism was made by Breda de Haan only in 1900. Ocfemia in 1922 reported the occurrence of the disease in USA, Japan, Italy, Phillippines and other countries. The first report on the disease in India was made by Sundararaman from Madras in 1919. The disease was one of the principle cause of the famous Bengal famine of 1942-43. The disease is widely distributed throughout the country in West Bengal, Orissa, Andhra Pradesh, Tamil Nadu and Karnataka.

Symptoms:

The disease causes blight of seedling. In mature plant leaf spotting is the most commonly and readily observable symptoms. The spots on the leaves and leaf sheaths are brown, round to oval, measuring about 0.5-2 mm x 2-5 mm may coalase to form bigger spots. Spots appear on grains, stem, brown spots appear so much that the yield will be affected. At times the neck region is infected, causing symptoms similar to those of neck blast. The disease is associated with a physiological disorder known as 'akiochi' in Japan. Abnormal soil conditions (deficiency of nitrogen and potassium) predisposed the plants to heavy infections.

Disease cycle:

The fungus produces brown mycelium which is inter and intracellular in host. From the mycelium conidiophore emerges throughout the stomata. Conidiophore are septate and bear conidia at regular intervals on the upperpart of conidiophore. Conidia are elongated with cylindrical 8-10 celled. Diseased seeds may give rise to the seedling blight in the first phase of the disease. Secondary infection takes place through conidia.

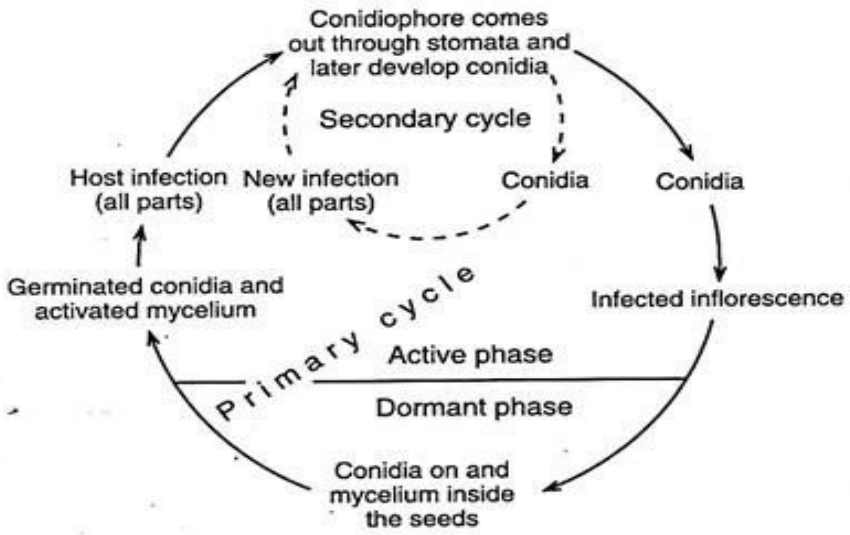


Fig. 5.8 : Disease cycle of Brown spot of rice

Disease Management:

Field sanitation-removal of collateral hosts and infected debris from the field. Use of slow release nitrogenous fertilizers is advisable. Grow tolerant varieties *viz.,* Co44 and Bhavani. Use disease free seeds. Treat the seeds with Thiram or Captan at 4 g/kg. Spray the nursery with Edifenphos 40 ml or Mancozeb 80 g for 20 cent nurseries. Spray the crop in the main field with Edifenphos 500 ml or Mancozeb 2 kg/ha when grade reaches 3. If needed repeat after 15 days.

14. Blast of paddy

Causal organism: *Magnaporthe grisea* (Hebert) Barr

Kingdom: Fungi

Phylum: Ascomycota

Class: Sordariomycetes

Order: Magnaporthales

Family: Magnaporthaceae

Genus: *Magnaporthe*

Host: *Oryza sativa* L.

Distribution:

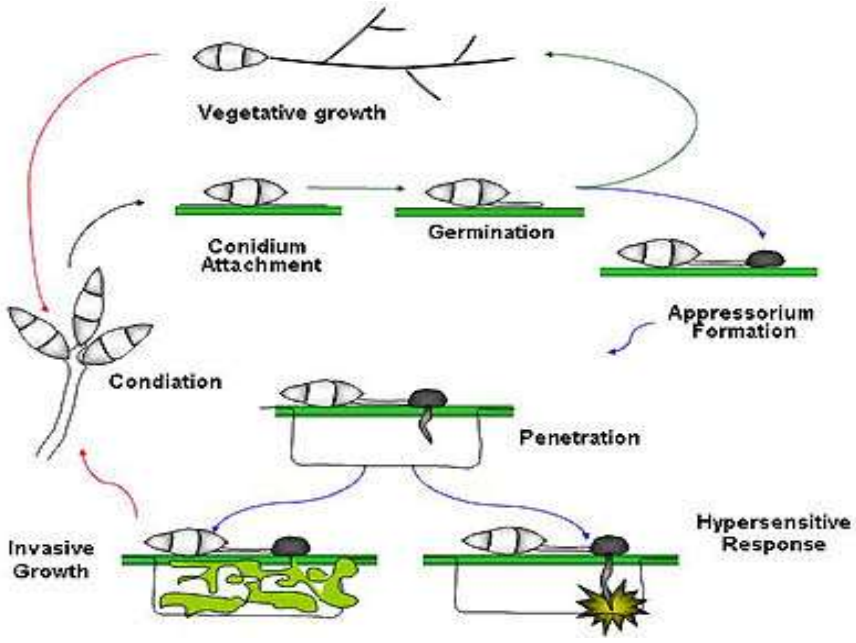
Rice blast was probably first recorded as rice fever disease in China in 1637. It was later described as imochi-byo in Japan in 1704, and as brusone in Italy in 1828. The fungus is currently reported to be present in at least 85 countries. In 1996 rice blast was found in rice in California, and has since been found in grasses on golf courses in the mid-western United States. In India, occurrence of this disease was first reported form the Tanjore district of Tamil Nadu in 1918, Maharashtra in 1923 and subsequently in several other parts. It now occurs in almost all principal rice growing states.

Symptoms:

Initial symptoms are white to grey-green lesions or spots with darker borders produced on all parts of the shoot, while older lesions are elliptical or spindle-shaped and whitish to grey with necrotic borders. Lesions may enlarge and coalesce to kill the entire leaf. Symptoms are observed on all above-ground parts of the plant. Lesions can be seen on the leaf collar, culm, culm nodes, and panicle neck node. Internodal infection of the culm occurs in a banded pattern. Nodal infection causes the culm to break at the infected node (rotten neck). It also affects reproduction by causing the host to produce fewer seeds. This is caused by the disease preventing maturation of the actual grain.

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 pathogen penetrates the plant. *M. grisea* then sporulates from the diseased rice tissue to be 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 favourable 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 rice crops.



Disease Management:

Disease can be controlled by Field sanitation, destructive of collateral hosts and other cultural practices. Application of nitrogen fertilizers reduces the host resistance. Sowing dates should also be adjusted to avoid disease. Seed treatment with organomercurials like Agrosan GN. Among fungicides, the effective ones have been copper compounds like Bordeaux mixture, copper oxychloride, cuprous oxide, etc. Resistant cultivars developed for the disease are Co 4, pe TKM-1, Co 29, Co 30, T-603, A-67, A-90, IR-579, etc.

15. Powdery mildew of Cucurbits

Causal organism: *Sphaerotheca fuliginea* and *Erysiphe cichoracearum*

Kingdom: Fungi

Division: Ascomycota

Class: Leotiomycetes

Order: Erysiphales

Family: Erysiphaceae

Genus*: Sphaerotheca fuliginea*

Erysiphe cichoracearum

Host: Cucumber, pumpkin, watermelon, muskmelon,

Distribution

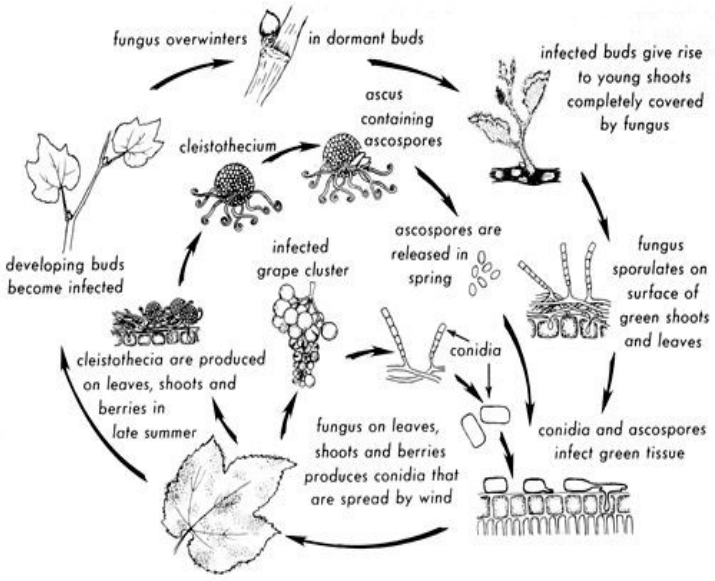
The disease is common and occurs almost every year in all cucurbit-growing areas. The disease is worldwide in distribution. In Asia, the disease is reported to occur in Iran, Iraq, Saudi Arabia, Israel, Malaysia, Singapore, China, Japan, Taiwan and India. From India Butler in 1981, reported the disease for the first time from U.P and Bihar.

Symptoms:

Disease appears first in the form of minute, white to dirty grey spots on both leaf surfaces, stems and petioles. In later stages Mildew develops as powdery fungal growth. The superficially powdery mass finally covers the entire surface of affected parts. In winter season, minute dark-brown, pinhead bodies appear intermixed with the white powdery mass. These are the perfect state bodies, cleistothecia. Infected leaves shrivel and die; fruits remain undersized and deformed, plants may senesce prematurely.

Disease Cycle:

The pathogens are obligate parasites and cannot survive in the absence of living hosts. Initial inoculum is most likely airborne conidia, other possible sources include greenhouse grown cucumbers and alternate hosts. Under favourable conditions, Powdery mildew develops rapidly; the time between infection and symptom expression can be as short as 3 days and many spores are produced. Conditions favouring infection include a dense plant canopy, low intensity light, high nitrogen fertilization, and high relative humidity (although infection can occur at relative humidity of less than 50%). Optimum temperatures for disease development are 68-80° F; infection can occur between 50-90° F. Temperatures of 100° F or above stop Powdery mildew development.



Disease Management:

Field sanitation is useful in reducing the incidence of disease. Diseased plant debris should be controlled and burnt in the field. Disease can be effectively controlled by using timely sprays of foliar fungicides, soon after the appearance of the disease. Sulphur dust, copper sulphate and Karathane have been found highly effective. Spraying of crop with 0.05 to 0.2% Karathane @ 1000 L/ha is a usually recommended practice. Elosal (wettable powder with 80% elementary sulphur) (0.5%)also provides effective control.

16. Wilt of Tomato

Four kinds of wilt diseases are found in Tomato plants:

- ***Fusarium* wilt of tomato**
- ***Verticillium* wilt of tomato**
- **Bacterial wilt of tomato**
- **Tomato spotted wilt virus**

a. *Fusarium* wilt of tomato

Causal organism: ***Fusarium oxysporum* f.sp. *lycopersici*.**

Symptoms:

Lower leaves turn yellow, sometimes on one side of the plant or one side of a branch. This is followed by leaf and stem wilting. When an infected stem is scraped or split lengthwise browning of the vascular tissue can be seen. Clogging of the vascular tubes (that carry food and water in the plant) that produces the wilting and yellowing.

Disease Management:

There is no cure for *Fusarium* and since it persists in the soil for years, it can be very hard to eradicate. Using disease Resistant Varieties could control the disease. Using sterile soil during Potting will reduce the

disease intensity. Raise the Soil pH - Apply lime to the soil in garden, to bring the pH up into the neutral range of about 6.5 - 7.0. Avoid injuring plants' roots by not using a hoe or cultivator around tomato plants. Damaged roots are entry points for the pathogen. Crop rotation: Since the pathogen can persist in the soil for years, a 5 to 7 year rotation is recommended, but that does not guarantee the pathogen will be entirely gone.

b. *Verticillium* wilt of tomato

Causal organism: ***Verticillium albo-atrum***

Symptoms:

Yellow spots appear on lower leaves, followed by brown veins. Leaves then turn brown and fall off. Infection pattern often resembles a V-shape. Symptoms progress up the stem. Plants may wilt during the day and recover at night. Interior of main stem (when split) shows discoloured streaks about 10-12 inches above the soil line, the result of plugged water-conducting tissue. If cool conditions persist, the plant may die.

Disease management:

To date, there is no chemical treatment available. To slow the disease, use fertilizers lower in nitrogen and higher in potassium. Rotate crops- The verticillium fungus can survive indefinitely in the soil. Plant tomatoes no more than once every four years in the same spot. Avoid planting other *Solanaceous* crops (potato, pepper, and eggplant) in the same area, too – they are susceptible to the fungus. Choose disease-resistant tomato varieties. Plant tomatoes in well-drained soil. Remove and destroy affected plants at the end of the season.

c. Bacterial wilt of tomato

Causal organism: The bacterium, ***Ralstonia (Pseudomonas) solanacearum***

Symptoms:

Rapid wilting and death of plants without yellowing or spotting of leaves. Brown discoloration and decay are evident inside the stems of infected plants. The disease is easily diagnosed by suspending a clean, cut section of diseased stem in clear water. A white milky stream of bacterial cells and slime flow from infected stems into the water after a few minutes.

Disease management:

Control of bacterial wilt in infested soils is difficult. Therefore, avoid using diseased transplants and establish plantings in non-infested soil. Soil fumigation may provide partial control, cut does not completely eliminate bacteria from the soil. When infected plants are identified, remove and destroy them immediately.

d. Tomato spotted wilt virus

Causal organism: ***Tomato Spotted Wilt Virus*** and ***Impatiens Necrotic Spot Virus***.

Symptoms:

Stunting of new growth, bronzing of young leaves, and brown streaking on terminal stems. Dark purple to brown ringspots may develop in some leaves. Infections in older plants often do not affect all the terminals and may result in one-sided growth. Ringspots are light green in colour on green fruit and a more conspicuous yellow on ripe fruit.

Disease management:

Avoid producing vegetable transplants in a greenhouse where ornamentals have been imported or are being vegetatively propagated. Inspect incoming plants, especially ornamentals, for virus symptoms and thrips infestations. Remove and destroy any symptomatic plants. Controlling thrips with insecticides, particularly in the field, usually does not reduce disease because thrips are able to infect plants before the chemicals can act to kill them. Resistant tomato varieties.

17. Bunchy top of Banana

Causal organism: *Banana Bunchy Top Virus* (BBTV)

Group : Group II

Family : Navoviridae

Genus : Babuvirus

Species: *Banana Bunchy Top Virus*

Host: *Musa* sp.

Distribution:

Disease was first reported by a scientist from Australia. It also occurs in Fiji, Egypt and Srilanka. The disease is believed to have been introduced through infected suckers brought from Srilanka to Kerala. Now it is prevailing all over India.

Symptoms:

In infected plants leaves become bunched together at the apex of pseudostem to form rosette or bunchy top. The margin of the leaves becomes wavy and sometimes they roll upwards as a result of reduction in size of the petiole, the whole plant gets stunted.

Disease cycle:

Banana bunchy top is a viral disease caused by a single-stranded DNA virus called the Banana Bunchy Top Virus (BBTV). Virus is not transmitted through mechanical inoculation. The chief insect vector is *Pentalonia nigronervosa.* The aphid acquires the virus in a feeding period of 24 hrs and transmit the virus to a healthy plant in an infection period of 1 ½ hrs

Disease Management

Diseased plants should be carefully removed and destroyed. Killing plants along with aphid feeding on them by pouring kerosene. The plants can be killed by hormones weed killer MCPA (2 methyl 4 chloro phenoxy acetic acid). Use of certified virus free suckers for planting.

18. Grassy shoot of sugarcane

Causal organism: **Mycoplasma like organism (MPLO) or Pleuro pneumonia like organism (PPLO)**

Host: ***Saccharum officinarum***

Distribution:

The disease also named as yellowing and white leaf diseases of sugarcane is known to occur in India, Sri Lanka, Burma, Burma, Thailand, Sudan and Taiwan. From India, the disease was first observed in 1919 in Maharashtra. Since then this has been reported from U.P., Punjab, Bihar, Rajasthan, Andhra Pradesh, Tamil Nadu and Orissa. Several varieties of sugarcane are attacked by this disease.

Symptoms:

Profuse tillering and grassy appearance of the diseased stools are the characteristic symptoms of the disease. Various other symptoms at different stages of plant growth have been reported like chlorotic disease, albino disease, yellowing disease and leaf tuff disease. Shoots emerging from diseased sets remain stunted and dwarfed. Leaves become almost white due to complete chlorosis or yellowing due to partial chlorosis. Lower nodes produce large number of grassy shoots.

Disease cycle:

The disease is caused by phytoplasma. Mycoplasma-like bodies have been identified in the phloem of infected parts. Phytoplasma is pleomorphic, appearing as ovoid, spherical or irregular in shape. The phytoplasma is readily transmitted by sap inoculation in the field. It is transmitted through infected plant and through aphids.

Disease management:

The disease is controlled by eradication of diseased plants as soon as symptoms are noticed. Avoiding selection of plant from the diseased areas, pre-treatment of healthy canes with hot water at 25^{O} C for 1 hr. Moist heat treatment at 54^{O} C (90-95% relative humidity) for 1 ½ hr also inactivated the phytoplasma. Treatment of canes with hedermycin has given complete protection. Control of insect vectors with sprays of 0.16% malathion during early stages of crop growth can reduce the incidence of disease.

19. Little leaf of brinjal

Causal organism: Phytoplasma

Vector: Leaf hopper (*Hishimonus phycitis* and *Amarasca bigutulla*)

Host: *Solanum melongena*

Distribution:

The disease is very common in India and other neighbouring countries. From India, the disease was first reported from Coimbatore, Tamil Nadu. Since then this has been reported to cause considerable damage to the crop in brinjal growing areas of most states.

Symptoms:

The disease affected plants appear short, proliferation of large number of branches, roots, leaves occur. Internodes get shortened, many buds appear at the leaf axils giving the plant a bushy appearance. Flower parts get deformed and the plants do not bear any fruit. If any fruit is formed, it formed hard and fails to mature.

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.

Disease management:

Eradication of weed hosts like *Datura* and *Vinca* and the diseased plants of eggplant help in reducing the disease. Control of insect vectors by insecticide is also helpful in reducing the spread of the disease. Experimental control of disease has been obtained by tetracyclines. Resistant cultivars like BB-7, BWR-12, Pant Rituraj and H-8 can been used.

20. Potato spindle tuber disease

Causal organism: *Potato spindle tuber viroid* (PSTVd)

Host: *Solanum tuberosum*

Distribution:

The disease occurs in North America, Russia and South Africa and few years back also reported from India. A strain of the viroid causing extensive veinal necrosis in four wild species of Solanum was reported in 1992 from Shimla hills, Himachal Pradesh. The disease poses a threat to production of seed potatoes and maintenance of germplasm.

Symptoms:

Mild strains generally cause no obvious symptoms in potatoes. However severe strains in sensitive cultivars may cause foliage to be spindly, very upright, with overlapping leaflets and sometimes with upward rolling of terminal leaflets. Plants will be stunted. Tubers may show the following deformities; small, elongated, cylindrical, spindle or dumb-bell-shaped, with prominent eyes evenly distributed over the tuber, and cracking. Sprouting is slower than in healthy tubers.

Disease cycle:

The disease is mechanically transmitted by contact between healthy and diseased plants, tractor wheels, tools, etc. Within potato plants, it is found most readily in the upper leaves and tubers. Transmission in true seed of potato depends upon the cultivar. PSTVd can pass through both the pollen and ovule. Long Range dispersal mainly occurs through the movement of infected tubers and true seed. PSTVd can also be spread by aphids. But this only occurs in the presence of Potato leaf roll virus (PLRV) and is unlikely to be a major problem in the UK.

Disease management:

There are no chemical or biological controls available to control PSTVd within infected plants. Therefore, control is essentially through the use of healthy virus-free seeds or planting material, and good crop sanitation practices. If infection does occur, destruction of infected plants and strict hygiene measures to prevent infection of subsequent crops are the only courses of action.

21. Tobacco Mosaic disease

Causal Organism: Tobacco Mosaic virus

Group : Group IV (ssRNA)

Family : Virgaviridae

Genus : Tobamovirus

Species: Tobacco Mosaic virus

Host: *Nicotiana tabacum*

Distribution:

Tobacco mosaic is the first recognised viral disease of plant. It is world-wide in distribution. It is most common on Tobacco in India appearing in every tobacco growing area of the country. The tobacco mosaic virus affects all dicotyledonous plants of which most important are tobacco and tomato.

Symptoms:

The primary symptom on young leaves is faint circular chlorotic lesions appear with gradual vein clearing. Chlorosis, curling, mottling, dwarfing, distortion and blistering of leaves, light discolouration of flowers. The leaf develops characteristics light and dark green areas, giving a mosaic appearance. The dark green areas are confined to the veins. At times leaf spots also appears. Stunting of younger plants is common. In severe infections, leaves are narrowed, puckered and thin, in general malformed beyond recognition.

Disease cycle:

The virus is rod shaped, measuring 280µ in length by 15µ, in width, and is made up of genetically placed RNA covered with a protein coat called capsid. The virus is disseminated from plant to plant by mechanical transmission, Virus is sap transmissible and enters the host through wounds, on the plant which becomes the passage for entry of virus. It is most resistant to adverse environmental and climatic conditions and hence, when it reaches the field it may become infective any time the crop is sown. The virus is not seed

transmitted in tobacco, except as a contaminant on the surface, but it is reported to be seed transmitted in tomato. The intensity and type of symptoms produced by the virus on a given host also vary with the physiological condition of the plant and environmental factors such as temperature.

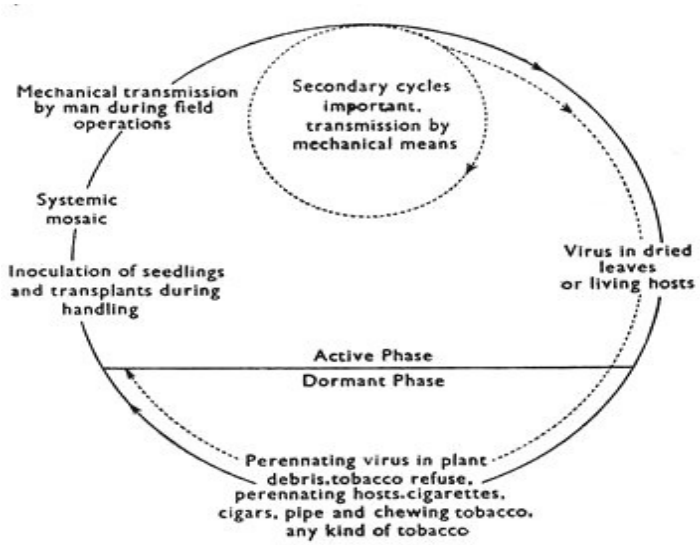


Fig. 394. Disease cycle of Tobacco Mosaic Virus.

Disease management

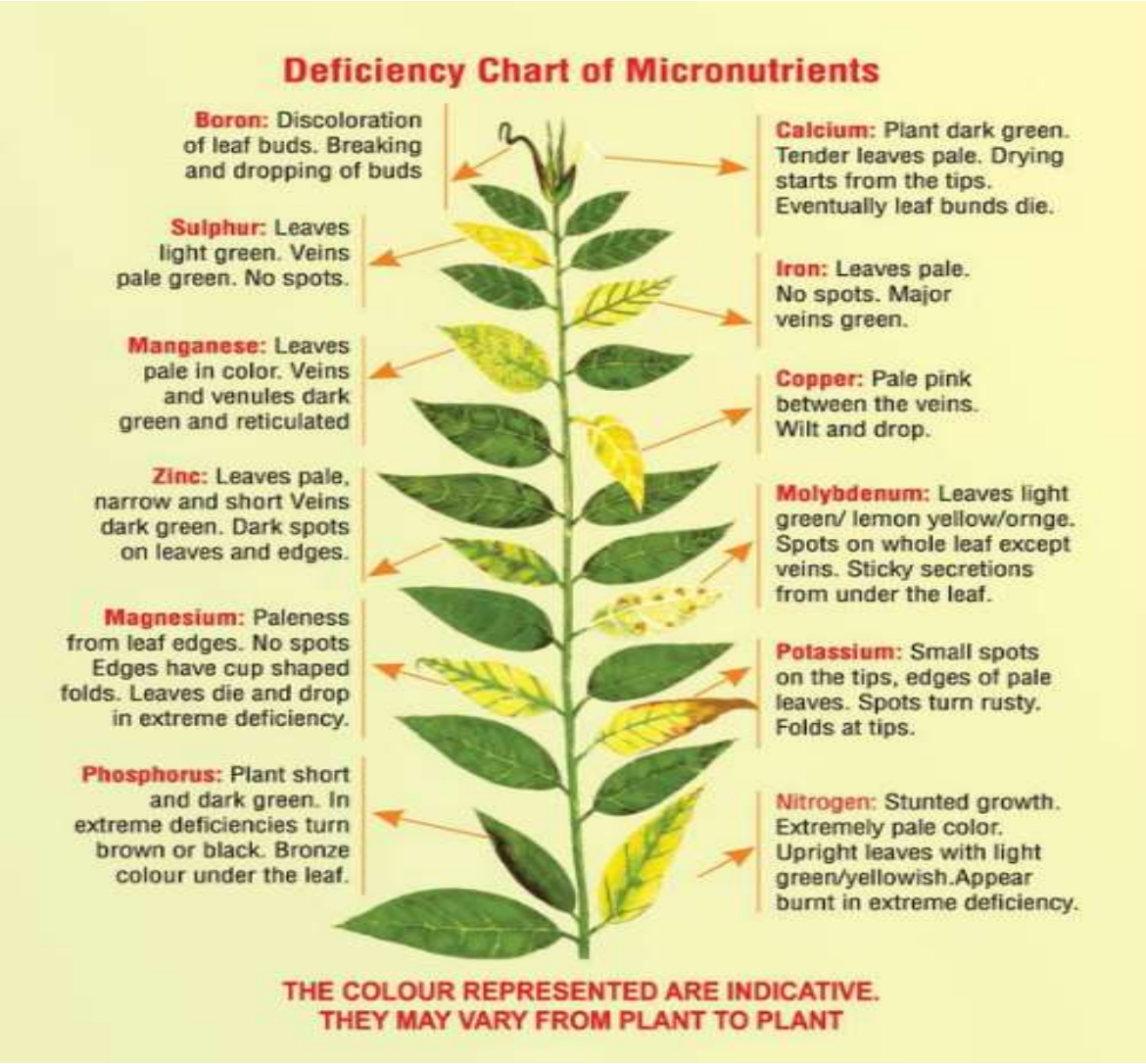
The only method is to grow resistant varieties of tobacco and practicing field sanitation. Cultivars GSH-3, L-2663a, MDS-13, L series 687 to 772, V2809 to 2814 are resistant to mosaic. The field should be weeded thoroughly. After weeding, the workers should wash their hands thoroughly with disinfectant and running water. Regular roguing of diseased plants and weeds in the field should be carried out. Infected plants should be removed and killed by spraying with milk. Dipping the seedling in milk during transplantation greatly reduces the spread of TMV. Seed beds should be located at a great distance from the tobacco warehouses. Care should be taken to avoid contamination through hands and cultivation implements. Susceptible hosts, weed or otherwise in which virus may harbour, should be destroyed.

Mineral deficiency of crop plants

For the normal growth, plants require **major elements** (Nitrogen, Potassium, Phosphorus, Calcium, Magnesium and Sulphur) as well as **minor elements** (Iron, Boron, Manganese, Zinc, Copper, Molybdenum, Chlorine).

When they are present in the plant in amounts smaller than the minimum levels required for normal growth, the plants suffer from several disorders which manifest as various external and internal symptoms on any or all organs of plants, including leaves, stems, roots, flowers, fruits and seeds.

Nutrient deficiencies cause chronic disease in plants. When nutrients are lacking, important molecules like chlorophyll, DNA, RNA, proteins, and lipids cannot be manufactured. Enzymes may not carry out important chemical transformations. In general, plant growth is slowed, and susceptibility to disease may increase. Flowering potted plants may be dwarfed, develop chlorosis or necrosis, have fewer flowers, and otherwise be unattractive.



1. **Nitrogen:**

It is easily leached and must be supplied to plants frequently to prevent deficiency. A general chlorosis of the entire surface of older leaves, progressing upwards, is the most common symptom of nitrogen deficiency. Leaves may be reduced in size, internodes are shortened, and eventually a general loss of vigour or growth occurs. Ex: Red leaf of cotton

2. **Phosphorus**

One of the first symptoms of phosphorus deficiency is the production of small leaves and shortened internodes. Older leaves may lose their shine and become dull and eventually chlorotic. Green pigments are lost, so that red, yellow, and blue pigments show through, especially near main veins on the underside of leaves. Ex: Dwarfing of cotton

3. **Potassium**

Leaf and stem size are often reduced in plants that are deficient in potassium. The foliage remains its normal colour on some plants; on others, necrosis and chlorosis occur, developing first on older leaves Ex: Cotton rust leaf spot of alfa alfa.

4. **Magnesium**

A general reduction in plant vigour and reduced leaf size are common symptoms of magnesium deficiency. Interveinal and marginal chlorosis and necrosis also occur, developing first on older leaves. Chlorosis occurs in a marginal V-shaped pattern. The chlorosis is a bronze colour, and veins remain dark green. Premature senescence of older leaves may occur in mild cases. Ex: Sand drown disease in tobacco

5. **Calcium**

Deficiency is not common in foliage or flowering plants. Small yellow lesions form on the basal half of older leaves of calcium-deficient plants. Water-soaked spots often develop within the chlorotic areas. Symptoms progress into younger leaves, and the chlorotic spots become necrotic, so that leaves sometimes abscise prematurely. Ex: Blossom end rot or tomato, wither tip of flaz.

6. **Iron**

Can become deficient under interiors cape conditions and when the pH of the growing medium is above 7. The occurrence of iron deficiency is largely dependent on the specific requirements of the plant. Chlorosis of the youngest leaves, often with the veins remaining green, is the most common symptom of iron deficiency. Yellowing, stunting, and abscission of new leaves can also occur. Soil pH can influence the availability of iron to plants and should be monitored periodically. The ability of roots to absorb iron is reduced by poor root health caused by inadequate soil aeration resulting from excess soil water.

7. **Sulphur**

Sulphur deficiency is rare under normal conditions of plant production. An overall chlorosis of new leaves occurs. This symptom is easily confused with the chlorosis caused by nitrogen deficiency in some plants.

8. **Manganese**

Manganese deficiency occurs in some plants such as large palms with deformation and chlorosis of newly emerging leaves as the most obvious symptom. Plants that completely lack manganese can be severely stunted. Ex: Gray speaks disease of Oats Marsh spot on garden pea.

9. **Boron**

Boron deficient plants have shortened internodes, thickened stems, and reduced leaf size. New leaves of deficient are stunted and deformed and become brittle and stiff. Terminal leaves are especially distorted. Ex: Hallow stem of brassica of mango

10. **Copper**

Copper deficiency causes severe distortion and stunting of new growth. Leaves are distorted and dwarfed and sometimes have a hooked appearance, with the edges rolled upward toward the centre. Terminal buds die, and laterals sometimes initiate growth, forming a witches'-broom. Ex: Dieback of citrus

11. **Zinc**

Zinc deficiency has been identified in only one foliage plant, *Chrysalidocarpus lutescens* H. Wendl. (areca palm). Leaves of all ages become uniformly chlorotic and terminal leaves are triangular, stunted, and deformed.

12. Molybdenum

It is needed in small amounts by plants, but the use of soilless media and fertilizers lacking this element can result in deficiencies. Symptoms are similar to those of nitrogen or iron deficiency and ammonium toxicity. Plants may be stunted, leaves are small and chlorotic, and leaf margins may become scorched. Leaves tend to curl upward. Ex: Whiptail disease of cauliflower.

SEED-BORNE DISEASES

Seed is the basic unit in crop production technology. It has attracted the agriculturist even in early days. Seed plays a vital role in associating micro-organisms which prove hazardous for the seed or new plant created from it. Important seed borne pathogen/ microorganism are various fungi, bacteria, viruses, nematodes etc.

The associated micro-organism may be pathogenic, weak parasite or saprophytes. They may be associated internally or externally with the seed or as concomitant contamination as sclerotia, galls, fungal bodies, bacterial ooze, infected plant parts, soil particles etc. mixed with the seed. Seed borne pathogen generally plays a negative in human welfares as well as agriculture production.

Seed borne pathogens causes diseases at various stages of crop growth from germination of seed up to crop maturity and heavy losses have been observed, caused by seed borne pathogen in various crops. Seed borne pathogens causes seed and seedling rots, i.e. pre- and post- emergence losses, diseases at various stages of crop growth like root rot, stem rot, fruit rot, wilt, blight, leaf spot etc. influence the crop stand and ultimate yield. Therefore, the good seed must not be affected by any seed borne pathogen. Pathogen free seed is a factor which needs the maximum attention of farmer for an increase crop production. Thus, detection of plant pathogen from seed and their estimation and management is very important for agriculture production/yield.

List of seed borne diseases

DISEASES	PATHOGEN
Black point of wheat	*Bipolaris sorokiniana*
Brown spot of rice	*Bipolaris oryzae*
Alternaria blight of mustard	*Alternaria brassicae*
Leaf spot of wheat	*Alternaria tritica*
Purple stain of soybean	*Cercospora kikuchi*
Red rot of Sugarcane	*Colletotrichum falcatum*
Black band of Jute	*Botryodiplodia theobromae*
Anthracnose of jute	*Colletotrichum corchori*
Stem rot of jute	*Macrophomina phaseolina*

Ways of Infection of Seed Borne Pathogen:

1. **Externally Seed Borne Pathogen:**

The seed inoculum in such cases is superficial and confined to the surface of seed, usually as adhering propagules, e.g., spores sclerotia, mycelium, bacteria, nematodes, virus particles etc. Contamination of seed surface, especially by fungi is often detectable by direct observation under microscope or by examining seed washing.

2. **Internally Seed Borne Pathogen:**

The inoculum lies with the tissues i.e., this pathogen is carried inside the seed, usually as adhering by vegetative cell, spores, pycnidia, nematodes or virus particles. Dry seed may look perfectly healthy when examined under a binocular microscope and no signs of infection. Seed borne pathogen established with seed coat, testa, pericarp, endosperm and embryo.

3. **Concomitant Contamination:**

The inoculum is present as contamination mixed with seed in the form of infected debris fungal sclerotic, bacterial ooze, nematode cysts, infected soil particles etc. Such contamination is difficult to detect.

Role of unhealthy seed

- Germination failure
- Seedling diseases
- Adult plant infection
- Inoculation of virgin soil
- Introduction of new area
- Foci of primary infection
- Secure virulent physiologic races
- Seed discoloration
- Viability loss
- Lower market value

Transmission of Seed Borne Pathogens or Disease:

Seed plays a vital role in the transmission of pathogens directly or indirectly. It is essential to understand precisely how the organisms are associated with the seed and get transmitted. The type of pathogen transmitted includes seeds of plant (phanerogamic plant parasite), nematodes, fungi, bacteria and viruses.

Plant pathogens are seed transmissible by

(i) adhering to seed surface

(ii) becoming internally established with in the seed and

(iii) Accompanying the seed lot as infected plant debris, soil clad or adhering to containers or otherwise.

Detection Techniques of Seed Borne Pathogens:

Several methods have been developed to detect seed borne micro flora. The method of detection may be general or specific for individual pathogen.

The selection of seed testing method for a particular study is based on certain Objectives of Doyer, (1938).

(i) Testing for quarantine purposes.

(ii) Testing for national seed certification schemes.

(iii) Testing for evaluating the planting value of the seed.

(iv) Testing for storage fungi.

Generally, according to the International seed Testing Association (ISTA) (1999) until and unless otherwise stated, a minimum of 400 seed should be tested for each sample.

Prevention Methods against Seed Borne Pathogens:

An outline of the measures of prevention of diseases due to seed borne pathogens, as modified from Baker (1972), is enumerated below:

(a) Management Practices:

1. Seed source-pathogen free seed.
2. Selection of seed-production area and season when and where the seed is not
3. Likely to carry pathogens.
4. Seed-field inspection.
5. Seed certification.
6. Quarantine.

(b) Cultural Practices in Seed-Production Fields:

1. Sowing methods, e.g., deep sowing and planting.
2. Pathogen control in seed field- control of weed hosts, production including pre- harvest earhead spray with fungicides as in wheat or rice if there is rain.
3. Avoidance of overhead watering -Ditch irrigation, rather than sprinkler irrigation is favourable for seed crops. This is particularly true in semiarid areas where foliage would otherwise remain uninfected.
4. Harvesting method- Delaying harvesting, and various cares taken during harvesting.
5. Eradication of infected host plants- Applicable when a disease is newly introduced in an area.
6. Ageing of seed to utilize the phenomenon that some seeds remain viable for a period longer than the period of survival of the pathogen, as in cucurbits.
7. Treatment of field soil-Occasionally effective.

(c) Curative or Eradicative Measures for Seed Already Contaminated:

1. Seed indexing.
2. Separating procedures.
3. Chemical seed treatment.

Seed treating chemicals with low mammalian toxicity such as antibiotics (aureofungin, blasticidin, etc. against rice blast and brown spot), organic sulphur, systemic fungicides and their combinations (thiram+carboxin; thiram+bavistin, etc.) have been developed to replace organic mercury.

4. Thermotherapy of seed:

The above measures are intended for reduction of established inoculum.

(d) Breeding for Resistance against Seed Transmission:

That the amount of primary inoculum should be limited in seed should be the chief objective of control of the diseases due to seed borne pathogens. Thus, breeding disease resistant varieties is likely to be a very successful measure against these diseases. The integration of different measures would be necessary for the evaluation of a recommendation for any specific case.