B.Sc III Paper III Unit 4
&
B.Sc. I sem. Unit VII (NEP)

Plant Pathology(B.Sc. III)
Disease and Control (B.Sc. I sem.)

Dr.Sanjay Srivastava

Botany Department

Harish Chandra P. G. College

Varanasi (U.P.)

Mobile 9415635846

Email: sanjaychandravns@gmail.com

TOPICS DISCUSSED:

- General points in plant pathology
- Citrus Canker
- Little leaf disease of brinjal
- Red rot of sugarcane
- Tobacco mosaic virus disease
- Yellow vein mosaic of Bhindi

PLANT PATHOLOGY: The term **pathology** is derived from two greek words, **pathos** (suffering) and **logos** (study or discourse) i.e. pathology means the study of suffering. Taking this into account, **plant pathology** means the study of sufferings of plants, i.e. plant diseases.

Plant pathology deals with the study of diseases of agricultural, forestry and horticulture plants.

De Bary (1831-1888) is known as the **father of modern plant pathology**.

E.J.Butler is known as the **father of mycology and plant pathology in India**.

Names of Some Indian plant pathologists: **J.F.Dastur** (late blight of potato), **G.S.Kulkarni** (Downy mildew of bajra and jowar and smut of jowar, **K.C.Mehta** (problem of cereal rust in India) Mehta studied the Indian rust cycle and reported that barberry plays no role in it, **K.S.Bhargava, K.S.Thind, Kamal** and many others.

Plant diseases which earned their name in history:

Irish Famine (1845-46) occurred due to **late blight of potato caused by *Phytophthora infestans***

Bengal famine(1942-43) occurred due to leaf spot disease of paddy caused by ***Helminthosporium oryzae***

Koch's postulates: The pathogenicity test

This is done in order to confirm that a particular microorganism is the real cause of the disease.

The steps are as under:

1. The microorganism must be associated with the disease in all the diseased plants.
2. The microorganism should be isolated from pure culture and when inoculated on healthy plant should produce the same symptoms as observed in field.
3. The microorganism could be isolated from artificially inoculated plant and show same growth characteristics in pure culture.

CITRUS CANKER

ORIGIN AND INCIDENCE

- Disease originated in China.
- Its incidence is common in countries like India, China, Japan etc.

SYMPTOMS

- Symptoms develop on all parts such as leaf, fruit, twig, thorn, stem etc.
- Raised white or grey spots first appear on lower surface of leaf.
- Later both the surface are covered.
- Spots develop crater like structure in the centre.
- Spot are surrounded by yellow halo.

CAUSAL ORGANISM

Bacterium *Xanthomonas axonopodis* pv. *citri*

Rod shaped, Gram negative, 1.5-2.0 x 0.5-0.75 µm, form capsule and chain, endospore not formed.

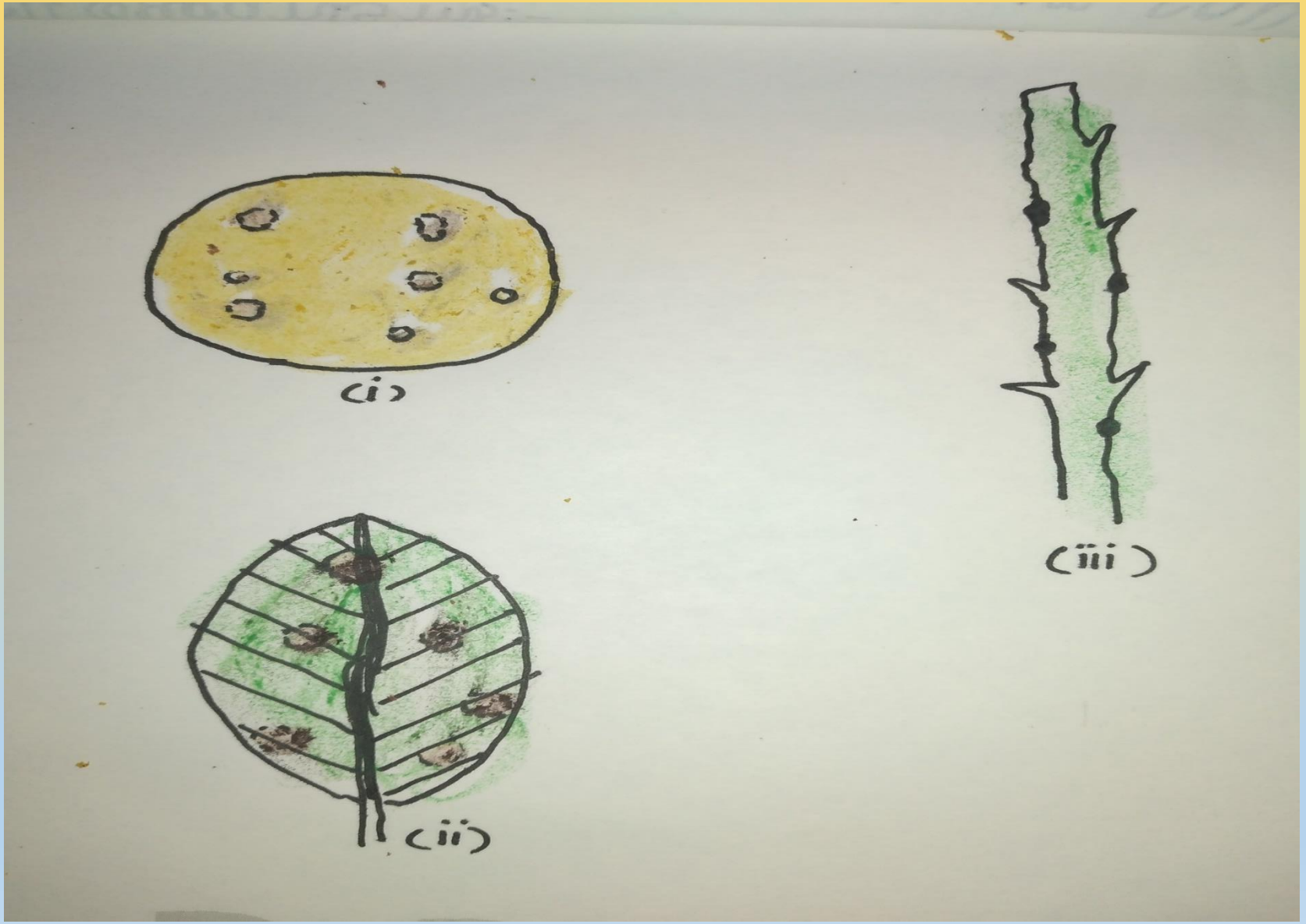
Enters the host through stomata or wounds, multiplies in the intercellular space, digests middle lamella and then establishes itself in the cortical cells.

Moderate temperature and moist weather are favourable.

Disease spread from the canker by rain splash, insect or by man through diseased nursery stock.

CONTROL

Diseased plant destruction, use of disease free nursery stock, spray of 1% Bordeaux mixture/neem cake/streptomycin, growing disease resistant varieties.



Citrus Canker on lemon : Symptoms i) on fruit ii) on leaf iii) on stem

LITTLE LEAF DISEASE OF BRINJAL

The disease was first reported from Coimbatore (India) in 1939.
It is one the most serious disease of brinjal (egg plant).
The disease is prevalent in all areas in India where brinjal is grown.
It results in great loss (more than 90%) in yield.

SYMPTOMS:

- Chlorosis in young leaves.
- Axillary buds stimulated to grow into short branches (excessive branching).
- Reduced size of leaves along with shortened internodes and petioles (leaves almost look sessile).
- This results in stunted growth and bushy appearance of plant.
- Phylloid flowers (abnormal development of flower parts into leafy structures) are produced.
- When early infection occurs, no fruits are formed.
- In late infection, fruits formed are shrivelled and malformed.

CAUSAL ORGANISM:

The disease is caused by Phytoplasma (earlier called MLO i.e., mycoplasma like organism).
These are obligate intracellular parasites of phloem tissue.
Phytoplasma are devoid of cell wall and have pleiomorphic shapes.

DISEASE CYCLE

They are transmitted by grafting and through leaf hopper vector *Hishimonas phycitis.*
SAP TRANSMISSION of disease in not possible.

MANAGEMENT

The infected plants should be removed from field and burnt.
Spraying of Metasystox (6 ml in 9 litres of water) several times.
Treatment with antibiotics like Tetracycline provides only temporary relief.
Use of resistant varieties.

RED ROT OF SUGARCANE

Red rot of sugarcane is one of the oldest disease of sugarcane.
It severely affects sugarcane crop in tropical and sub-tropical countries.
In India, the disease resulted in epidemic in Bihar and U.P. in the year 1939-40 and 1946-47.
The disease was first reported in 1893 in Jawa by Went.
He named it " red smut" disease.
Later, Butler gave the name RED ROT to the disease.
The disease occurs in all parts of India.

SYMPTOMS

Symptoms usually appear quite late i.e. when plant is full grown with
Sucrose formed.
The spindle leaves dry and loose colour.
Dark reddish areas (blood red lesions) with straw coloured central portion develop alon
Dark dots appear amidst the infection courts.
These dark spots are actually the acervuli (the fruiting bodies of the pathogen).
At later stage of the disease the stem shows symptoms.
Stem gets shrivelled and the rind shrinks.
When split open, the pith of stem shows reddening of tissues.
Reddening is due to a red coloured substance secreted by the host as a reaction to the entry of the pathogen.
Horizontal bands of white areas are found in the reddened pith region.
The cane juice develops a bad odour. This is due to fermentation of sucrose and its conversion into glucose and alcohol by the pathogen.

CAUSAL ORGANISM *Colletotrichum falcatum*

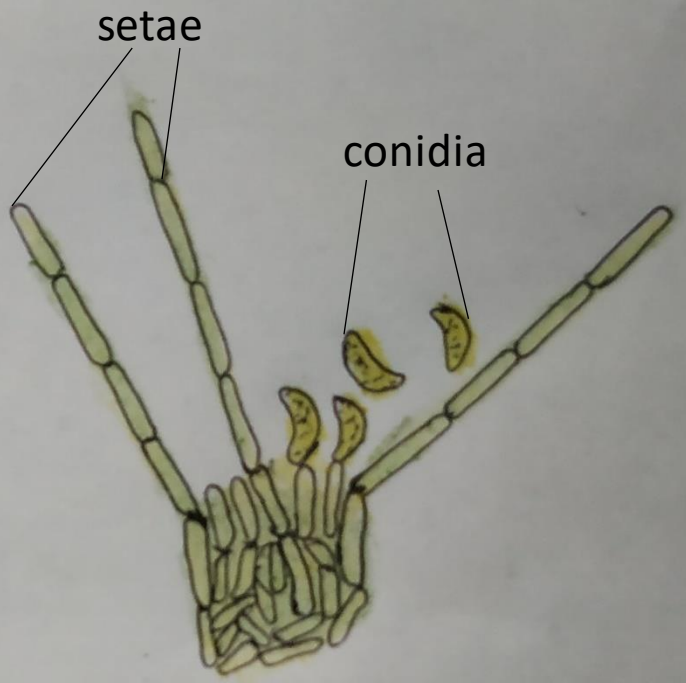
Classification:

Kingdom……Fungi
Division……..Eumycota
Sub-division….Deuteromcotina
Class……..Coelomycetes
Order……..Melanconiales
Family……..Melanconiaceae
Genus……..*Colletotrichum*
Species……..*falcatum*

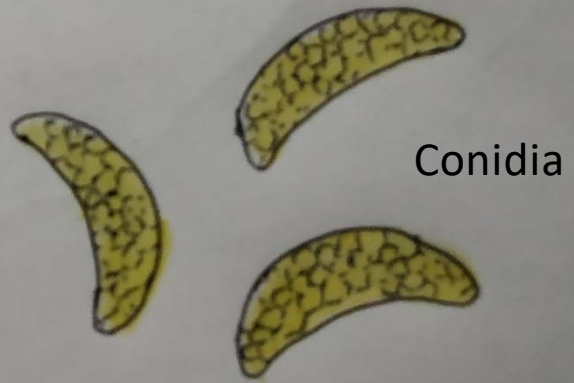
Perfect stage of the fungus is *Glomerella tucumanensis* (Ascomycetes).
Mycelium of the fungus is hyaline, septate, profusely branched, inter as well as intracellular.
The hyphae aggregate under the host epidermis and develop fruiting bodies on host surface called acervuli.
Each acervulus contains conidiophores, conidia and setae.
Setae (singular seta) are sterile structures which are long, septate, rigid, dark coloured and pointed at apex.
Conidiophores are small, single celled, club shaped, hyaline structures which are found aggregated in the acervulus.
Conidia are produced at the tip of conidiophores.
Conidia are falcate (sickle shaped), single celled, produced singly at the tip of conidiophore.
Each conidium contains a single large oil globule.

Acervulus on leaf

Brown stripes on stem



Acervulus in sectional view



Conidia

Red rot of sugarcane

DISEASE CYCLE

Perennation:

The fungus perennates with the help of mycelia in the diseased canes and through chlamydospores present in the plant debris in soil.

Primary Infection:

If diseased canes are used as seed sets (planting material), then the fungus grows systemically with the growing seedling. Apart from this, mycelia and chlamydospores present in the plant debris also serve as primary inoculum. They infect through the roots of growing plant. Ratoon cropping is also responsible for primary infection.Symptoms first appear on leaves. Conidia are short lived and therefore not a source of primary infection.

Secondary Infection:

Conidia produced on infected plants serve as secondary inoculum and bring about secondary infection.
Conidia are disseminated through wind, water or insects.
Germ tubes of conidia enter the host through wounds caused by insects.

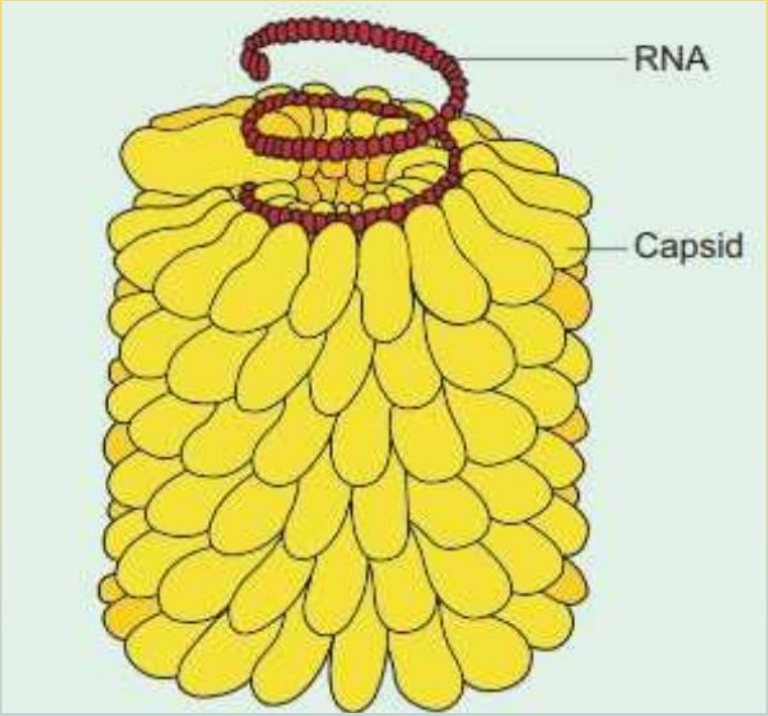
Predisposing factors:

High atmospheric humidity.
Poor cultural practices.
Water logging in field.
Repeated cultivation of susceptible varieties.

MANAGEMENT:

Disease canes should be dug out and burnt.
Crop ratooning should be avoided in disease prone areas.
Crop rotation should be practised.
Use of healthy and disease free seed setts.
Seed setts should be treated with Carbendazim @ 2.5 gm/ litre of water for 30 minutes.
Hot water treatment of setts (52^{o} C for 8 hours, 54^{o} C for 2 hours).).
Hot air treatment of setts (54^{o} C for 3 hours).
Use of resistant varieties eg. Co 7314, Co 6907, Co 7219, CoLk 7710, Bo 91, Bo 99 etc.

TOBACCO MOSAIC VIRUS DISEASE



Disease is worldwide in distribution.
TMV reduces both the quality and quantity of tobacco crop.

SYMPTOMS:

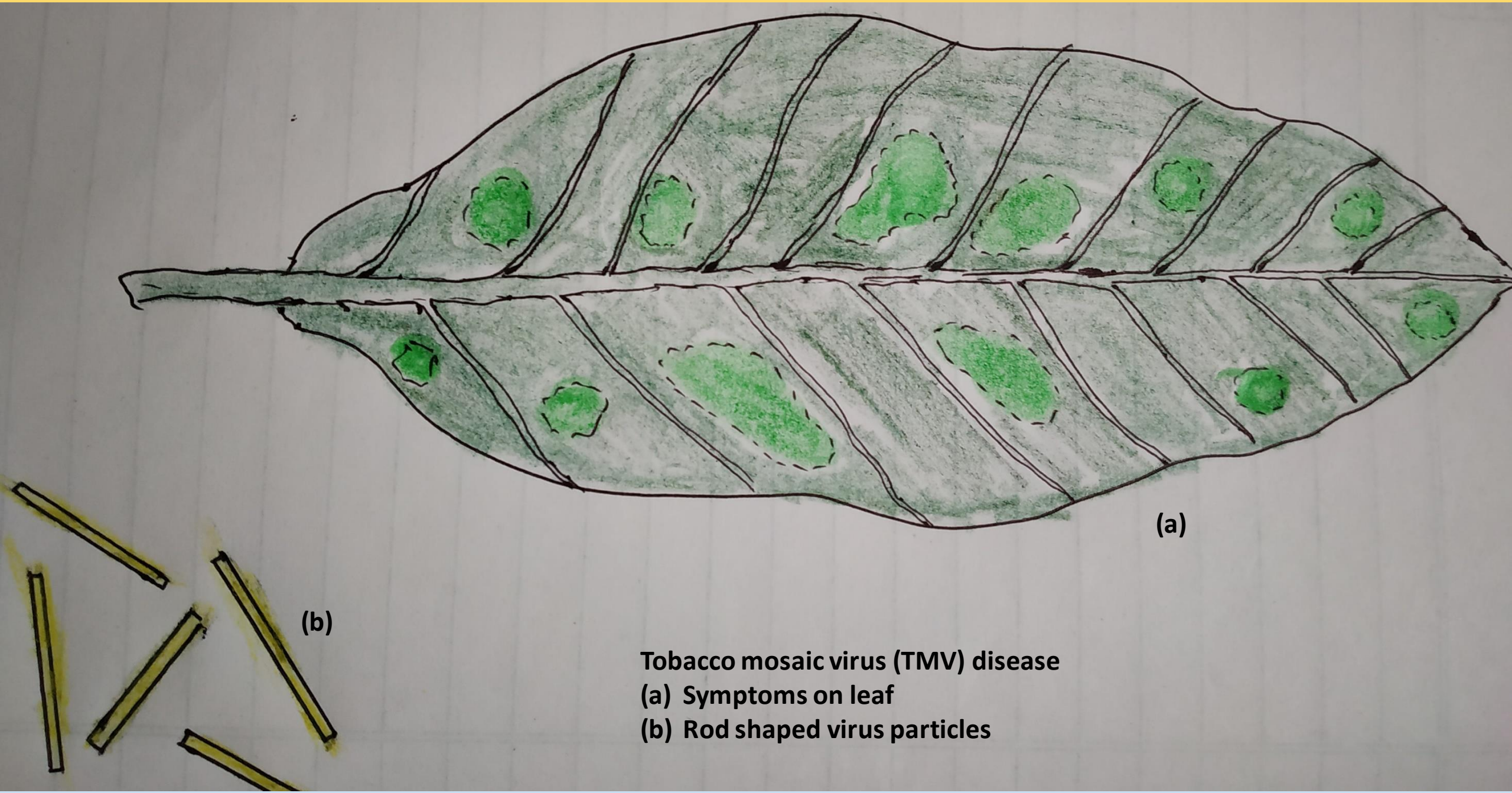
Mottled dark green and light green areas with sharp boundaries on leaves.
Dark green areas are darker in colour than healthy tissues and are slightly raised.
These symptoms appear in young leaves. Later the plant shows stunted look.
Curling and distortion of leaves also observed.

CAUSAL AGENT:

Tobacco Mosaic Virus belonging to Tobamovirus group.
Virus is rod shaped measuring 300 nm x 15 nm.
It has a single stranded RNA (5.6%) in the centre surrounded by a protein envelope (94.4%).
TMV is a thermostable virus.
Purified TMV can retain its infectious nature even after 50 years of storage.

DISEASE CYCLE:

TMV is sap transmissible i.e. it is easily transmitted when a diseased leaf is rubbed against a healthy leaf.
The virus gets transmitted through agricultural tools, hands of plantation workers and smoked cigerettes.
Virus enters a plant through wounds.



Tobacco mosaic virus (TMV) disease
(a) Symptoms on leaf
(b) Rod shaped virus particles

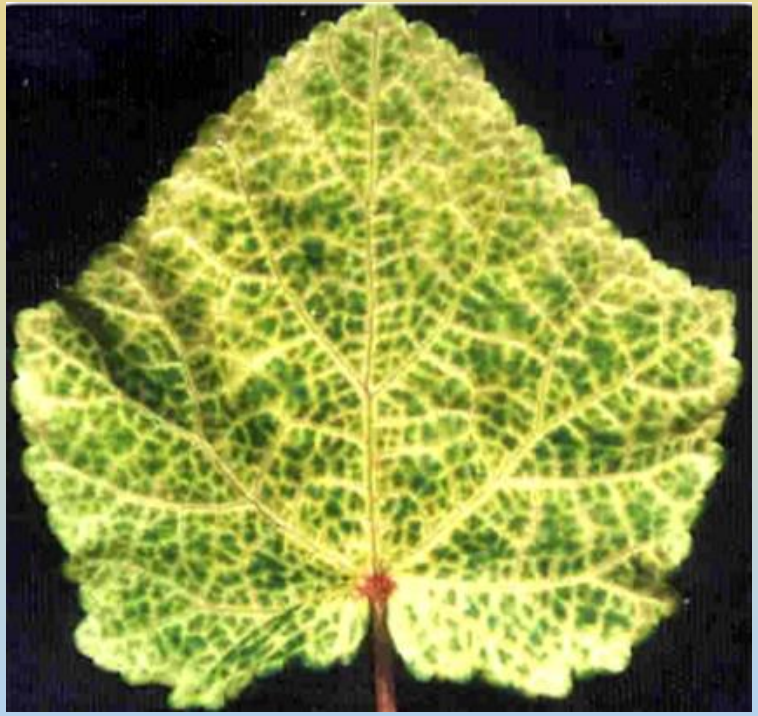
PRIMARY INFECTION:
Primary infection occurs through contaminated hands of workers.
Virus enters through wounds and multiplies in the host cells and spreads in the entire plant body.
Virus moves from one cell of the host to another through plasmodesmata.

SECONDARY INFECTION:
Secondary infection occurs through "suckering" and agricultural implements.
Secondary infection continues throughout the cropping season.

MANAGEMENT:
Horticultural practices – All tools should be washed with soap.
TMV infected soil should also be avoided.
Cross Protection – Inoculation of young plants by a milder strain of virus to protect them from subsequent infection by a severe strain.
Transgenic plants can also help in avoiding the disease.
Plantation workers should be asked to abstain from chewing and smoking tobacco.
Field sanitation by destruction of diseased plants and plant debris should be done.
TMV resistant varieties should be grown.

YELLOW VEIN MOSAIC OF BHINDI

It is the most common disease of bhindi or okra (*Abelmoscus esculentus*).
If plants get infected 34-35 days after germination then the plants develop few leaves and fruit.
Total loss can reach up to 94%.



SYMPTOMS:
Typical symptoms are vein-clearing and vein-chlorosis.
First the small marginal veins show vein-clearing followed by vein-chlorosis.
Later, the entire leaf shows a yellow network of veins.
Thickening of veins on the lower surface of leaf is observed.
Fruits are dwarfed, malformed and yellow green in colour.

CAUSAL AGENT:
Causal agent is Yellow Vein Mosaic Virus (YVMV).
Virus is small in size and spherical in shape.

DISEASE CYCLE:
The virus perennates in several weed hosts eg. *Ageratum* sp., *Croton sparsiflora*, *Malvastrum tricuspidatum* etc.

YVMV is sap transmissible.
It is also transmitted by white fly (Bemisia tabaci).
The insect virus acquires the virus in 15 to 30 minutes.
It undergoes an incubation period of 7 hours in insect body.
After the incubation period the virus can be inoculated in a healthy plant.
Both primary and secondary infection are brought by insect vectors.

MANAGEMENT:
Destruction of all weed hosts of the virus.
Diseased bhindi plants should be dug out and burnt.
Insect vector population can be controlled by spraying insecticide like follidol (0.3%).
Use of resistant varieties (Parbhani kranti, Arka Abhay, Arka Anamika, Varsha Uphar etc.)
Spraying monocrotophos 1.5 ml/litre of water can restrict the disease spread.
Application of Chlorpyriphos 2.5 ml + neem oil 2 ml/ litre of water also helps in disease control.

References

- B.Sc. I sem. Botany book by Singh, Pande and Jain
- Plant pathology by R.P.Singh
- Plant Pathology by AGRIOS
- Plant Pathology by R.S Singh