AGS 322- Diseases of field and horticultural crops and their management

1. Diseases of Wheat

Black or stem rust - *Puccinia graminis tritici*

**Symptoms**

Symptoms are produced on almost all aerial parts of the wheat plant but are most common on stem, leaf sheaths and upper and lower leaf surfaces. Uredial pustules (or sori) are oval to spindle shaped and dark reddish brown (rust) in color. They erupt through the epidermis of the host and are surrounded by tattered host tissue. The pustules are dusty in appearance due to the vast number of spores produced. Spores are readily released when touched.

As the infection advances teliospores are produced in the same pustule. The color of the pustule changes from rust color to black as teliospore production progresses. If a large number of pustules are produced, stems become weakened and lodge. The pathogen attacks other host (barberry) to complete its life cycle. Symptoms are very different on this woody host. Other spores are *Pycnia (spermagonia)* produced on the upper leaf surface of barberry which appears as raised orange spots. Small amounts of honeydew that attracts insects are produced in this structure. *Aecia*, produced on the lower leaf surface, are yellow. They are bell-shaped and extend as far as 5 mm from the leaf surface.

Brown or leaf rust - *Puccinia triticina (P. recondita)*

**Symptom**
The most common site for symptoms is on leaf blades, however, sheaths, glumes and awns may occasionally become infected and exhibit symptoms. Uredia are seen as small, circular orange blisters or pustules on the upper surface of leaves.

Orange spores are easily dislodged and may cover clothing, hands or implements. When the infection is severe leaves dry out and die. Since inoculum is blown into a given area, symptoms are often seen on upper leaves first. As plants mature, the orange urediospores are replaced by black teliospores. Pustules containing these spores are black and shiny since the epidermis does not rupture. Yield loss often occurs as a result of infection by *Puccinia recondita* f. sp. *tritici*. Heavy infection which extends to the flag leaf results in a shorter period of grain fill and small kernels.

**Yellow or stripe rust - *Puccinia striiformis***

Mainly occur on leaves than the leaf sheaths and stem. Bright yellow pustules (Uredia) appear on leaves at early stage of crop and pustules are arranged in linear rows as stripes. The stripes are yellow to orange yellow. The teliospores are also arranged in long stripes and are dull black in colour.
Pathogen

The uredospores of rust pathogen are almost round or oval in shape and bright orange in colour. The teliospores are bright organge to dark brown, two celled and flattened at the top. Sterile paraphyses are also present at the end of sorus.

Disease Cycle

In India, all these rusts appear in wheat growing belt during Rabi crop season. Uredosori turn into teliosori as summer approaches. The inoculum survives in the form of uredospores / teliospores in the hills during off season on self sown crop or volunteer hosts, which provide an excellent source of inoculum. In India, role of alternate host (Barberis) is not there in completing the life cycle.

The fungus is inhibited by temperatures over 20˚C although strains tolerant of high temperatures do exist. The complete cycle from infection to the production of new spores can take as little as 7 days during ideal conditions. The disease cycle may therefore be repeated many times in one season. During late summer, the dark teliospores may be produced. These can germinate to produce yet another spore type, the basidiospore, but no alternate host has been found. Although the teliospores seem to have no function in the disease cycle they may contribute to the development of new races through sexual recombination.
Life cycle of *Puccinia graminis*

**Favourable Conditions**

- Low temperature (15-20°C) and high humidity during November – December favour black and brown rusts.
- Temperature less < 10° favours yellow rusts.

**Disease cycle**

Uredospores and dormant mycelium survive on stubbles and straws and also on weed hosts and self sown wheat crops. Wind borne uredospores from hills are lifted due to cyclonic winds and infect the crop in the plains during crop season.

**Management**

- Mixed cropping with suitable crops.
- Avoid excess dose of nitrogenous fertilizers.
- Spray *Zineb* at 2.5 kg/ha or *Propiconazole* @ 0.1 %.
- Grow resistant varieties like PBW 343, PBW 550, PBW 17

Loose smut - *Ustilago nuda tritici* (*Ustilago tritici*)

**Symptoms**

It is very difficult to detect infected plants in the field until heading. At this time, infected heads emerge earlier than normal heads. The entire inflorescence is commonly affected and appears as a mass of olive-black spores, initially covered by a thin gray membrane. Once the membrane ruptures, the head appears powdery.

Spores are dislodged, leaving only the rachis intact. In some cases remnants of glumes and awns may be present on the exposed rachis. *Smutted heads* are shorter than healthy heads due to a reduction in the length of the rachis and peduncle. All or a portion of the heads on an infected plant may exhibit these symptoms. While infected heads are shorter, the rest of the plant is slightly taller than healthy plants. Prior to heading affected plants have dark green erect leaves. *Chlorotic streaks* may also be visible on the leaves.

**Disease Cycle**

Ears of infected plants emerge early. The spores released from the infected heads land on the later emerging florets and infect the developing seed. Infection during flowering is favored
by frequent rain showers, high humidity and temperature. The disease is internally seed borne, where pathogen infects the embryo in the seed.

Management

Treat the seed with Vitavax @ 2g/kg seed before sowing. Burry the infected ear heads in the soil, so that secondary spread is avoided.

Flag smut - *Urocystis tritici*

Symptoms

The symptoms can be seen on stem, clum and leaves from late seedling stage to maturity. The seedling infection leads to twisting and drooping of leaves followed by withering. Grey to grayish black sori occurs on leaf blade and sheath. The sorus contains black powdery mass of spores.
Pathogen

Aggregated spore balls, consisting 1-6 bright globose, brown smooth walled spores surrounded by a layer of flat sterile cells.

Favourable Conditions

- Temperature of 18-24°C.
- Relative humidity 65% and above.

Disease cycle

Seed and soil borne. Smut spores are viable for more than 10 years.

Management

- Treat the seeds with carboxin at 2g/kg.
- Grow resistant varieties like Pusa 44 and WG 377.

Hill bunt or Stinking smut - *Tilletia caries* / *T.foetida*
**Symptoms**

The fungus attacks seedling of 8-10 days old and become systemic and grows along the tip of shoot. At the time of flowering hyphae concentrate in the inflorescence and spikelets and transforming the ovary into smut sorus of dark green color with masses of chlamydospores. The diseased plants mature earlier and all the spikelets are affected.

**Pathogen**

Reticulate, globose and rough walled. No resting period. Germinate to produce primary sporidia which unite to form ‘H’ shaped structure.

**Life cycle**

The spores on the seed surface germinate along with the seed. Each produces a short fungal thread terminating in a cluster of elongated cells. These then produce secondary spores which infect the coleoptiles of the young seedlings before the emergence of the first true leaves. The mycelium grows internally within the shoot infecting the developing ear. Affected plants develop apparently normally until the ear emerges when it can be seen that grain sites have been replaced by bunt balls. In India disease occurs only in Northern hills, where wheat is grown.
Favourable Conditions
- Temperature of 18-20°C.
- High soil moisture.

Disease cycle
- Externally seed borne

Management
- Treat the seeds with carboxin or carbendazim at 2g/kg.
- Grow the crop during high temperature period.
- Adopt shallow sowing.
- Grow resistant varieties like Kalyan sona, S227, PV18, HD2021, HD4513 and HD4519.

Karnal bunt - *Neovassia indica*

Symptoms
Symptoms of Karnal bunt are often difficult to distinguish in the field due to the fact that incidence of infected kernels on a given head is low. There may be some spreading of the glumes
due to sorus production but it is not as extensive as that observed with common bunt. Symptoms are most readily detected on seed after harvest.

The black sorus, containing dusty spores is evident on part of the seed, commonly occurring along the groove. Heavily infected seed is fragile and the pericarp ruptures easily. The foul, fishy odor associated with common bunt is also found with karnal bunt. The odor is caused by the production of trimethylamine by the fungus. Seed that is not extensively infected may germinate and produce healthy plants.

**Foot rot - Pythium graminicolum** and **P. arrhenomanes**

Symptoms
The disease mainly occurs in seedlings and roots and rootlets become brown in colour. Seedlings become pale green and have stunted growth. Fungus produces sporangia and zoospores and oospores.

**Favourable Conditions**

Wet weather and high rainfall.

**Disease cycle**

Through soil and irrigation water.

**Management**

- Follow crop rotation.
- Treat the seeds with Carboxin or Carbendazim at 2g/kg.

**Powdery mildew - *Erysiphe graminis var. tritici***

**Symptoms**

Greyish white powdery growth appears on the leaf, sheath, stem and floral parts. Powdery growth later become black lesion and cause drying of leaves and other parts.

Pathogen

Fungus produces septate, superficial, hyaline mycelium on leaf surface with short conidiophores. The conidia are elliptical, hyaline, single celled, thin walled and produced in
chains. Dark globose cleistothecia containing 9-30 ascii develop with oblong, hyaline and thinwalled ascospores.

Disease cycle

Fungus remains in infected plant debris as dormant mycelium and asci. Primary spread is by the ascospores and secondary spread through airborne conidia.

Favourable Conditions

- Temperature of 20-21°C.

Management

- Spray Wettable Sulphur 0.2% or Carbendazim @ 500 g/ha

Leaf blight - *Alternaria triticina* / *Bipolaris sorokiniana*

Symptoms

Reddish brown oval spots appear on young seedlings with bright yellow margin. In severe cases, several spots coalesce to cause drying of leaves. It is a complex disease, having association of *A. triticina, B. sorokiniana* and *A. alternate.*
**Disease cycle**

Primary spread is by externally seed-borne and soil borne conidia. Secondary spread by air-borne conidia.

**Favourable Conditions**

- Temperature of 25°C and high relative humidity.

**Management**

- Spray the crop with Mancozeb or Zineb at 2 kg/ha.

**Other minor diseases**

Helminthosporium leaf spot: *Helminthosporium* spp.

Tundu or yellow ear rot: *Corynebacterium tritici* + *Anguina tritici*

Seedling blight: *Rhizoctonia solani* and *Fusarium* sp

Sclerotinia rot: *Sclerotinia sclerotiorum*

Molya disease: *Heterodera avenae* (Nematode)
2. Diseases of Sugarcane

Red rot - *Colletotrichum falcum* (Perfect stage: *Physalospora tucumanensis*)

**Symptoms**

The first external symptom appears mostly on third or fourth leaf which withers away at the tips along the margins. Typical symptoms of red rot are observed in the internodes of a stalk by splitting it longitudinally. These include the reddening of the internal tissues which are usually elongated at right angles to the long axis of the stalk. The presence of cross-wise white patches are the important diagnostic character of the disease. The diseased cane also emits acidic-sour smell. As the disease advances, the stalk becomes hollow and covered with white mycelial growth.

Later the rind shrinks longitudinally with minute black, velvety fruiting bodies protruding out of it. The pathogen also produces tiny reddish lesions on the upper surface of leaves with dark dots in the centre. The lesions are initially blood red with dark margins and later on with straw coloured centres. Often the infected leaves may break at the lesions and hang down, with large number of minute black dots.

**Pathogen**

The fungus produces thin, hyaline, septate, profusely branched hyphae containing oil droplets. The fungus produces black, minute velvety acervuli with long, rigid bristle-like, septate setae. **Conidiophores** are closely packed inside the acervulus, which are short, hyaline and single celled. The **conidia** are single celled, hyaline, falcate, granular and guttulate. Fungus
also produces large number of globose and dark brown to black perithecia with a papillate ostiole.

Asci are clavate, unitunicate and eight-spored. Large number of hyaline, septate, filiform paraphyses is also present among asci. Ascospores are ellipsoid or fusoid, hyaline, straight or slightly curved and unicellular which measure 18-22 µm x 7-8µm.

**Favourable Conditions**
- Monoculturing of sugarcane.
- Successive ratoon cropping.
- Water logged conditions and injuries caused by insects.

**Disease cycle**

The fungus is sett-borne and also persists in the soil on the diseased clumps and stubbles as chlamydospores and dormant mycelium. The primary infection is mainly from infected setts. Secondary spread in the field is through irrigation water and cultivation tools. The rain splash, air currents and dew drops also help in the spread of conidia from the diseased to healthy plants in the field. The fungus also survives on collateral hosts *Sorghum vulgare*, *S. halepense* and *Saccharum spontaneum*. If the conidia settle on the leaves they may germinate and invade the leaves through various types of wounds. Stem infection may take place through insect bores and root primordia. The soil-borne fungus may also enter the healthy setts through cut-ends, and
cause early infection of the shoots. Though the perfect stage of the fungus has been observed in nature, the role of ascospores in the disease cycle is not understood.

Management

- Adopt crop rotation by including rice and green manure crops.
- Select the setts from the disease free fields or disease free areas.
- Avoid ratooning of the diseased crop.
- Soak the setts in 0.1% Carbendazim or Triademefon 0.05% solution for 15 minutes before planting.
- Grow resistant varieties CO 62198, CO 7704 and moderately resistant varieties CO 8001, CO8201.
- Setts can be treated with aerated steam at 52 °C for 4 to 5 hours and by moist hot air at 54°C for 2 hours.

Smut - *Ustilago scitaminea*

Symptoms

It is a culmiculous smut. The affected plants are stunted and the central shoot is converted into a long whip-like, dusty black structure. The length of the whip varies from few inches to several feet. In early stages, this structure is covered by a thin, white papery membrane. The whip may be straight or slightly curved.

On maturity it ruptures and millions of tiny black smut spores (teliospores) are liberated and disseminated by the wind. Affected plants are usually thin, stiff and remain at acute angle. The whip like structure, representing the central shoot with its various leaves, may be produced by each one of the shoots/tillers arising from the clump.
The smutted clumps also produce mummified arrows in which lower portion consisted of a normal inflorescence with typical flowers and the upper portion of the rachis is converted into a typical smut whip. Occasionally smut sori may develop on the leaves and stem.

**Pathogen**

The fungal hyphae are primarily intercellular and collect as a dense mass between the vascular bundles of host cell and produce tiny black spores. The thin membrane which covers the smut whip represents the host epidermis. The smut spores are light brown in colour, spherical, echinulated and measuring 6.5-8.5 \( \mu \text{m} \) in diameter. Smut spores germinate to produce 3-4 celled, hyaline promycelium and produce 3-4 sporidia which are hyaline and oval shaped with pointed ends.

**Favourable Conditions**

- Monoculturing of sugarcane.
- Continuous ratooning and dry weather during tillering stage.

**Disease cycle**

Teliospores may survive in the soil for long periods, upto 10 years. The spores and sporidia are also present in the infected plant materials in the soil. The smut spores and dormant mycelium also present in or on the infected setts. The primary spread of the disease is through diseased seed-pieces (setts). In addition, sporidia and spores present in the soil also spread through rain and irrigation water and cause soil-borne infection. The secondary spread in the
field is mainly through the smut spores developed in the whips, aided by air currents. The fungus also survives on collateral hosts like *Saccharum spontaneum*, *S. robustum*, *Sorghum vulgare*, *Imperata arundinacea* and *Cyperus dilatatus*.

**Management**

- Plant healthy setts taken from disease free area.
- Remove and destroy the smutted clump (collect the whips in a thick cloth bag/polythene bag and immerse in boiling water for 1 hr to kill the spores).
- Discourage ratooning of the diseased crops having more than 10 per cent infection.
- Follow crop rotation with green manure crops or dry fallowing.
- Grow redgram as a companion crop between 2 rows of sugarcane.
- Grow resistant varieties like Co 7704 and moderately resistant varieties COC 85061 and COC 8201.

**Sett rot or Pineapple disease - *Ceratocystis paradoxa***

**Symptoms**

The disease primarily affects the setts usually two to three weeks after planting. The fungus is soil-borne and enters through cut ends and proliferates rapidly in the parenchymatous tissues. The affected tissues first develop a reddish colour which turns to brownish black in the later stages. The severely affected setts show internodal cavities covered with the mycelium and abundant spores. A characteristic pineapple smell is associated with the rotting tissues. The setts may decay before the buds germinate or the shoots may die after reaching a height of about 6-12 inches. Infected shoots are stunted.
Pathogen

The fungus produces both macroconidia and microconidia. Conidiophores are linear, thin walled with short cells at the base and a long terminal cell. The microconidia are hyaline when young but become almost black at maturity. They are thinwalled, cylindrical and produced endogenously in chains in the long cells of conidiophores and pushed out in succession. Macroconidia are produced singly or in chains on a short, lateral conidiophores. Macroconidia are spherical or elliptical or truncate or pyriform and are hyaline to olive green or black measuring 16-19x10-12 um.

The fungus also produces chlamydospores on short lateral hyphae in chains, which are oval, thick walled and brown in colour. The perithecia are flask shaped with a very long neck. The bulbous base of the perithecium is hyaline or pale yellow, 200-300µm in diameter and ornamented with irregularly shaped, knobbed appendages. The ostiole is covered by numerous pale-brown, erect tapering hyphae. Asci are clavate and measures 25x10µm and ascospores are single celled, hyaline, ellipsoid, more convex on one side, measures 7-10 x 2.5-4µm.

Favourable Conditions

- Poorly drained fields.
- Heavy clay soils
- Temperature of 25-30°C
- Prolonged rainfall after planting.

Disease Cycle

The fungus survives as conidia and chlamydospores in the soil and in the infected, burried cane tissues. The inoculum moves from field to field through wind-borne conidia or irrigation or rain water. Inside the sett it spreads rapidly through the parenchymatous tissues and causes sett rot.

The insects like cane borer (Diatraea dyari) also helps in the spread of the disease. The pathogen also survives on coconut, cocoa, mango, papaya, coffee, maize and arecanut. Insects also play a part in the dissemination of the pathogen.

Management

- Soak the setts in 0.05% Carbendazim 15 minutes.
- Use long setts having 3 or 4 buds.
- Provide adequate drainage during rainy seasons.
Wilt - *Cephalosporium sacchari*

**Symptoms**

The first symptom of the disease is visible in the canes of 4-5 month age. The canes may wither in groups. The affected plants are stunted with yellowing and withering of crown leaves. The midribs of all leaves in a crown generally turn yellow, while the leaf lamina may remain green. The leaves dry up and stem develop hollowness in the core. The core shows the reddish discolouration with longitudinal red streaks passing from one internode to another. In severe cases, spindle shaped cavities tapering towards the nodes develop in each internode. The canes emit a disagreeable odour, with lot of mycelial threads of the fungus cover the cavity.

**Pathogen**

The fungal mycelium is hyaline, septate and thin walled. The conidiophores are simple or branched and produce single celled, hyaline, oval to elliptical microconidia.

**Favourable Conditions**

- High day temperature (30-35°C).
- Low humidity (50-60%).
- Low soil moisture and alkaline soils.
- Excess doses of nitrogenous fertilizers.

**Disease Cycle**

The fungus is soil-borne and remains in the soil as saprophyte for 2-3 years. The disease is primarily transmitted through infected seed pieces. The secondary spread is aided by wind, rain and irrigation water.

**Management**

- Select the seed material from the disease-free plots.
- Avoid the practice of ratooning in diseased fields.
- Burn the trashes and stubbles in the field.
- Grow coriander or mustard as a companion crop in the early stages of crop.
- Dip the setts in 40ppm Boran or Manganese for 10 minutes or in 0.25% Emisan or 0.05% Carbendazim for 15 minutes.
Rust - *Puccinia erianthi* (Syn: *P. melanocephala* and *P. kuehnii*)

**Symptoms**

Minute, elongated, yellow spots (uredia), usually 2-10 x 1-3 mm appear on both the surfaces of young leaves. The pustules turn to brown on maturity. Late in the season, dark brown to black telia appear on the lower surface of leaves. In severe cases, the uredia also appear on the leaf sheath and the entire foliage looks brownish from a distance.

**Pathogen**

The mycelium is hyaline, branched and septate. *P.kuehnii* produces ovoid or pear shaped, single celled uredospores measuring 29-57 x 8-37µm with apical thickening and golden yellow in colour. Teliospores are produced in scanty which are yellow in colour, club shaped, two celled, smooth walled and measuring 24-34 X 18-25µm single celled, dark yellow coloured with 4 equatorial pores. Teliospores are produced in abundance, which are pale to brick colour, two celled, smooth walled and slightly septum. Occurrence of pycnial and aecial stages and the role of alternate host are unknown.

**Favourable Conditions**

- Temperature of 30°C.
- Humidity between 70 and 90 per cent.
- High wind velocity and continuous cloudiness.

**Disease Cycle**

The fungus survives on collateral hosts like *Erianthus fulvus* and *Saccharum spontaneum*. The uredospores also survive in the infected stubbles in the soil. The disease is mainly spread through air-borne uredospores.
Management

- Remove the collateral hosts.
- Spray Tridemorph 1 kg or Mancozeb 2 kg/ha.

Gummosis - *Xanthomonas axonopodis pv. vascularum*

Symptoms

The bacterium produces two distinct types of symptoms. On the mature leaves, longitudinal stripes or streaks, 3-7mm in width and several cm in length, appear around the affected veins, near the tip. Initially these stripes are pale yellow in colour, later turn to brown. The affected tissues slowly dry up.

The infected canes are stunted with short internodes, giving a bushy appearance. When such canes are cut transversely or split open longitudinally, a dull yellow bacterial ooze comes out from the cut ends and bacterial pockets are seen inside the slitted cane. The fibro vasicular bundles are deep red and internodal cavities formed in the severe cases are filled with yellow coloured bacterial gums.

Pathogen

The bacterium is a short rod, Gram negative, non spore forming measuring 1.0 to 1.5µm X 0.4 to 0.5µm, with a single polar flagellum. It is facultative anaerobe and it produces yellow slinky growth.

Disease Cycle

The bacterium remains viable in the soil as well as in infected canes. The primary transmission is through naturally affected diseased setts or through soil-borne contamination. The secondary spread may be through wind splashed rain, harvesting implements, animals and insects. The bacterium can survive in the insect's body for a long time and in this way may be transmitted long distances. On entry into the host the bacterium reaches the vascular tissues and becomes systemic. The bacterium also perpetuates on maize, sorghum, pearl millet and other weed hosts, which also serve as sources of inoculum.

Management

- Remove and burn the affected clumps and the stubbles in the field. Select setts from disease free areas.
Avoid growing collateral hosts like maize, sorghum and pearlmillet near the sugarcane fields.

**Red stripe - *Pseudomonas rubrilineans***

**Symptoms**

The disease first makes its appearance on the basal part of the young leaves. The stripes appear as water soaked, long, narrow chlorotic streaks and become reddish brown in a few days. These stripes are 0.5 to 1 mm in width and 5-100 mm in length, run parallel to the midrib. The stripes remain confined to lower half of the leaf lamina and whitish flakes spread to growing points of the shoot and yellowish stripes develop, which later turn reddish brown. The rotting may commence from the tip of the shoot and spreads downwards. The core is discoloured to reddish brown and shrivelled and form cavity in the centre. In badly affected fields, a foul and nauseating smell appears.

**Pathogen**

The bacterium is a short rod (0.7 X 1.67μm), gram negative, non capsulate with a polar flagellum.

**Favourable Conditions**

- Continuous ratooning and prolonged rainy weather with low temperature (25°C)

**Disease cycle**
The pathogen remains viable in the soil and infected plant residues. The bacterium also survives on sorghum, pearl millet, maize, finger millet and other species of *Saccharum*. The bacterium primarily spreads through infected canes. The secondary spread is mainly through rainsplash, irrigation water and insects. Infected parenchymatous cells may collapse and normal functioning of the plant parts may fail. Several grasses, including ragi and bajra, have been reported to be infected by the bacteria and these hosts may also play a role in the perpetuation and spread of the pathogen.

**Management**

- Whenever the disease is noticed; the affected plants should be removed and burnt.
- Growing resistant varieties Select setts from the healthy fields.
- Avoid growing collateral hosts near the sugarcane fields.

**Sugarcane Mosaic** - *Sugarcane mosaic potyvirus*

**Symptoms**

The disease appears more prominently on the basal portion of the younger foliage as chlorotic or yellowish stripes alternate with normal green portion of the leaf. As infection becomes severe, yellow stripes appear on the leaf sheath and stalks. Elongated necrotic lesions are produced on the stalks and stem splitting occurs. The necrotic lesions also develop on the internodes and the entire plant becomes stunted and chlorotic.

**Pathogen**

Sugarcane mosaic *potyvirus* is a flexous rod, 650-770nm long X 12-15nm with ss RNA genome.

**Disease cycle**
The virus is mainly transmitted through infected canes used as seed. The virus also infects *Zea mays* and a number of other cereals (*Sorghum vulgare, Pennisetum americanum, Eleusine indica, Setaria lutescens, Echinochloa crusgalli, Stenotaphrum secondatum, Digitaria didactyla*) which serve as potential sources of virus inoculum. The virus also spreads through viruliferous aphids viz., *Melanaphis sacchari, Rhopalosiphum maidis* in a non-persistant manner. The virus is also sap-transmissible. The incubation period varies from 7 to 20 days, depending upon the host variety and virus strain. The symptoms may be prominent or masked depending on the environmental conditions and variety.

**Management**

- Roguing of infected plants and use of disease free planting material.
- Chemical sprays to manage the insect vector population in early crop stage.
- Grow mosaic-resistant or, at least, tolerant varieties.
- Breeding mosaic-resistant varieties is needed.
- *Saccharum spontaneum* L. and *S. barberi* (Jesweit) carry resistance to mosaic and so varieties with this background must be preferred.
- Rogue out the diseased clumps periodically. Select setts from the healthy fields as the virus is sett-born. Aerated Steam Therapy (AST) at 56˚C for 3 hrs, for setts before planting is advised.

**Grassy shoot - Phytoplasma**

**Symptoms**

The disease appears nearly two months after planting. The disease is characterised by the production of numerous lanky tillers from the base of the affected shoots. Leaves become pale yellow to completely chlorotic, thin and narrow. The plants appear bushy and ‘grass-like’ due to reduction in the length of internodes premature and continuous tillering. The affected clumps are stunted with premature proliferation of auxillary buds. Cane formation rarely occurs in the affected clumps, if formed, thin with shorter internodes having aerial roots at the lower nodes. The buds on such canes usually papery and abnormally elongated.

**Pathogen**
The disease is caused by a **phytoplasma**. Two types of bodies are seen in ultrathin sections of phloem cells of infected plants. The spherical bodies of 300-400 nm diameter and **filamentous** bodies of 30-53 mm diameter in size.

**Disease cycle**

The primary spread of the phytoplasma is through diseased setts and cutting knifes. The pathogen is transmitted secondarily by aphids *viz.*, *Rhopalosiphum maydis*, *Melanaphis sacchari* and *M. idiosacchari*. Sorghum and maize serves as natural collateral hosts.

**Management**

- Eradication of diseased parts as soon as symptoms are seen.
- Avoid selection of setts from diseased area.
- Pre-treating the healthy setts with hot water at 52°C for 1 hour before planting
- Treating them with hot air at 54°C for 8 hours.
- Spraying the crop twice a month with insecticides.

**Ratoon stunting -** *Clavibacter xyli sub sp. xyli* (*Rickettsia Like Organism - RLO*)

**Symptoms**

Diseased clumps usually display stunted growth, reduced tillering, thin stalks with shortened internodes and yellowish foliage. Orange-red vascular bundles in shades of yellow at the nodes are seen in the infected canes.

**Pathogen**

The pathogen (*Clavibacter xyli sub sp. xyli*) is a RLO known to be present in the xylem cells of infected plants. They are small, thin, rod shaped or **coryneform** (0.15 to 0.32µm wide and 1.0-2.7µm long) and Gram positive.
Disease cycle

The primary spread is through the use of diseased setts. The disease also spreads through harvesting implements contaminated with the juice of the diseased canes. Maize, sorghum, Sudan grass and Cynodon serves as collateral hosts for the pathogen.

Management

- Select the setts from disease free fields or from disease free commercial nursery.
- Remove and burn the clumps showing the disease incidence.
- Treat the setts before planting, as specified for grassy shoot disease.

Minor diseases

Damping-off - *Pythium aphanidermatum, P. debaryanum, P. graminicola, P. ultimum*

Germinating seeds and young seedlings are attacked and killed in pre-emergence phase and seedlings show water soaked lesions at collar region, leading to withering and drying in post emergence stage.

Downy mildew - *Peronosclerospora sacchari*

Downy fungal growth with yellow stripes on upper surface, shredding of older leaves, rapid elongation of internodes of affected canes.

Eye spot - *Helminthosporium sacchari*

The water soaked spot develops on leaves, later elongated and turns to form “eye” shaped spot with reddish brown centre surrounded by straw yellow tissues.

Ring spot – *Leptosphaeria sacchari*

The water soaked spots appear on leaves and turns to straw colour later surrounded by a thin reddish brown band and a diffused discolouration zone.

Leaf scald - *Xanthomonas albilineans*
Whitish lines appear on the leaves, run to the full length of leaves and sheaths. Later leaves wither and dry from tip down-wards, gives a scald appearance to the clump. Sprouting of lateral buds of the matured canes occurs in acropetal fashion.

**White leaf - Phytoplasma**

Sugarcane white leaf is of minor importance and is caused by phytoplasma. The plants exhibit pure white leaves, stripped leaves and mottled leaves. Its vector is *Matsumuratettix hiroglyphicus.*
3. Diseases of Turmeric

Leaf Spot - *Colletotrichum capsici*

**Symptoms**

Oblong brown spots with grey centres are found on leaves. The spots are about 4-5 cm in length and 2-3 cm in width. In advanced stages of disease black dots representing fungal **acervuli** occur in concentric rings on spot. The grey centers become thin and gets teared. Severely effected leaves dry and wilt. They are surrounded by yellow halos. Indefinite number of spots may be found on a single leaf and as the disease advances; spots enlarge and cover a major portion of leaf blade.

**Favorable condition**

- The disease is usually appears in October and November
- Relative humidity of 80% and temperatures of 21 – 23°C favours the primary infection

**Disease cycle**

The fungus is carried on the scales of rhizomes which are the source of primary infection during sowing. The secondary spread is by wind, water and other physical and biological agents. The same pathogen is also reported to cause leaf-spot and fruit rot of chilli where it is transmitted through seed borne infections. If chilli is grown in nearby fields or used in crop rotation with turmeric, the pathogen perpetuates easily, building up inoculum potential for **epiphytotic** outbreaks.

**Management**
- Select seed material from disease free areas.
- Treat seed material with mancozeb @ 3g/litre of water or carbendazim @ 1 g/litre of water, for 30 minutes and shade dry before sowing.
- Spray mancozeb @ 2.5 g/litre of water or carbendazim @ 1g/litre; 2-3 sprays at fortnightly intervals.
- The infected and dried leaves should be collected and burnt in order to reduce the inoculum source in the field.
- Spraying Blitox or Blue copper at 3 g/l of water was found effective against leaf spot.
- Crop rotations should be followed whenever possible.
- Cultivate tolerant varieties like Suguna and Sudarshan.

Leaf Blotch *Taphrina maculans*

**Symptoms**

This disease usually appears on lower leaves in October and November. The individual spots are small 1-2 mm in width and are mostly rectangular in shape. The disease is characterized by the appearance of several spots on both the surfaces of leaves, being generally numerous on the upper surface. They are arranged in rows along the veins. The spots coalesce freely and form irregular lesions. They first appear as pale yellow discolorations and then become dirty yellow in colour. The infected leaves distort and have reddish brown appearance.

**Disease cycle**

The fungus is mainly air borne and primary infection occurs on lower leaves with the inoculum surviving in dried leaves of host, left over in the field. The ascospores discharged from
successively maturing **asci** infect fresh leaves without dormancy, thus causing secondary infection. Secondary infection is most dangerous than primary one causing profuse sprouting all over the leaves. The pathogen persists in summer by means of ascogenous cells on leaf debris, and dessicated ascospores and blastospores in soil and among fallen leaves.

**Management**

- Select seed material from disease free areas.
- Treat the seed material with Mancozeb @ 3g/litre of water or Carbendazim @ 1 g/litre of water for 30 minutes and shade dry before sowing.
- Spray mancozeb @ 2.5 g/litre of water or Carbendazim @ 1g/litre; 2-3 sprays at fortnightly intervals.
- The infected and dried leaves should be collected and burnt in order to reduce the inoculum source in the field.
- Spraying Cpper oxy chloride at 3 g/l of water was found effective against leaf blotch.
- Crop rotations should be followed whenever possible.

**Minor diseases**

- **a.** Dry rot - *Rhizoctonia bataticola*
- **b.** Leaf spot - *Cercospora curcuma*
- **c.** Leaf Blight - *Rhizoctonia solani*
- **d.** Brown rot - It is a complex disease caused by the nematode *Pratylenchus* sp. associated with *Fusarium* sp
4. Diseases of Sunflower

Leaf blight - *Alternaria helianthi*

**Symptoms**

The pathogen produces brown spots on the leaves, but the spots can also be seen on the stem, sepals and petals. The lesions on the leaves are dark brown with pale margin surrounded by a yellow halo. The spots later enlarge in size with concentric rings and become irregular in shape. Several spots coalesce to show bigger irregular lesions leading to drying and defoliation.
Pathogen

The pathogen produces cylindrical conidiophores, which are pale grey-yellow coloured, straight or curved, geniculate, simple or branched, septate and bear single conidium. Conidia are cylindrical to long ellipsoid, straight or slightly curved, pale grey-yellow to pale brown, 1 to 2 septate with longitudinal septa.

Favourable Conditions

- Rainy weather.
- Cool winter climate.
- Late sown crops are highly susceptible.

Disease cycle

The fungus survives in the infected host tissues and weed hosts. The fungus is also seed-borne. The secondary spread is mainly through wind blown conidia.

Management

- Deep summer ploughing.
- Proper spacing
- Clean cultivation and field sanitation.
- Use of resistant or tolerant variety like B.S.H.1.
- Application of well rotten manures.
- Practicing crop rotation.
- Planting in mid-September.
- Remove and destroy the diseased plants
- Treat the seeds with Thiram or Carbendazim at 2 g/kg. Spray Mancozeb at 2 kg/ha.
5. Diseases of Cotton

Wilt - *Fusarium oxysporum* f.sp. *vasinfectum*

Symptoms

The disease affects the crop at all stages. The earliest symptoms appear on the seedlings in the cotyledons which turn yellow and then brown. The base of petiole shows brown ring, followed by wilting and drying of the seedlings. In young and grown up plants, the first symptom is yellowing of edges of leaves and area around the veins i.e. discoloration starts from the margin and spreads towards the midrib. The leaves loose their turgidity, gradually turn brown, droop and finally drop off.

Symptoms start from the older leaves at the base, followed by younger ones towards the top, finally involving the branches and the whole plant. The defoliation or wilting may be complete leaving the stem alone standing in the field. Sometimes partial wilting occurs; where in only one portion of the plant is affected, the other remaining free. The taproot is usually stunted with less abundant laterals.
Browning or blackening of vascular tissues is the other important symptom, black streaks or stripes may be seen extending upwards to the branches and downwards to lateral roots. In severe cases, discolouration may extend throughout the plant starting from roots extending to stem, leaves and even bolls. In transverse section, discoloured ring is seen in the woody tissues of stem. The plants affected later in the season are stunted with fewer bolls which are very small and open before they mature.

Pathogen

Macroconidia are 1 to 5 septate, hyaline, thin walled, falcate with tapering ends. The microconidia are hyaline, thin walled, spherical or elliptical, single or two celled. Chlamydospores are dark coloured and thick walled. The fungus also produces a vivotoxin, Fusaric acid which is partially responsible for wilting of the plants.

Favourable Conditions

- Soil temperature of 20-30˚C
- Hot and dry periods followed by rains
- Heavy black soils with an alkaline reaction
- Increased doses of nitrogen and phosphatic fertilizers
- Wounds caused by nematode (*Meloidogyne incognita*) and grubs of Ash weevil (*Myllocerus pustulatus*).

Disease cycle

The fungus can survive in soil as saprophyte for many years and chlamydospores act as resting spores. The pathogen is both externally and internally seed-borne. The primary infection is mainly from dormant hyphae and chlamydospores in the soil. The secondary spread is through conidia and chlamydospores which are disseminated by wind and irrigation water.
Management

- Treat the acid delinted seeds with Carboxin or Carbendazim at 2 g/kg.
- Remove and burn the infected plant debris in the soil after deep summer ploughing during June-July.
- Apply increased doses of potash with a balanced dose of nitrogenous and phosphatic fertilizers.
- Apply heavy doses of farm yard manure or other organic manures. Follow mixed cropping with non-host plants.
- Grow disease resistant varieties of *G. hirsutum* and *G. barbadense* like Varalakshmi, Vijay Pratap, Jayadhar and Verum.
- Spot drench with Carbendazim 1g/litre.

Verticillium wilt - *Verticillium dahliae*

Symptoms

The symptoms are seen when the crop is in squares and bolls. Plants infected at early stages are severely stunted. The first symptoms can be seen as bronzing of veins. It is followed by interveinal chlorosis and yellowing of leaves. Finally the leaves begin to dry, giving a scorched appearance. At this stage, the characteristic diagnostic feature is the drying of the leaf margins and areas between veins, which gives a “Tiger stripe” or “Tiger claw” appearance.

The affected leaves fall off leaving the branches barren. Infected stem and roots, when split open, show a pinkish discolouration of the woody tissue which may taper off into longitudinal streaks in the upper parts and branches. The infected leaf also shows brown spots at the end of the petioles. The affected plants may bear a few smaller bolls with immature lint.
Pathogen

The fungus produces hyaline, septate mycelium and two types of spores. The conidia are single celled, hyaline, spherical to oval, borne singly on verticillate condioophores. The micro sclerotia are globose to oblong, measuring 48-120 X 26-45um.

Favourable Conditions

- Low temperature of 15-20°C,
- Low lying and ill-drained soils,
- Heavy soils with alkaline reaction
- Heavy doses of nitrogenous fertilizers.

Disease Cycle

The fungus also infects the other hosts like brinjal, chilli, tobacco and bhendi. The fungus can survive in the infected plant debris and in soils as micro sclerotia upto 14 years. The seeds also carry the micro sclerotia and conidia in the fuzz. The primary spread is through the
micro sclerotia or conidia in the soil. The secondary spread is through the contact of diseased roots to healthy ones and through dissemination of infected plant parts through irrigation water and other implements.

**Management**

- Treat the delinted seeds with **Carboxin** or **Carbendazim** at 2 g/kg.
- Remove and destroy the infected plant debris after deep ploughing in summer months (June-July).
- Apply heavy doses of farmy and manure or compost at 100t/ha.
- Follow crop rotation by growing paddy or lucerne or chrysanthemum for 2-3 years.
- Spot drench with 0.05g/l benomyl or carbendazim 500mg/l.
- Grow disease resistant varieties like Sujatha, Suvin and CBS 156 and tolerant variety like MCU 5 WT.
- Apply farm yard manure at 10t/ha or neem cake at 150 Kg/ha.
- Adjust the sowing time, early sowing (First Week of April) or late sowing (Last week of June) so that crop escapes the high soil temperature conditions.
- Adopt intercropping with sorghum or moth bean (*Phaseolus aconitifolius*) to lower the soil temperature.

**Anthracnose - *Colletotrichum capsici***

**Symptoms**

The pathogen infects the seedlings and produces small reddish circular spots on the cotyledons and primary leaves. The lesions develop on the collar region, stem may be girdled, causing seedling to wilt and die. In mature plants, the fungus attacks the stem, leading to stem splitting and shredding of bark. The most common symptom is boll spotting. Small water soaked, circular, reddish brown depressed spots appear on the bolls. The lint is stained to yellow or brown, becomes a solid brittle mass of fibre. The infected bolls cease to grow and burst and dry up prematurely.
Pathogen

The pathogen forms large number of acervuli on the infected parts. The conidiophores are slightly curved, short, and club shaped. The conidia are hyaline and falcate, borne single on the conidiophores. Numerous black coloured and thick walled setae are also produced in acervulus.

Favourable Conditions

- Prolonged rainfall at the time of boll formation
- Close planting.
Disease Cycle

The pathogen survives as dormant mycelium in the seed or as conidia on the surface of seeds for about a year. The pathogen also perpetuates on the rotten bolls and other plant debris in the soil. The secondary spread is by air-borne conidia. The pathogen also survives in the weed hosts viz., *Aristolachia bractiata* and *Hibiscus diversifolius*.

Management

- Treat the delinted seeds with Carbendazim or Carboxin or Thiram or Captan at 2g/kg.
- Remove and burn the infected plant debris and bolls in the soil.
- Rogue out the weed hosts.
- Spray the crop at boll formation stage with Mancozeb 2kg or Copper oxychloride 2.5 kg or or Carbendazim 500g/ha.

Bacterial blight - *Xanthomonas axonopodis pv. malvacearum*

Symptoms

The bacterium attacks all stages from seed to harvest. Usually five common phases of symptoms are noticed.

i) Seedling blight:

Small, water-soaked, circular or irregular lesions develop on the cotyledons, later, the infection spreads to stem through petiole and cause withering and death of seedlings.

ii) Angular leaf spot:

Small, dark green, water soaked areas develop on lower surface of leaves, enlarge gradually and become angular when restricted by veins and veinlets and spots are visible on both the surface of leaves. As the lesions become older, they turn to reddish brown colour and infection spreads to veins and veinlets.

iii) Vein blight or vein necrosis or black vein:

The infection of veins cause blackening of the veins and veinlets, gives a typical ‘blighting’ appearance. On the lower surface of the leaf, bacterial oozes are formed as crusts or scales. The affected leaves become crinkled and twisted inward and show withering. The infection also spreads from veins to petiole and cause blighting leading to defoliation.

iv) Black arm:

On the stem and fruiting branches, dark brown to black lesions are formed, which may girdle the stem and branches to cause premature drooping off of the leaves, cracking of stem and
gummosis, resulting in breaking of the stem and hang typically as dry black twig to give a characteristic “black arm” symptom.

v) Square rot / Boll rot:

On the bolls, water soaked lesions appear and turn into dark black and sunken irregular spots. The infection slowly spreads to entire boll and shedding occurs. The infection on mature bolls lead to premature bursting. The bacterium spreads inside the boll and lint gets stained yellow because of bacterial ooze and looses its appearance and market value. The pathogen also infects the seed and causes reduction in size and viability of the seeds.

Angular leaf spot

Bacterial blight lesions on leaf and the blackleg symptom on the leaf petiole
Pathogen

The bacterium is a short rod with a single polar flagellum. It is Gram negative, non-sporforming and measures 1.0-1.2 X 0.7-0.9 µm.

Favorable Conditions

- Optimum soil temperature of 28°C,
- High atmospheric temperature of 30-40°C,
- Relative humidity of 85 per cent, early sowing,
- Delayed thinning,
- Poor tillage, late irrigation and
- Potassium deficiency in soil.
- Rain followed by bright sunshine during the months of October and November are highly favorable.

Disease Cycle

The bacterium survives on infected, dried plant debris in soil for several years. The bacterium is also seed-borne and remains in the form of slimy mass on the fuzz of seed coat. The bacterium also attacks other hosts like *Thumbergia thespesioides*, *Eriodendron anfructuosum* and *Jatropha curcus*. The primary infection starts mainly from the seed-borne bacterium. The secondary spread of the bacteria may be through wind, wind blown rain splash, irrigation water, insects and other implements.

Management

- Delint the cotton seeds with concentrated sulphuric acid at 100ml/kg of seed. Treat the delinted seeds with carboxin or oxycarboxin at 2 g/kg or soak the seeds in 1000 ppm *Streptomycin sulphate* overnight.
- Remove and destroy the infected plant debris. Rogue out the volunteer cotton plants and weed hosts.
- Follow crop rotation with non-host crops.
- Early thinning and early earthing up with potash.
- Grow resistant varieties like Sujatha, 1412 and CRH 71.
- Spray with Streptomycin sulphate + Tetracycline mixture 100g along with Copper oxychloride at 1.25 Kg/ha.

Leaf Curl Disease- *Cotton leaf curl virus*

**Symptoms**

Downward and upward curling of leaves and thickening of veins and enation on underside of leaves are the characteristic symptoms of the disease. In severe infection all the leaves are curled and growth retarded. Boll bearing capacity is reduced.
Pathogen

It is caused by *Cotton leaf curl virus* - a *begomovirus* of family geminiviridae. The virions are typical *geminate* particles, *ss circular DNA, bipartite genome* with DNA-A and DNA-B components.

Disease Cycle

The primary source is the *viruliferous* whitefly vector *Bemisia tabaci*. The alternate hosts and cultivated hosts serve as virus reservoirs throughout the year. Not transmitted by seed or contact.

Management

- Management of planting date to avoid peak vector population.
- Elimination of volunteer perennial cotton and alternate hosts including malvaceous hosts like wild okra
- Use of fungus *Paecilomyces farinosus* which parasitizes *B.tabaci*. It brings down vector population.
- Foliar application of neem leaf extract and 1% neem oil resulted in 80% reduction of virus transmission.
- Vector management by application of granular *systemic insecticides.*
6. Diseases of Bengal gram

Ascochyta blight - *Ascochyta rabiei*

**Symptoms**

All above ground parts of the plant are infected. On leaf, the lesions are round or elongated, bearing irregularly depressed brown spot and surrounded by a brownish red margin. Similar spots may appear on the stem and pods. The spots on the stem and pods have pycnidia arranged in concentric circles as minute block dots. When the lesions girdle the stem, the portion above the point of attack rapidly dies. If the main stem is girdles at the collar region, the whole plant dies.
Pathogen

The fungus produces hyaline to brown and septate mycelium. Pycnidia are spherical to sub-globose with a prominent ostiole. Pycnidiospores are hyaline, oval to oblong, straight or slightly curved and single celled, occasionally bicelled.

Favourable conditions

- High rainfall during flowering.
- Temperature of 20-25˚C.
- Relative humidity of 60%.

Disease cycle

The fungus survives in the infected plant debris as pycnidia. The pathogen is also externally and internally seed-borne. The primary spread is from seed-borne pycnidia and plant debris in the soil. The secondary spreads is mainly through air-borne pycnidiospores (conidia). Rain splash also helps in the spread of the disease.

Management

- Remove and destroy the infected plant debris in the field.
- Treat the seeds with Thiram 2g or Carbendazim 2 g or Thiram + Carbendazim (1:1 ratio) at 2 g/kg.
- Exposure of seed at 40-50˚C reduced the survival of A. rabiei by about 40-70 per cent.
- Spray with Carbendazim at 500 g/ha or Chlorothalonil 1kg/ha.
- Follow crop rotation with cereals.

Rust - Uromyces ciceris-arietini

Symptoms

The infection appears as small oval, brown, powdery lesions on both the surface, especially more on lower surface or leaf. The lesions, which are uredosori, cover the entire leaf surface. Late in the season dark teliosori appear on the leaves. The rust pustules may appear on petioles, stems and pods. The pycnial and aecial stages are unknown.

Pathogen

The uredospores are spherical, brownish yellow in colour, loosey echinulated with 4-8 germ pores. Teliospores are round to oval, brown, single celled with unthickened apex and the walls are rough, brown and warty.
Mode of Spread and Survival

The fungus survives as uredospores in the legume weed *Trigonella polycerata* during summer months and serve as primary source of infection. The spread is through wind-borne uredospores.

Management

- Destory weed host.
- Spray Carbendazim 500 g/ha or Propiconazole 1L/ha.

Wilt - *Fusarium oxysporum f.sp. ciceris*

Symptoms

The disease occurs at two stages of crop growth, seedling stage and flowering stage. The main symptoms on seedlings are yellowing and drying of leaves, drooping of petioles and rachis, withering of plants. In the case of adult plants drooping of leaves is observed initially in upper part of plant, and soon observed in entire plant. Vascular browning is conspicuously seen on the stem and root portion.

Pathogen

The fungus produces hyaline to light brown, septate and profusely branched hyphae. Microconidia are oval to cylindrical, hyaline, single celled, normally arise on short conidiophores. Macroconidia which borne on branched conidiophores, are thin walled, 3 to 5 septate, fusoid and pointed at both ends. Chlamydospores are roughwalled or smooth, terminal or intercalary, may be formed singly or in chains.
Favourable conditions

- High soil temperature (above 25°C).
- High soil moisture.

Disease cycle

The disease is seed and soil borne. The primary infection is through chlamydospores in soil, which remain viable up to next crop season. The secondary spread is through irrigation water, cultural operations and implements.

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or Carbendazim 1 g + Thiram 1 g/kg or treat the seeds with *Trichoderma viride* at 4 g/kg (10^6 cfu/g) *Pseudomonas fluorescens* @ 10 g/kg (10^6 cfu/g) of seed.
- Apply heavy doses of organic manure or green manure.