DERE MARKOS UNIVERSITY

COLLEGE OF AGRICULTUR E AND NATURAL RESOURCE

DEPARTMENT OF PLANT SCIENCE

Management of crop diseases and insect pests of economic importance

Course code (Plsc 2054)

Debre markos, Ethiopia

CHAPTER ONE

1. INTRODUCTION

Principles of pest management

Pest definitions, kinds and outbreak

PEST - Derived from French word 'Peste' and Latin term 'Pestis' meaning plague or contagious disease.

- Pest is any animal which is noxious, destructive or troublesome to man or his interests.
- A pest is any organism which occurs in large numbers and conflict with man's welfare, convenience and profit.
- A pest is an organism which harms man or his property significantly or is likely to do so

Pests are sufficiently numerous to cause economic damage

Pest is any organism detrimental to man his property or any form of plant or animal life or any pathogenic agent potentially injurious to plants, plant products, livestock or man.

- Pests are organisms which impose burdens on human population by causing:

(i) Injury to crop plants, forests and ornamentals

(ii) Annoyance, injury and death to humans and domesticated animals

(iii) Destruction or value depreciation of stored products.

Pests include insects, nematodes, Fungus, weeds and vertebrates like rats, birds, etc.

KINDS OF PESTS

Based on occurrence following are pest categories

Regular pest: Frequently occurs on crop - Close association e.g. Rice slem borer, Brinjal fruit borer

Occasional pest: Infrequently occurs, no close association e.g. Caseworm on rice,Mango stem borer

Seasonal pest: Occurs during a particular season every year e.g. Red hairy caterpillar on groundnut, Mango hoppers

Persistent pests: Occurs on the crop throughout the year and is difficult to control e.g. Chilli thrips, mealy bug on guava

Sporadic pests: Pest occurs in isolated localities during some period. e.g. Coconut slug caterpillar

Based on level of infestation

Pest epidemic: Sudden outbreak of a pest in a severe form in a region at a particular time e.g**.** BPH in Tanjore, RHC in Madurai, Pollachi

Endemic pest: Occurrence of the pest in a low level in few pockets, regularly and confined to particular area e.g. Rice gall midge in Madurai, Mango hoppers in Periyakulam

Parameters of insect population levels

General equilibrium position (GEP)

The average density of a population over a long period of time, around which the pest population over a long period of time, around which the pest population tends to fluctuate due to biotic and a biotic factors and in the absence of permanent environmental changes.

Economic threshold level (ETL)

Population density at which control measure should be implemented to prevent an increasing pest population from reaching the ETL.

Economic injury level (EIL)

The lowest population density that will cause economic damage

Damage boundary (DB)

The lowest level of damage which can be measured. ETL is always less than EIL. Provides sufficient time for control measures.

PEST CATEGORIES ACCORDING TO EIL, GEP AND DB

(A) Key pest

- Most severe and damaging pests
- GEP lies above EIL always
- Spray temporarily bring population below EIL
- These are persistent pests
- The environment must be changed to bring GEP below EIL, e.g. Cotton bollworm, Diamond back moth

(B) Major pest

- GEP lies very close to EIL or coincides with EIL
- Economic damage can be prevented by timely and repeated sprays e.g. Cotton jassid, Rice stem borer

(C) Minor pest/Occasional pest

- GEP is below the EIL usually
- Rarely they cross EIL
- Can be controlled by spraying e.g. Cotton stainers, Rice hispa, Ash weevils

(D) Sporadic pests

- GEP generally below EIL
- Sometimes it crosses EIL and cause severe loss in some places/periods e.g. Sugarcane pyrilla, White grub, Hairy caterpillar

(E) Potential pests

- They are not pests at present
- GEP always less than EIL
- If environment changed may cause economic loss e.g. *S. litura* is potentia pest in North India

CAUSES OF PEST OUTBREAK

Activity of human beings which upsets the biotic balance of ecosystem is the primary cause for pest outbreak. The following are some human interventions – Reason for outbreak

I. Deforestation bringing under cultivation

- Pest feeding on forest trees are forced to feed on cropped
- Biomass/unit area more in forests than agricultural land
- Weather factors also altered - Affects insect development

II. Destruction of natural enemies

- Due to excess use of insecticides, natural enemies are killed
- This affects the natural control mechanism and pest outbreak occurs, e.g. Synthetic pyrethroid insecticides kill NE.

III. Intensive and Extensive cultivation

- Monoculture (Intensive) leads to multiplication of pests
- Extensive cultivation of susceptible variety in large area - No competition for food
- multiplication increases e.g. Stem borers in rice and sugarcane

IV. Introduction of new varieties and crops.

- Varieties with favourable physiological and morphological factors cause
- multiplication of insects. e.g. Succulent, dwarf rice varieties favour leaf folder

- Combodia cotton favours stem weevil and spotted bollworm
- Hybrid sorghum (CSH 1), cumbu (HB1) favour shoot flies and gall midges

V. Improved agronomic practices

- Increased N fertilizer - High leaf folder incidence on rice
- Closer planting - BPH and leaf folder increases
- Granular insecticides - Possess phytotonic effect on rice

VI. Introduction of new pest in new environment

- Pest multiplies due to absence of natural enemies in new area
- Apple wooly aphid *Eriosoma lanigerum* multiplied fast due to absence of
- *Aphelinus mali* (Parasit)

VII. Accidental introduction of pests from foreign countries (through air/sea ports) e.g.

- Diamondback moth on cauliflower (*Plutella xylostella*)
- Potato tuber moth *Phthorimaea operculella*
- Cottony cushion scale *Icerya purchasi* on wattle tree
- Wooly aphid - *Eriosoma lanigerum* on apple
- Psyllid - *Heteropsylla cubana* on subabul
- Spiralling whitefly - *Adeyrodichus dispersus* on most of horticultural crops

viii. Large scale storage of food grains Serve

- as reservoir for stored grain pests
- Urbanisation - changes ecological balance
- Rats found in underground drainage

Losses caused by pests

- Crop loss from all factors - 500 billion US $ annually world wide
- Insect pests - 15.6% loss of production
- Plant pathogens - 13.3%
- Weeds - 13.2%

Most organisms are not pests. A species may be a pest in some situations and not in others. An organism should not be considered a pest until it is proven to be one.

Pest Identication

Accurate identification is the first step in an effective pest management program.

Correct identification of a pest allows you to determine basic information about it, including its life cycle and the time that it is most susceptible to being controlled.

To be able to identify and control pests, you need to know:

The physical features of the pests likely to be encountered. Characteristics of the damage they cause, their development and biology, whether they are continuous, sporadic or potential pests.

Pest Control

Use a control strategy that will reduce the pest numbers to an acceptable level.

Cause as little harm as possible to everything except the pest.

Even though a pest is present, it may not do very much harm. It could cost more to control the pest than would be lost because of the pest's damage.

Pest Control Goals

Whenever you try to control a pest you will want to achieve one of these three goals. or some combination of them:

Prevention - keeping a pest from becoming a problem.

Suppression - reducing pest numbers or damage to an acceptable level, and .

Eradication - destroying an entire pest population.

Pest Monitoring

Monitoring phytophagous insects and their natural enemies is a fundamental tool in IPM - for taking management decision

Monitoring - estimation of changes in insect distribution and abundance

- Information about insects, life history
- Influence of biotic and abiotic factors on pest population

Pest Surveillance

Refers to the constant watch on the population dynamics of pests, its incidence and damage on each crop at fixed intervals to forewarn the farmers to take up timely crop protection measures.

Three basic components of pest surveillance

Determination of

A. the level of incidence of the pest species

B. the loss caused by the incidence

C. the economic benefits, the control will provide

Pest Forecasting

Forecasting of pest incidence or outbreak based on information obtained from pest surveillance.

Uses

- Predicting pest outbreak which needs control measure
- Suitable stage at which control measure gives maximum protection

Two types of pest forecasting

a. Short term forecasting - Based on 1 or 2 seasons

b. Long term forecasting - Based on affect of weather parameters on pest

Objectives of Pest Surveillance

To know existing and new pest species

To assess pest population and damage at different growth stage of crop

To study the influence of weather parameters on pest

To study changing pest status (Minor to major)

To assess natural enemies and their influence on pests

To know effect of new cropping pattern and varieties on pest

Survey

Conducted to study the abundance of a pest species

Two types of survey - Roving survey and fixed plot survey

Roving survey

- Assessment of pest population/damage from randomly selected spots representing larger area
- Large area surveyed in short period
- Provides information on pest level over large area

Fixed plot survey

Assessment of pest population or damage from a fixed plot selected in a field.

The data on pest population/damage recorded periodic from sowing till harvest.

Qualitative survey - Useful for detection of pest

Quantitative survey - Useful for enumeration of pest

Sampling Techniques

Absolute sampling - To count all the pests occurring in a plot

Relative sampling - To measure pest in terms of some values which can be compared over time and space e.g. Light trap catch, Pheromone trap

Methods of sampling

a. *In situ* counts - Visual observation on number of insects on plant canopy (either entire plot or randomly selected plot)

b. Knock down - Collecting insects from an area by removing from crop and (Sudden trap) counting (Jarring)

c. Netting - Use of sweep net for hoppers, odonates, grasshopper

d. Norcotised collection - Quick moving insects anaesthetized and counter

e. Trapping - Light trap - Phototropic insects

Pheromone trap - Species specific

Sticky trap - Sucking insects

Bait trap - Sorghum shootfly - Fishmeal trap

Emergence trap - For soil insects

Chapter 2
Management methods of diseases and Insect Pests

I. Cultural method or use of agronomic practices

1. Crop rotation
2. Crop refuses destruction
3. Tillage of soil
4. Variation in time of Planting or harvesting
5. Pruning or thinning
6. Fertilizer management
7. Water management
8. Intercropping
9. Trap crop

II. Host plant resistance - Antixenosis, antibiosis, tolerance

III. Mechanical methods of pest control

1. Hand destruction

2. Exclusion by screens, barriers

3. Trapping, suction devices, collecting machine

4. Crushing and grinding

IV. Physical methods

Heat, cold, energy - light trap, irradiation, light regulation,sound

V. Biological methods

1. Introduction, artificial increase and colonizing specific parasitoids and predators

2. Pathogens on insects like virus, bacteria, fungi and protozoa

3. Use of botanicals like neem, pongam, etc.

VI. Chemical methods

Attractants, Repellents, Insecticides -Carbamates, pyrethroids, etc., insect growth inhibitors, Chemosterilants

VII. Behavioral methods.

Pheromones, Allelochemicals

VIII. Genetic/biotechnology method

- Release of genetically incompatible/sterile pests, transgenic plant

IX. Regulatory/legal method

- Plant/animal quarantine, eradication and suppression program me

Integrated Pest Management

Integrated Pest Management (IPM) is a system that, in the context of associated environment and population dynamics of the pest species, utilizes all suitable techniques and methods in as compatible a manner as possible and maintains pest populations at levels below those causing economic injury.

IPMI is also defined as the intelligent selection and use of pest control tactics that will ensure favorable economical, ecological and sociological consequences.

CHAPTER THREE
MAJOR SEEDLING DISEASE and INSECT PESTS

Major Seedling Disease

Damping off (*Pythium spp.*)

- ✓ Seedlings occur worldwide in valleys and forest soils, in tropical and temperate climates, and in every greenhouse.
- ✓ Rotting and collapse of seedlings at soil level or prevention of seedling emergence (e.g. damping-off of vegetables and tobacco)
- ✓ It is destruction of seedlings near the soil line, resulting in the seedlings falling over on the ground

In all cases, however, the greatest damage is done to the seed and seedling roots during germination either before or after emergence.

- ✓ Losses vary considerably with soil moisture, temperature, and other factors.
- ✓ Quite frequently, seedlings in seedbeds are completely destroyed by damping-off or they die soon after they are transplanted.
- ✓ In many instances, poor germination of seeds or poor emergence of seedlings is the result of damping-off infections in the pre-emergence stage.
- ✓ Older plants are seldom killed when infected with the damping-off pathogen, but they develop root and stem lesions and root rots, their growth may be retarded considerably, and their yields may be reduced drastically

- ✓ Some species of the damping-off oomycete also attack the fleshy organs of plants, which rot in the field or in storage.
- ✓ *Pythium debaryanum, P. ultimum,* and *P. aphanidermatum* cause damping-off of vegetables and fruit trees.
- ✓ *Pythium arrhenomanes, P. graminicola,* and *P. tardicrescens* cause root rot in cereals.

Symptoms of Damping off

- When seeds of susceptible plants are planted in infested soils and are attacked by the damping-off fungi, they fail to germinate, become soft and mushy, and then turn brown, shrivel, and finally disintegrate
- Young seedlings can be attacked before emergence at any point on the plant, from which the infection spreads rapidly, the invaded cells collapse, and the seedling is overrun by the oomycete and dies (pre-emergence damping-off).

- Seedlings that have already emerged are usually attacked at the roots and sometimes in the stems at or below the soil line.
- The invaded areas become water soaked and discolored and they soon collapse
- The basal part of the seedling stem becomes softer and much thinner than the un invaded parts above it; as a result, the seedling falls over on the soil.
- The fungus continues to invade the fallen seedling, which quickly withers and dies (post-emergence damping-off).
- In cereals and turf grasses, the pathogen causes "*Pythium* blight," i.e., it invades and kills the roots and whole seedlings and even young plants, causing the appearance of numerous empty patches on the lawn or field
- Several species of *Pythium* cause pre- and post-emergence damping-off.
- Certain other oomycetes and fungi, however, such as *Phytophthora*, *Rhizoctonia*, and *Fusarium*, often cause symptoms quite similar to those described earlier.
- Several more fungi, and even some bacteria, when carried in or on the seed, also cause damping-off and kill seedlings.

Pythium produces a white, rapidly growing mycelium

- The mycelium gives rise to sporangia, which germinate directly by producing one to several germ tubes or by producing a short hypha at the end of which forms a balloon-like secondary sporangium called a vesicle
- In the vesicle, 100 or more zoospores are produced, which, when released, swarm about for a few minutes, round off to form a cyst, and then germinate by producing a germ tube.
- The germ tube usually penetrates the host tissue and starts a new infection, but sometimes it produces another vesicle in which several secondary zoospores are formed, and this may be repeated.

Disease cycle

- The fungus survives in the soil as oospores and chlamydospores.
- The primary infection is from the soil-borne **oospores** and secondary spread through **sporangia** and **zoospores** transmitted by wind and irrigation water.

Favorable Conditions

- Overcrowding of seedlings, ill drained nursery beds, heavy shade in nursery, high atmospheric humidity (90-100 %), high soil moisture, low temperature (below 24^0C) and low soil temperature of about 20^0C.
- **Management:** *Pythium* diseases in the greenhouse can be controlled through the use of soil sterilized or pasteurized by steam or dry heat and through the use of chemically treated seed.
- Greenhouse benches and containers must also be sterilized or treated with an appropriate chemical solution.
- Raised seed beds of 15-45 cm height should be formed.
- Avoid overcrowding of seedlings by using optimum seed rate of **3-3.5 kg/ha** (1 to 1.5g/2.5m2)
- Provide adequate drainage facility and avoid excess watering of the seedlings.
- Burn the seed beds with paddy husk or dry twigs before sowing.
- Drench the seed bed with 0.4% per cent Bordeaux mixture or 0.2 per cent Copper oxychloride, two days before sowing.
- Spray the nursery beds twice with 0.4% Bordeaux mixture or 0.2 Copper oxychloride or Metalaxyl or Mancozeb at 20 and 30 days after germination.
- Soil incorporation of *Trichoderma viride* or *T. harzianum* in seed beds one week before seed sowing and thereafter Bordeaux mixture should be sprayed at 0.4 per cent.

2. **Root rots**

- Caused by *Rhizoctonia spp*.

Symptoms ;

- Root rot is wide spread and destructive.
- The fungus causes different types of symptoms, *viz.*, seedling disease and root rot.
- Germinating seedlings of one to two weeks old are attacked by the fungus at the **hypocotyl** and cause black lesions, girdling of stem and death of the seedling, causing large gaps in the field.
- In sore-shin stage (4 to 6 weeks old plants), **dark reddish-brown cankers** are formed on the stems near the soil surface which later turns dark brown or black and plant breaks at the collar region leading to drying of the leaves and subsequently the entire plant.
- **Pathogen**

- ✓ The fungal hyphae are septate and fairly thick and produce black, irregular **sclerotia** which measure 100 µm in diameter.

- **Disease cycle**

- ✓ The disease is mainly soil-borne and the pathogen can survive in the soil as **sclerotia** for several years.
- ✓ The spread is through sclerotia which are disseminated by **irrigation water**, implements, **heavy winds** and other **cultural operations**.

❖ **Favorable Conditions**

- Dry weather following heavy rains, high soil temperature (**35-39^{o}C**), low soil moisture (**15-20%**), cultivation of favorable hosts like vegetables, oil seeds and legumes preceding cotton and wounds caused by ash-weevil grubs and nematodes.

❖ **Management**

- Treat the seeds with *Trichoderma viride* @ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Treat the seeds with Carboxin or Thiram at 4 g or Carbendazim at 2g/kg.
- Spot drench with 0.1% Carbendazim or 0.05% Benomyl.
- Apply farm yard manure at 100 t/ha or neem cake at 2.5t/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.
- Grow resistant varieties

CHAPTER FOUR

MAJOR DISEASES AND INSECTPESTS OF FIELD CROPS

4.1. WHEAT DISEASES

4.1.1. Wheat Stem rust (*Pucciniagraminis*)

Significance and hosts

The early Romans recognized stem rust and called it the ‘greatest of plant diseases’. Detailed characterization of its disease cycle began in 1767. Stem rust (Black rust, black stem rust) is caused by *Pucciniagraminis*. That also parasitizes certain barley, rye, and oat cultivars and some grasses, especially wild barley and goat-grass. Attacks on non–wheat hosts normally are weak

and marginally significant, but some grasses may be sources of inoculums. Alternate hosts include barberry plant (*Berberis vulgaris*) that is an erect woody shrub that reaches 3 m in height.

Favourable condition for epidemics

Stem rust develops optimally near 20^{o}C and is seriously hampered below 15^{o}C and above 40^{o}C. Delayed crop maturity especially favours the disease.

Control measures

Wheat rusts are best controlled by resistant cultivars. In the last 90 years the heritability of rust resistance has been utilized by breeders to systemically develop resistant wheat. Destroying alternate hosts interrupts the life cycle of rust fungi, limits their diversity and indirectly increases the stability of resistant cultivars. It also prevents the production of early spring inoculums or aeciospores. Low cost, protectant or eradicant fungicides are sometimes used for rust control.

They are applied as foliar sprays where cost–benefit analyses show them profitable. Sulfur has been effective, non–specific protectant chemical, but its present commercial value is uneconomical. Avoiding monocultures of a given cultivar over vast areas restricts rust damage since heterogeneity in the rust population for epidemics to occur. Heterogeneity can be accomplished on a field basis by planting several different cultivars and on a plant basis by planting mixtures of seed differing in genes for resistance (multilines). Phosphorus application can reduce infection.

4.1.2. Leaf rust (*Puccinia recondite*)

Significance and distribution

Leaf rust (syns. brown rust, dwarf rust, orange rust) may be the most widely distributed of wheat diseases. Practically coexistent with the crop, it is most prevalent where wheat matures late as in spring wheat regions. Leaf rust is a serious disease of wheat, rye, triticale and many grasses. The pathogen is mildly parasitic on barley, but does not infect oats. The causal fungus, *Pucciniarecondite* is a weak parasite on certain cultivars of barley and some *Aegilops* (goat – grass).

Causal pathogen

The causal pathogen is *Puccinia recondite*. The uredospores of *P. recondita* are orange red to dark red, oblong, spherical, stalked and have 1-10 germ pores and usually measure 20 –28 um in diameter. Uredospores penetrate through closed stomata. Teliospores are dark brown, two–celled

(bicellular) with thick walls, always covered by the epidermis, and are rounded or flattened at the apex. The telia develop during the later stages of plant development in sori on leaf sheaths and on both surfaces of the leaf blades. Teliospores remain in the leaf tissues and covered by the epidermis. In the tropics, it over–summer at higher elevations and in cooler areas.

Disease Cycle

The wind blownuredorespores, which survive on volunteer crops in hills, serve as the primary inoculums. The uredospores spread the disease and end up with the production of teliospores. Both spores fail to remain their ability in the hot intervening summer months.

Control measures (same as stem rust)

Leaf rust is best controlled by the use of resistant cereal crop varieties or multiline. Fungicides, such as Triadimefon (Bayleton) and Butrizol (Indar), are effective in controlling leaf rust and can be used economically in epidemic situations.

4.1.3. Yellow stripe rust (*Pucciniastriiformis*)

Significance and host ranges

Stripe rust (synons. Yellow rust, glume rust) is confined to higher elevations and cooler climates and does not persist in many regions where leaf and stem rusts occur. Stripe rust caused by *Pucciniastriiformis* is not known to have alternate hosts or sexual stages. It has a wider host range than stem or leaf rust fungi infecting rye and over 18 genera of grasses. Barley, in addition to wheat, is damaged economically and many perennial grasses are important reservoirs for the fungus.

Disease Cycle

Stripe rust of wheat originates from uredospores that survive locally or are wind–borne from distant hosts. Wheat at lower elevations sometimes is infected by uredospores from grasses at higher altitudes. Infections may occur throughout autumn and winter since mycelium remains viable to -5 ^{o}C.Urediospores lose viability rapidly at temperatures above 15 ^{o}C. They germinate optimally between 5 and 15 ^{o}C with limits near 0 and 21^{o}C. Disease development is most rapid between 10 and 15 ^{o}C with intermittent rain or dew. Disease development occurs at lower temperatures than are optimal for leaf and stem rusts.

Control measures (same as stem rust)

Symptoms of the three important Rusts

No	*Characters*	*Leaf rust*	*Stem rust*	*Stripe rust*
1	*Color*	*Orange brown*	*Dark reddish brown*	*Orange yellow*
2	*Shape*	*Pustules are circular or slightly elliptical*	*Elliptical*	*Spherical*
3	*Size of pustules*	*Least*	*Bigger one*	*Medium*
4	*Appearance*	*Mostly upper but in rare cases on surfaces of lower leaves and leaf sheaths*	*On stem, both sides of leaves and spikes and glumes*	*On upper leaf surface, also leaf spikes and glumes, rarely leaf sheath*
5	*Condition of the appearance of pustules*	*Appear singly do not coalesce* *Flecks do not appear*	*Primary pustules are separate, scattered but heavy ones coalesce* *Flecks appear and epidermis broken and torn appearance*	*Seen as harrow stripes* *Flecks do not appear* *No breakage of epidermis*

Ecology requirement

- *Leaf rust requires low land and midland.*
- *Stem rust requires midland and highland.*
- *Stripe rust requires highlands and temperate areas.*

4.2. DISEASES OF BARLEY (*Hordeumvulgare*)

4.2.1. Smut of barley (*Ustilagohordei*)

Significance and distribution of covered smut of barley

Smuts are common in barley fields. Serious crop losses from smut occur only in barley and oats.

Disease symptoms

⇒ The smut does not become evident in the field until the ears are formed.

⇒ The affected ears may emerge about the same time as the healthy ears but remain shorter and are usually retained with in the sheath for a longer time before appearing or may sometimes fail to emerge at all.

⇒ The black spore mass of this smut remains covered by more or less firmly adhering membranes of the grain and the basal part of the glumes.

⇒ Every ear in a diseased stool and every grain in a diseased ear are affected.

⇒ The awns remain intact though they may be sometimes found partly withered.

⇒ The spore masses are held together due to deposition of fatty substances, a feature which renders seed treatment difficult unless the fat is removed.

⇒ As the crop approaches maturity the smutted ears become very clearly visualize.

The smut sori are mostly broken when the grains are threshed and then the spores get mixed with and stick to healthy seeds.

micro–organism of smut of barley

Covered smut of barley is caused by the fungus *Ustilagohordei*. The spore masses are dark brown to black. Teliopores (chlamydospores) are spherical or sub- spherical to angular, brown by transmitted light, paler or lighter on one side.

Host range

The pathogen commonly attacks barly and oats in the graminae family.

Disease transmission

The fungus is carried over from cycle to cycle as a seed – borne pathogen, either as dormant spores or a mycelium in hulls and on seed coat of grain. The disease is externally seed borne of whose spore masses are dispersed and contaminate the healthy seeds and grains during threshing. Longevity of the organism is 23 Years.

Disease cycle

Germinating teliospores develop a promycelium and four primary sporidia, from which abundant secondary sporidia are produced. Primary infection occurs on young seedlings by sporidia when the latter have contaminated the seed coat surface or from the soil. Secondary spread is always by means of sporidia. The infecting chlamydospores germinate at the same time as the seed. Further development of the pathogen follows the growth of the host. The sporidia infecting the seedlings establish dikaryotic mycelium that grows systemically in the host. In the ovary, the mycelium is transformed into teleutospores, which adhere to seed surface or fall on the ground and perenate till the next crop is sown

Favourable environment for the disease

The disease is favoured by cool climatic conditions and this situation is usually met in the highlands and medium elevations.

Management of covered smut of barley

Use of resistant varieties is the best and most effective control method against covered smut of barley appropriate fungicides (organomercurials 2.5 gm, carboxin or pyracarbolid 1-2 gmthiabendazole 1.2 gm per kg seed) also give good results as seed treatment.

4.3. MAIZE (*ZEA MAYS* L.)

4.3.1. Head smut or Common smut of maize (*Ustilagomaydis*or *Ustilagozeae*)

Causal organism

-Ustilagomaydis

Symptom

- The fungus induces gall formation on infected tissues. The extent of damage depends up on the site of galls.
- When the galls formed on the cobs, they cause extensive damage. These galls may also appear on the stem, leaves, axillary buds and parts of the male flower.
- On the whole, they appear more commonly where ever embryonic tissues are present.

- As the galls are enlarging, they appear light colored to almost white. With the darkening of inner tissues due to spore formation the white outer membrane (epidermis) ruptures and exposes the black spore mass.
- Each individual tumor usually starts from an independent infection at the site where it is formed.
- Stem galls result in loss of yield and bending of the stalk.
- Infection of female flowers gives rise to galls instead of grains.
- If seedlings are affected they remain stunted and weak.
- Late development of galls may sometimes kill the entire plant or plant parts.
- Deep seated alterations may be caused in the inflorescence as a result of infection.

Disease development

The pathogen is carried over on the seed coat but this method of perennation and spread is not responsible for most of the infections. Manure heaps and crop refuse are the chief sources of harboring the primary inoculums. Having a strong adaptability to saprophytic life, the fungus can survive on these sources and produce infective sporidia at the suitable time. Infection of the host occurs during the period of vegetative development. Maize ears are susceptible to infection by the fungus from silk emergence until 8-14 days after emergence. During this period incidence of ears with galls decreases as the silks age.

Systemic infection in the late seedling stage has also been reported but local infections of exposed embryonic tissues are more common. Once the tissues have ceased growth and have reached maturity, the infection can not occur. Mature stems may be infected if there is mechanical injury to the tissues. On infection by sporidia the host tissues are induced to grow in to galls. The mycelium in the galls is intercellular during most stages of gall formation. Before sporulation the enlarged host cells are invaded then they collapse and die.

CONTROL

- Crop rotation.
- Field sanitation and seed treatment may be of considerable help to reduce the incidence of the disease
- The use of resistant varieties results in the best management of the disease.
- Rouging of diseased plants before the smut galls raptures.
- Fungicides sprays give only limited success as control agent. However,carboxin+thiram and benomyl have given systemic protection in to the growing season.

4.3.2. Leaf rust (*Pucciniasorghi*)

Geographical distribution and hosts

Aecial infection occurs infrequently in temperate regions of Europe, USSR, USA, Mexico, South Africa, India, and Nepal on *Oxalis* species. Uredial and telial stages occur worldwide where maize is grown.

Disease symptoms

Pustules (sori) may appear on any aboveground part, being most abundant on the leaves. The pustules occur nearly simultanecously on both leaf surfaces on contrast with southern rust, which has very sparse pustule development on the lower leaf surface. The circular to elongate, golden-brown to cinnamon- brown pustules are sparsely scattered over both leaf surfaces becoming brownish black as the plant matures and teliopores develop. When severe, chlorosis and death of the leaves and leaf sheath may occur. The pustules become erumpent and powdery early in their development.

Causal organism

The uresdospores are cinnamon brown, mostly spherical to broadly elliptical, and 21–30 x 24 -33 um. The spore walls are golden or cinnamon brown, 1.5 – 2 um think, and moderately echinulate with three to four equatorial germ pores. Each spore is binucleate as is the mycelium that chestnut brown to golden brown, smooth, oblong to ellipsoid or obovoid, two – celled with slight

constriction at the septa, measure 14 – 25 x 28–46 um, and are attacked to pale yellowish to brownish pedicels (up to 80 um long) once or twice the length of the spores. The aeciospores are pale yellow, verucose, mostly spherical or ellipsoidal, 13–19 X 18–26 um, and occur in "cluster–cups" one species of Oxalis. *P. sorghi* can be cultured on detached maize leaves floated on a solution containing 5% sucrose and 20 ppm of kinetin

Disease cycle

Teliospores germinate in the spring on certain areas of the world to form basidia on which small, thin–walled, hyaline, haploid basidiosopres are produced. These spores germinate and penetrate the leaves of wood sorrel (*Oxalis* spp.) forming spermagonia with minute spermatia on the upper leaf surface. The spermatia fuse with flexuous (receptive) hyphae of the opposite mating type initiating the aecial stage on the lower surface of Oxalis leaves. The binucleate aeciospores in the "cluster– cups" are windborne and infect maize leaves. These infections give rise to uredospores, the repeating stage of the fungus. In most temperate areas of the world, the fungus does not infect *Oxalis*. Spores are windblown into temperate regions from nearby subtropical an tropical areas where the pathogen persists on living maize plants.

Disease epidemiology

Cool temperatures (16-23^{o}C) and high relative humidity (100%) favour rust development and spread. Some inbred lines show a resistant flecking reaction to infection. Older maize tissue is generally resistant to *P. sorghi*. Numerous physiological races of *P. sorghi* can be separated by their reactions on lines of maize having specific genes for resistance. Resistance in mature plants is polygenically controlled..

Maize rust control measures

1. Resistant hybrids and varieties
2. Fungicides applications, starting when pustules first appear on the leaves may be feasible, especially in seed–production fields.

4.4. DISEASES OF SORGHUM (*Sorghum bicolor* (L.) Moench)

4.4.1. Bacterial Streak of Sorghum (*Xanthomonascampestris*pv. *Holcicola*)

Significance and distribution

Bacterial streak has been reported from Argentine, USA, Australia, South Africa, Nigeria, Argentina, New Zealand, Mexico, and perhaps from other sorghum–growing areas, especially the USSR, India and the Philippines. There is no evidence to show the extent of crop losses due to this disease, but considering the leaf damage it can cause under favourable conditions, forage and grain losses could be considerable.

Disease symptoms

Lesions first appear as small, interveinal, water- soaked streaks, which under favourable condition, broaden into irregularly shaped areas with necrotic centres and red margins. Symptom development varies in different hosts; genotypes produce symptoms closely resembling those of bacterial leaf stripe. Color of lesions corresponds to host genotype. Lesions may extend to 30 cm under favourable conditions often coalesce to produce blotches covering most of the lower portion of the leaf. Abundant bacterial exudate is produced on both surfaces of infected leaves.

Causal micro – organism

Bacterial leaf streak is caused by *Xanthomonascampestris*. The cells are Gram- negative, non–spore forming rods measuring 0.4- 0.9 x 1.0-2.4 um and having one or two polar flagella. Colonies on yeast extract–glucose–calcium carbonate agar slow-growing, yellow, round, and very viscous.

Host range

Bacterial streak of sorghum occurs in grain sorghum, maize and sudangrass. Pathogenicity tests gave negative results on sugarcane, pearl millet, finger millet, and proso millet.

Disease transmission

The bacterium perenates in infested debris and dissemination over long distances probably occurs by means of debris and infested seed. Seed transmission canot be overruled, since the infection is seen as early as the second – leaf stage in seedlings.

Disease cycle

Xanthomonascampestris enters the host plant probably through wounds, stomata, or hydathodes. The pathogen is most likely spread by rain and wind, and dissemination appears to be favored by warm weather.

Epidemiology

Bacterial streak appears and can be severe in warm, wet or humid weather. Growth chamber studies at 50, 75 and 90% RH showed that bacterial streak is not influenced by high relative humidiity levels (as is bacterial strip), but optimum temperatures for disease expression were 30^{o}C.

Disease management

Bacterial streak can be controlled through:

1. Planting resistant cultivars or hybrids
2. Crop rotation including non – host crops and legumes
3. Destruction of crop residues after harvesting

4.4.3. Leaf rust (*Pucciniapurpurea*)

Significance and distribution

Leaf rust caused by *Pucciniapurpurea* is known to occur wherever sorghum is grown. The disease naturally stabilizes at a low prevalence where there is survival stress imposed by environments with unfavorable temperature and humidity and widespread cultivation of slow-rusting commercial sorghum. However, under environmental conditions favouring the disease, poor panicle exertion and shriveled grain caused by rust infection may be responsible for grain yield losses of up to 65%.

Disease symptom

In the field, young plants are rarely affected by rust. The typical symptoms are more readily expressed in plants that are one and one half to three months old. Scattered purple, red, or tan flecks appear on both surfaces of leaves; the color depends on the plant pigmentation. In cultivars that react hypersensitivity, the flecks remain restricted. In susceptible cultivars, the flecks enlarge to form blister like, dark reddish brown pustules (uredinia) about 2.0 mm long, which lie parallel to and between the veins. Eventually, the dried brown epidemis over the pustules ruptures as aggregates of uredioniospores push through it to reveal the powdery mass of reddish brown spores underneath. Gradually, most uredinia are converted into telia, recognizable by a change in the color of pustules to blackish brown. New teliosori are produced independently of the uredinia on both surfaces of leaves, especially on the lower surface. Such telial pustules are elliptic to oblong. On peduncles, reddish brown to blackish brown uredinia and telia are seen as long streaks, stripes, or narrowly elongated lesions, which are rarely sporopogenous. Sporulation on peduncles occurs more readily on elliptic to oval pustules when the environment is favourable.

Causal organism

Pucciniapurpurea is the fungus that causes sorghum leaf rust. Unicellular urediniospores (30-42 x22-30 μm) are pedicelled, tawny reddish yellow, and oval to elliptic. Four to 10 equatorial or scattered germ pores are located in the finely echinulate, cinnamon to reddish brown spore wall. Teliospores (40-60 x25-32μm) are bicelled, smooth, oblong to ellipsodia, and dark brown and have a hyaline to yellowish –tinged, stout, persistent pedicel of variable length. The spore wall is constricted at the centrally placed horizontal septum and is uniformly thick. A single, indistinct germ pore is located apically or laterally near the septum of teliospores. Through this germ pore, a long, apically triseptatepromycelium is produced. Each of the four cells of the promycelium bears a single elliptic basiospore on sterigmata.

In the pustules, urediniospores and teliospores are interspersed with abundant paraphyses, which are short, clavate to capitates, hyaline-to ochraceous-walled, and usually bent inwards.

Disease cycle and epidemiology

Spores of *P. purpurea* are short-lived in the absence of a living host. Several infected perennial and collateral hosts and stray sorghum plants provide windblown urediniospores, sometimes from great distances, which function as initial inoculums for infection in the field. Urediniodpores thrive on rationed and successively planted sorghum. Secondary spread in the field occurs by means off airborne urediniospores and is especially favoured by intermittent light drizzles and heavy dew.

Urediniospores germinate within 1-2 hours by a single germ tube, which alternates closely to leaf surface. The tip of the germ tube swells to form an appressorium over a stoma within 4 hour of inoculation. Penetration always occurs through the stoma by an infection peg, which originates from the appressoruim. Abundant, irregularly septate, intercellular hyphae branch extensively within the leaf tissue at and near the site of infection. The first macroscopic symptoms are pinhead-sized chrolotic flecks, which appear within 48 hours after inoculation. Before sporulation, hyphae aggregate beneath the epidermis in a pseudoparenchymatous layer with dense, broad cells from which urediniospores are produced. As the urediniospores mature, they gradually push against the epidermis and finally rapture it. Fully mature uredinia can appear 10-14 days after inoculation.

Disease control

In regions where rust becomes an economically important disease, the growing of less susceptible or resistant cultivars may provide the only practical control. Selective fungicides that suppress the disease are usually not economically justified.

Head smut of sorghum

Disease causing organism

⇒ *Sphacelothecareiliana*

Symptom

Infected sorghum plants are somewhat taller than the healthy ones and also mature earlier. The first signs of sorghum infection appear at heading time when the young head with in the enclosing “boot “is seen to be completely replaced by a large whitish gall. Sometimes the head may be only partially infected, the smut free portion being sterile and sometimes developing in to

a leafy shoot. Very often all the heads of an infected plant become smutted, but sometimes the upper panicles escape although usually remaining sterile.

The smut galls (sori) on sorghum may be large, up to about 10-15 cm, irregularly oval or pointed at the apex, at first covered by a whitish grey membrane of the fungus. This often ruptures before the head emerges from the boot exposing a mass of dark brown black powdery spores. The spores are blown away by wind leaving the dark net work of fibers. The sori of head smut are very characteristic usually they completely replace the head so that little evidence of its original inflorescence structure is apparent.

Disease development

The pathogen is mainly soil borne and to some extent seed borne, infection usually taking place at the seedling stage, that is from seed germination up to four leaf stages. The plumule is more susceptible than the roots. The hypocotyls are more severely affected than the coleoptiles. The infection site on all organs is the meristematic region. There could be a rare floral infection. Inoculums potential in the soil, soil temperature and soil moisture content determine seedling infection. Infection is favored by high soil temperature and by low soil moisture. The spore mass consists of numerous chlamydospores intermingled with sterile cells. The spore balls readily break up at maturity. The fungus is systemic with in the aerial parts of the plant. Best spore germination occurs between 27-31^{o}c

Economic importance

The disease does not involve serious over all crop loss to sorghum. Only relatively few plants are infected in general. In some areas, the disease may cause severe damage where by from 10 to 60% infection. There is reduction in stalk diameter and fodder value.

Control

- ✓ Collection and destruction of smutted heads before spore release
- ✓ Use of disease free seed
- ✓ Seed dressing to avoid seed borne spores such as copper carbonate and organomercurials
- ✓ Soil treatment with fumigants ex. Chloropicrin
- ✓ Use of resistant varieties

4.5. Fababean diseases

4.5.1. Chocolate spot (*Botrytis fabae)*

The Pathogen and Disease Development

Thirteen fungal diseases have been reported on faba bean in the Peoples' Republic of China. In Ethiopia the major diseases that inflict heavy crop damage are chocolate spot (*Botrytis fabae*Sard.*)*, rust (*Uromycesvicia-fabae*L.), and black root rot (*Fusariumsolani*L.) (Habtu and Dereje, 1985; Dereje and Tesfaye, 1994). Survey conducted in 1996 and 1997 revealed that viruses like Faba Bean Necrotic Yellow Viruse (FBNV) and Beet Western Yellow Virus (BWYV) were commonly attacking faba bean fields and cause yield reductions in Ethiopia.

Chocolate spot is the most common and most destructive disease on leaves stems, pods, seeds,flowersand reduces faba bean production globally. In the Maghreb region losses due to chocolate spot disease can reach 60-80% on susceptible cultivars. The disease may also causes total crop failure under severe epidemic conditions.

Chocolate spot disease of faba bean is caused by two species of *Botrytis* namely *B. fabae*and*B. cinerea*. Taxonomically the genus *Botrytis* belongs to sub Deutromycotina (The imperfect fungi), class: Hyphomycetes, order: Hyphales (Moniliales), family: Moniliaceae. The fungus is usually referred to by its anamorph (asexual) form because the sexual phase is rarely observed. For quite some time both of these species were considered equally responsible for disease development, but at present *B. fabae*is considered as the major cause of the disease especially under conditions of high humidity and rainfall. In Ethiopia only *B. fabae*is known to cause the disease.

Disease Management Approaches

Cultural practice suchas altering planting date, crop rotation, cropping pattern, application of potash fertilizer and ditching to improve drainage were found to reduce chocolate spot disease. The choice of planting date in relation to crop management offers a good means to reduce the disease in the field.

Mixed cropping is an important feature of cropping systems in the tropics both for disease management and increasing productivity. Mixed cropping slows disease progress compared to sole cropping in some pathosystems.

Spraying faba bean with benzmidazole fungicides at flowering and podding stages with either Bavistin (carbendazim) or Topsin-M (Thiophanate-methyl) was found to be effective in suppressing lesion development and inhibiting spore production. However, repeated application of fungicides may not be economically justified in chocolate spot control. Therefore, fungicides can only be applied in a high-risk situation and seed multiplication, especially for protecting precious germplasm materials.

Biological control using microbes is becoming a critically needed component of plant disease management. In Ethiopia, this method of disease management is at its infant stag

4.5.2. Faba Bean Rust

Geographical distribution and its economic importance

Faba bean rust is a disease present in almost every area of the world, where faba bean is grown. Severe attacks have been reported in the Middle East and in eastern Africa, where rust is currently a major threat to faba bean production. For instance, in northern parts of Egypt it is currently considered one of the most destructive disease attacking faba bean. In Ethiopia rust is widely distributed and economically important disease.

The pathogen

The pathogen, *Uromycesviciae-fabae*(Pers.) Schrot., causes rust in faba bean and lentil and belongs to the family Pucciniaceae, order Uredinales, class Urediomycetes, phylum Basidiomycota and kingdom Fungi. Oval, brown-colored uredial pustules, up to 1 mm in diameter, develop on both surfaces of leaflets, petioles, stems and pods. The echinulateduredospores, 22-28 µm x 19- 22 µm, have 3-4 germination pores and are borne on single pedicels. The dark brown to black, elongated telia are formed late in the season below the epidermis and then are erumpent on leaves, but remain covered by the epidermis on stems for an extended period. Globose to sub-globose unicellular teliospores, 25-40 µm x 18- 26 µm, have a single germination pore and are borne singly on pedicels.

Disease cycle

*Uromycesviciae-fabae*is a macrocyclic rust fungus, it exhibits all five spore forms known for the Uredinales. It is also autoecious, as all spore forms are produced on a single host (Mendgen, 1997). After overwintering on residual plant material, diploid teliospores germinate in the spring with a metabasidium. After meiosis, the later produces four haploid basidiospores with two different mating types. These are ejected from the metabasidium and after landing on

leaf of a host germinate and produce infection structures. Pycnia are produced which contain pycniospores and receptive hyphae. Pycniospores are exchanged between pycnia of different mating types and after spermatization, dikaryotization occurs in aecialprimordia. An aecium differentiates and dikaryotic aeciospores are produced. These aeciospores germinate and form infection structures from which uredia which produce urediospores, are formed. Urediospores are the major asexual spore forms of rust fungi produced in massive amounts through repeated infection of host plants during the summer in the temperate region. Urediospores are aerially dispersed and can travel thousands of kilometers carried by the wind (Brown and Hovmller, 2002). In the fall, uredia differentiate into telia, the nuclei fuse during sporogenesis and single-celled diploid teliospores develop for the winter (Voegele, 2006). Therefore, the fungus survives on stubble and self-sown volunteer bean, so removal of stubbles and avoidance of volunteer faba bean plants is sought to be an important factor in the order to minimize rust epidemics.

Epidemiology of Faba Bean Rust

Disease development

The amount of disease that develops in a plant community is dependent on properties of the host, the pathogen, the environment and time. The environment can affect both the susceptibility of the host (e.g. by creating stress in the plant) and the activity of the pathogen (e.g. providing moisture for spore germination). The pathogen and the host can affect each other's performance. The plant can also change its environment, by altering the microclimate around it. These factors operate in time for rust to develop.

Faba bean rust can infect the leaves, stems and pods of susceptible varieties. The disease usually starts late in the season, when pod filling has started Urediospores of *U. viciae-fabae*germinate well in a temperature that ranges 5-26^{0}C, with fastest germination at 20^{0}C. Exposure to 30^{0}C gave poor germination and damaged the spores. Infection of *Viciafaba*leaves depends on presence of moisture film. At 20^{0}C, some infection occurs with only 4 h leaf wetness, but longer wet periods of up to 24 h increase infection. Spore germination was delayed by daylight and by all artificial light sources that contained far-red (700-800 nm) wavelengths. The delay was increased at higher light intensities. When spores were subjected to alternating periods of light and darkness, it was found that 40 minutes of darkness was sufficient for the irreversible induction of germination at 20^{0}C. Infection of volunteer faba bean plants is thought to be an important factor in the early development of rust epidemics.

Symptoms

Numerous, small, orange-brown pustules, each surrounded by a light yellow halo develop on the leaves. As the disease develops, severely infected leaves wither and may fall from the plant. On stem, the rust pustules are similar, but often larger and longer than those found on the leaves. As the spots enlarge, the epidermis ruptures, releasing masses of brown urediospores. Teliospores are produced in higher numbers on stems and petioles towards the end of the season. Isolated rust pustules may also appear on the pods, which can reduce seed weight. Severe infection may cause premature defoliation, resulting in reduced seed size.

Survival and dissemination

The survival of a pathogen between cropping seasons and its effective dispersal to uninfected plants are crucial aspects of disease cycle. If either of these is prevented, disease will not occur. Most pathogens possess mechanisms to survive intercrop periods or periods of unfavourable environmental conditions. In production areas, where prolonged hot and dry summers prevail, infected crop debris and teliospores left in the field or carried in seeds serve as primary inocula.

Teliospores are resistant to adverse conditions and are the primary surviving structures between crop seasons. The teliospores readily germinate at 17-22^{0}C without dormancy and remain viable up to two years in a resting state. In cooler production regions, the uredospore may also be an important means of survival between crop seasons. Primary inoculum also comes from other hosts, such as vetch. Secondary spread is by means of aeciospores and uredospores, both of which readily germinate on plant surface under humid conditions and are dispersed by wind.

Management of Faba Bean Rust

Cultural management

Cultural control involves practices that modify the environment to favor crop growth, but discourage or avoid the conditions that favor disease development. In all instance, a detailed knowledge of the key factors that influence main activities of a disease is essential. Cultural methods, which can be manipulated to control disease, include mineral nutrition, time of sowing, seed rate, special cultivation, water management, crop rotation, sanitation and clean seed.

Use of resistant varieties

The use of varieties resistance to particular disease is one of the main methods of disease control. Host-plant resistance is the inherent ability of crop plants to restrict, reduce, or overcome pest infestation.

Chemical control of faba bean rust

Mancozeb and tebuconazole are the most effective fungicides for preventing yield reduction and severity of rust disease.

CHAPTER FIVE
MAJOR DISEASE AND INSECT PESTS OF INDUSTRIAL AND CASH CROPS

5.1. Disease of sugarcane (*Saccharumofficinarum* L.)

5.1.1. Sugarcane smut (*Ustilagoscitamiea* H. & P. Synd.)

Significance and distribution

Sugarcane smut is a well-known disease and is reported from India, Java, Formosa, Philippines, Africa (South Africa, Tanzania, Uganda, Kenya), Mauritius, Austitius, Italy, and other tropical and subtropical countries. If the whip smut incidence is serious, about 35-50% of the crop may be affected. The loss is both in yield of crop and sucrose content. It is primarily a disease of wild canes and those canes that most nearly approach the wild varieties are more susceptible than the improved thick canes. However, thick canes in the tropics are by no means immune. The causal organism occurs on grasses that may serve as collateral hosts.

Smut (*Ustilagoscitaminea*)

Economic importance: According to a yield loss assessment study carried out at Metahara, smut accounted for 19 to 43% cane yield loss.

Symptoms: The patent symptom of the disease is production of a black whip-like structure from the central core of the meristematic tissue. They vary in length from few cm to 1.5 m, with terminal whips usually longer than those from the side shoots. The whip when young is covered by a thin, white and papery membrane; this is the epidermis of the host. Upon maturity it ruptures and millions of tiny black spores are liberated and disseminated by wind.

Transmission:

- **Wind:** The major mechanism of transmission from plant to plant is by wind. Whips serve as sources of inoculum within a field. The distance over which spores can be wind disseminated without loss of viability is estimated up to 40 m.

- **Planting material:** The disease is also transmitted by latent infection in the planting material.
- **Soil:** Another possibility of transmission is by soil-borne inoculum. On the advent of wet weather the spores deposited on soil germinate and infect the actively developing shoots.
- **Water:** Spores which are found on or in the soil may also be carried by irrigation water or rain water during the rainy period and infect the cuttings at planting time or the buds on the basal portions of the cane stool. Similarly, spores initially deposited on leaves could be washed into the leaf axils and then to the buds by rainwater.

The causal organism

The disease is caused by *Ustilagoscitaminea* Synd. The fungus was first described by Rabenhorst in 1870 as *Ustilagosacchari*. The spores of *U. scitaminea* are spherical, smooth-walled, light-brown and about 5-10 μm in diameter. They are loose and very light, easily disseminated by wind. Under moist conditions they germinate easily forming short promycelium that divides into 3 or 4 cells by transverse septa. From each of these cells arises a sporidium on short stalk and is easily detached. The sporidia are elongate, single celled bodies that geminate to produce the infection tread. Under favourable conditions -good humus and moisture-the sporidia may bud off to produce more sporidia in short chains. The smut mycelium is intercellular and sends haustoria into the host cells.

Disease cycle

The optimum temperature for spore germination is about 25-30 ^{o}C, the maximum being between 36 and 40 ^{o}C and minimum between 5 and 9 ^{o}C. The spores are killed instantaneously at 62 ^{o}C but in ice they survive for more than 3 days. One hundred per cent relative humidity is essential for spore germination. No germination occurs at 90% relative humidity. The spore germination is practically complete in about 24 hours in water. Under dry conditions the spores remain viable for more than 7 months but under moist conditions they lose their viability within 3 weeks.

The spores from the whip- like structure on affected canes are blown about by wind and some of them fall on the near-by canes. They are found deposited at the junction of the leaf and the leaf sheath from where they travel down the sheath and reach the tender nodal region and the young eyes. There is plenty of moisture at the base of the leaf sheath facilitating germination of spores. Infection can also occur through the young germinating shoot. Any injury on the scales of eyes

and any part of the cane facilitates infection if spores fall on the injured tissues. Infection is most severe at optimum temperature and relative humidity for spore germination.

Field experiments have shown that the smut is perpetuated in any of the following ways: BY planting sets of smutted canes; by spores borne on buds; by infection of buds on standing canes; and rottoning the smutted canes.

Control:

Use of resistant varieties

Cultural practices:

a. **Roguing:** Roguing of smut affected stools or shoots has been widely recommended as a useful means of reducing smut inoculum from the field.
b. Pre-planting irrigation: Pre-planting irrigation may also suppress the disease inoculum in the soil apart from its uses for furrow correction.
c. **Hot water treatment (HWT):** Heat therapy is effective treatment in eliminating external infection (i.e. the spores contaminating the host surface) and the internal dormant infection of buds, if setts are subjected to appropriate hot water and time combination.
d. **Chemical control:** Fungicides eradicate smut from the planting material or prevent re-infection when they are used as a pre-plant treatment of setts. Fungicide treatment should be applied for varieties whose smut reaction is ranked from moderately susceptible to very highly susceptible. But, in case other sett-borne diseases such as pineapple disease and red rot are reported, it is advisable to treat all the varieties with fungicide.

5.2. DISEASES OF COFFEE (*Coffeaarabica* L.)

5.2.1. Coffee berry disease (CBD) (*Colletotrichumkahawae*)

Significance and distribution

About 60 different diseases attack coffee in the world and 11 diseases are recorded in Ethiopia during 1968-1984, coffee berry disease and coffee leaf rust being the most production limiting factors in East Africa. In Ethiopia, CBD is the only devastating diseased of coffee in Ethiopia while the rest are minor diseases. McDonald recorded CBD for the first time in Western Kenya in 1921. By 1939, it crossed the Rift Valley and reached upper Kiambu in 1951, since then it spread rapidly to other coffee growing areas in Kenya. Outside Kenya CBD was observed in

other countries including Angola (1930), Congo (1937), Cameroon (1957), Rwanda (1957), Uganda (1959), Tanzania (1964), and Ethiopia (1971).

The disease has how spread to most tropical African countries. There is no concrete evidence as to the existence of CBD outside Africa, although coffee necrosis has been reported from Guatemala, Costa Rica, India, Brazil, Colombia, and Java. Although CBD was identified in Ethiopia in 1971, the disease has how spread to almost all the major coffee producing areas including, Bale, GamuGofa, Hararghe, Illubabor, Kaffa, Shewa, and Sidama. Severe disease has been occurring since June 1972 in these areas. CBD chiefly attacks Coffee Arabica, the greatest damage occurring due primarily to infection of berries, which result in fruit (bean) rotting by active infection leading to total destruction of the crop.

The crop loss varies from place to place and from year to year due to variations in management practices, time and methods of assessment, spraying fungicides, weather conditions (especially rainfall, relative humidity, and drought), effect of diseases, insect pests, and such other factors.

In Kenya, the annual loss attributed to CBD usually ranges between 20 and 80% of the potential harvest depending on the climate and locations. In Ethiopia, crop loss due to CBD in some coffee producing areas exceeds 50%, even yield loss ranging between 30 and 100% in some locations. The national average loss in 1979 was estimated to be 18%, however, regular loss assessments made since 1974 have indicated the average loss as 20% of the potential production for Ethiopia. During 1972-1980, losses due to CBD on non-sprayed experimental plots (IAR) were 70.4% of the potential harvest of susceptible varieties.

Disease symptoms

The pathogen frequently invades the branches especially those with maturing bark changing from green into grayish cortex, though the severity is low. Acervuli with pinkish masses of conidia, in sticky mucilaginous matrix, are initially produced on the surface of maturing wood of twigs (branches). Generally the branches may die.

Circular to elongate, necrotic yellowish –brown to blackish brown marginal spots (with whitish-grey in the centre at later stages) appear on both sides of the leaves. The leaf veins borders the spots with up to 3 cm in size. The black dots on spots are concentrically arranged mainly on the

upper surface of the leaves. The leaves of the branches may defoliate or shed prematurely except the terminal parts of the leaves. Infection usually starts at the margins. However, leaf infection is not common.

A dark-brown streak on the petals is the first visible symptom. The whole flower is sometimes killed but losses are not generally serious. Small, dark-brown sunken spots or lesions with minute black dots, called acervuli, on the green berries characterize CBD. The acervuli produce pinkish conidial masses later on. The lesions appear on both green ripe berries and later change to black and coalesce. The majority of the diseased berries drop off or shed but a small number remain on the branches as black mummies. If the attack starts at an early stage in development of the fruits, the seeds turn black and decay or rot. Following late infection, it is only the pulp of the fruit that decays. Since the fruit pulp of attacked berries tightly adheres to the beans or seeds puling is rendered difficult.

Causal pathogen

The cause of CBD is the fungus *Colletotrichumcoffeanum*Noack, now named as *Colletotrichumkahawae* Waller. It belongs to the family Melanconiaceae, order Melanconiales and class Deuteromycetes. The prefect state is *Glomerellacingulata* characterized by perithecia and production of ascospores, rare or not known in nature. The acervuli are produced on lesions formed on leaves, twigs, and berries. They are 0.15-0.8 mm in diameter. These black flat structures are arranged in roughly concentric rings. Setae are borne by acervuli and are brown numerous sterile special hyphae that emerge from the acervuli. They measure 40x 4-5 μm. The conidiophores are short, hyaline, spore-bearing stalks or hyphae of the acervuli, containing spores at their apex, 18-20 x 4 μm. the spores or conidia are unicellular, hyaline, and with shapes between oblong and bean-like forms, i. e. cylindrical straight to curved, and measure 6-33 x 3-8 between μm, averaging approximately 20-4 μm.

Survival mechanism

The pathogen survives on all infected plant parts including the leaves, barks of branches, and mostly infected berries as mummies. In the absence of the infected berries, the bark constitutes the main source of primary inoculum for the start of the disease on the fruit berries. Epiphytotics

is initiated each year by conidia from the bark and the mummies. Once some berries have become infected, very large numbers of spores are produced and the disease starts very rapidly.

Spore production

Spores are formed on the twigs under humid circumstances, i.e., several hours of very humid conditions that could result from the rainfall in late afternoon are required for the production of spores on the fruiting bodies of the fungus situated on the maturing bark or tissue or on the lesions on the berries. The production of inoculum or sporulation is most abundant at the onset of the long rains and short rains. When the lesions on the berries start producing spores, berry-to-berry infection becomes very important.

Dispersal mechanisms

Tools such as pruning shears, animals, birds, human beings are suspected of transmitting the pathogen from plant to plant. Insect vectors may also transmit the disease. The spores are not easily distributed or detached by wind since the spores on the acervuli are mucilaginous or slimy in nature. The conidia are separated and dispersed slowly by water-splash over comparatively short distances in water films and in splash droplets cause by rainfall. After dispersal of the spores by water into the healthy berries the water droplets must remain on the berries for at least five hours to enable the spores to germinate and to infect the berries.

Pathogenesis

Spores need four hours wetness for germination and penetration of the outer layer of the berry. Lesions become visible after two to weeks if weather is favourable for disease development. The coffee flowers are easily infected with the conidia and the very young berries, pin-heads, become resistant to infection for the first 8 weeks after flowering. They become very susceptible, however, when they are expanding rapidly in the period 8-15 weeks after flowering.

By the 20^{th} week they reach ‘hard –green’ stage when they have obtained maximum size. At this stage, the berries are resistant to ‘active infection’ but they may develop ‘latent infection’ that remains invisible until the crop ripens 10-16 weeks later when the symptoms of ‘brown blight’ develop, i.e., 30-36 weeks after flowering. Active infection of expanding berries is likely to lead to direct loss of crop due to berry shedding. The latent infection of hard berries leading to brown

blight does not cause loss of the crop if berries are picked rapidly but it can cause difficulties in processing the berries and subsequently lowering of the quality of beans.

Epiphytotiology /epidemiology

CBD is favoured on areas above 1600 m above sea level. That is why plantation at higher altitudes are more liable to attacks than those on lower regions. The CBD incidence depends largely on climatic conditions including high atmospheric humidity, fog, and low temperature (15-28 ^{o}C, optimum 22 ^{o}C) favouring the spread of the pathogen. Hence CBD occurs primarily in damp districts where there is more than 1100 mm of rainfall per annum. It is also favoured by shade and too close or narrow spacing. During drought, it is dormant but commences to grow with the onset of rains. Under unfavourable circumstances, the fungus dies and the lesions become scab lesions. However, scab lesions, scab lesions could also be symptomatic indications of greater host resistance.

Management of coffee berry disease (CBD)

The early crop escapes infection, e.g. when the crop flowers earlier and berries usually expand rapidly in the dry months and hence the crop finds tends to escape an attack of active infection, for instance, by irrigation management to induce flowering earlier; however, it can be vulnerable to latent infection when long rains commence. When the main crop is late, the main flowering period will be during the small rainy season and, consequently, the crop will be vulnerable to active infection and losses from berry shedding at this can be severe.

Management of coffee berry disease may categorized into:

1) Improvement of the coffee growing conditions in the orchards through:
    a) Extra dose of fertilizers or manure application,
    b) Good crop husbandry,
    c) Microclimate improvement by weed control
    d) Microclimatic improvement by pruning coffee trees and shrubs,
    e) Well balanced shade conditions to reduce inoculums potential, and
    f) Removal and burning of infected branches.
2) Spraying with fungicides

3) Use of resistant varieties

Since the use of fungicides has got certain drawbacks (high cost of fungicides and equipment, low efficacy, tedious fungicide application, and the like), the use of resistant varieties has now been suggested as the best long-term prospect for CBD control in Ethiopia. The coffee varieties could be susceptible, tolerant, or highly resistant. You should not use susceptible varieties for raising seedlings. One of the mechanisms of defense in coffee against *C. kahawae* is the nature of cutin that acts as a physical barrier and enables a greater degree of resistance. Also, studies of cultivars of varying (Gibbs, 1978) indicated that scab lesions are symptomatic of greater resistance, possibly through the production of a phytoalexins.

5.2.3. Fusarium wilts (*Fusariumxylarioides*)

Disease symptoms

In herbaceous plants the chief symptom would be immediate wilting but in coffee there is chlorosis, rolling and crisping of the leaves that turn brown and fall. The infected leaves become yellowish and later turn brownish and finally fall or shed. The branches or twigs dry up and eventually the whole plant dies. Attacked braches show necrotic sunken lesions, mostly at their bases, the same damage is also caused in the root collar regions.

The appearance of the bark of trunk is typical; it is hypertrophied, split into numerous vertical or spiraling cracks through which brownish blackened deeper tissues can be seen. The bark of the main roots is also affected and the inner tissues wet. The conducting tissues or vessels are changed to a brownish colour and are filled with hyphae, deposits, and gum. Thus the symptoms appear first at the collar and the internal wood tissues are brown or black and full of fungal mycelium.

When the bark of the stem is peeled at the crown, brownish to blackish fungal structures are observed. Such coffee tree is not easily felled if pushed or pulled. The infected side of the stem is peeled. The incubation period, i.e., from infection to symptom appearance may take from 1 to 2 years.

Causal pathogen

The causal fungus of the disease is *Fusariumxylarioides* (anamorphic state) or *Gibberellaxylariods* (telemorphic state). It belongs to the family Hypocraceae, order Sphaeriales in the class Ascomycotina. In the course of growth after infection, bluish black perithecia containing numerous asci, are formed in the bark region; and in some case of humid weather, conidiophores appear bear three-septatedmacroconidia with an average size of 14-23 x 2-3 μm. The octocellularascospores are 12-14x 4-6 μm in size.

Pathogenesis

Infection is through wounds, cuts or bruises formed during splashing or cultivation, or by insects. The pathogen could be transmitted from plant to plant through agricultural tools such as digging tools, knife, cutlass, etc. The mycelium penetrates in bast (phloem) and wood and develops intercellularly. Due to the exuberant or plentiful growth of mycelia, especially in humid regions, the xylem vessels of the infected plants are more and more penetrated and eventually clogged. As a consequence, symptoms of external wilting appear which gradually kill the plant.

Control measures

The following measures can be taken to manage tracheomycosis on coffee plantations.

1) Slash, dig or cultivate the soil carefully so as not to damage or form wounds on the stems.
2) Dig out and spray on the spot with carbolic (copper fungicides) and burn the whole trees when dry
3) Replant at once since young trees are resistant
4) Use resistant varieties or species.

5.2.2. Coffee Leaf Rust

Coffee Leaf Rust (CLR) is caused by the fungus HemileiavastatrixBerk. and Br., which was discovered in Kenya in 1861; the first report of damage to coffee trees was from Sri Lanka, in 1869.Coffee rust is the most significant disease of coffee and is widespread in all regions where coffee is cultivated. In all of these regions, the main damage to coffee trees consists of early loss of leaves, drying of branches before budding and decreased productivity in the subsequent agricultural year. The damage caused by this disease varies regionally, depending on climate,

altitude, cultivar susceptibility, cultural practices, and the fruit load on plants.Heavy infections cause decreased photosynthesis and increased defoliation (and producers continueto incur significant costs due to crop losses and mitigation efforts, with yield losses of 6–13% and annual costs worldwide due to coffee leaf rust estimated to be US$1 billion.

Management Practice (Same to coffee berry disease)

1. Use of resistant varieties
2. Cultural practices
3. Chemical control method

5.3. Disease of cotton (*Gossypiumhirsutum* L.)

5.3.1. Cotton angular leaf spot, black arm, baxterial blight. Boll rot (*Xanthomonasmalvacearum* (E.F. Smith) Dowson)

Significance and distribution

Angular leaf spot or black arm is the most serious bacterial disease of this crop. It is found in all cotton growing areas of the world including USA, South America, Egypt, Sudan, and other African countries, Sri Lanka, China, Australia, etc.

Disease symptoms

The bacterium attacks all the plant parts above ground level at different stages of plant growth. The earliest symptoms of the disease is seen in the cotyledons of germinating seeds. Minute, water –soaked spots appear on the under-surface of the cotyledons. These later increase in diameter, turn brown to black and form irregular patches distorting the shape of the cotyledons and causing them to dry and wither. The disease spreads to new leaves formed and the seedling may ultimately collapse and die. On the leaves similar water-soaked spots appear or the under-surface first and then on the upper surface. They increase in size, become angular, bound by small veinlets of the leaf and turn brown to black.

Often the disease spreads along the edge of the veins, hence called ‘vein blight’ or [black vein’. Sometimes large areas are formed due to coalescence of a number of small spots leading to death and shedding of leaves. The infection may also spread to petiole causing them to collapse. In the

affected areas large amounts of bacterial slime are exuded which form a dry film on the brown lesions.

Lesions on stem, petioles, and fruiting branches are dark brown to sooty black. They are elongated and sunken. The affected stems show cracks and gummosis and are easily broken by wind or there may be girdling and death of affected organs. These are 'black arm' symptoms. On the bolls or fruits the disease is characterized by the appearance of water-soaked lesions on the surface. These lesions turn dark –brown and finally black, and are invariably sunken. Young infected bolls fall down prematurely. If they mature, lint is of not much commercial value. The bacterium within the boll passes along the fibers and infects the seed externally. It may also reach the interior of the seed either through micropyle or through punctures.

The causal organism

Xanthomonasmalvacearum (E.S. Smith) Dowson (Synon.*Pseudomonas mavacearum* E.F. Smith; *Bacterium malvacearum* E.F. Smith; *Bacillus malvacearum* (E .F. Smith) Holland; *Phytomonasmalvacearum* (E. F. Smith) Bergey*et al*. is the causal bacterium.

The bacterium is rod-shaped, 0.3-0.6 x 1.3-2.7 μm. It occurs singly or in pairs, is capsulated but forms no spores, and is motile by one polar flagellum. Strain reaction is Gram-negative. The bacterium is aerobic. Colonies on beef extract agar are pale yellow, round, thin, raised, smooth and shining. It liquefies gelatin, digests milk and does not reduce nitrates. Optimum temperatures for growth are variously reported to be 25 - 30 ^{o}C. Maximum temperature for growth is 42^{o}C and minimum 6 ^{o}C. Thermal death point is 50 ^{o}C.

The bacterium remains alive in dried leaves for 17 years and in dry or moist soil for 8 days at 21-33 ^{o}C. However, infected stalks buried in moist soil cause death of the bacterium. The main source of primary inoculum is the seed. The bacterium may be present as slimy mass on the fuzz or inside the seed. Infected cotton bolls, leaves, and twigs present on soil surface also form an important source of carry over of the disease. The infected seed lying dormant in the field and germinating in the crop season are also possible sources of primary infection. Leaves are infected mainly through the stomata. The secondary spread is through wind splashed rain and dew.

High humidity and moderate temperatures (28 ^{o}C) favour development of the disease. Soil temperature and moisture at the time of sowing and a few days after are important. A temperature of 30 ^{o}Cfavours primary infection and secondary infection is better AT 35 ^{o}C. Presence of moisture is very important for secondary infection during the first 48 hours. Dry and hot weather retards development of the disease. There are several races of the pathogen that vary in their pathogenicity on different species of *Gossypium*.

Management of cotton angular leaf spot

Removal and destruction of diseased plant debris is recommended to reduce the soil borne inoculums. Deep ploughing after harvest buries the infected stalks and thus reduces survival ability of the bacterium in soil. Presowing irrigation to enable the left over seeds to germinate followed by ploughing and then planting of the main crop, is followed in some countries. Crop rotation, late sowing, early thinning, good tillage early irrigation and addition of potash to soil help in reducing the disease incidence.

Seed borne inoculums can be laminated by seed treatments. Delinting of seed with concentrated sulphuric acid (by immersing in the acid for 10-15 minutes, then rinsing thoroughly by suspending in water to remove the acid and finally drying and treating with organo mercurial compounds like Agrosan GN, Ceresan, etc) destroys external inoculums on the cottonseed. Treating the seed with antibiotics like streptomycin eradicates the internally seed-borne infection. The bacterium is known to live in the seed for about a year or so and, therefore, ageing of the seed for two years before sowing has also been recommended.

Regular spraying with copper fungicides (0.2 to 0.3 per cent) can check the secondary spread. First spray is given when the crop is 5-6 weeks old. In all 3-6 sprays, depending on severity of the disease, are given at 15-day interval. Seed dressing of cotton with Agromycin (3 gm per 40 kg) and its spray (25 ppm) are most promising in controlling the black arm of cotton.

Control through the development of resistant varieties is possible and provides the best and most effective preventive measure. *Gossypiumherbaceum* and *Gossypiumarboreum* were considered to be practically immune to the disease whereas *G. barbadense*, *G. herbaceum* var. *typicum* and *G. hirsutum* are susceptible.

5.3.2. Cotton fusarium wilts (*Fusariumoxysporum* f. sp. *vasinfectum*(Atk.) Snyde& Hansen)

Significance of cotton fusarium wilt

Wilt disease of cotton is restricted to the black cotton soils, i. e., heavy clay soil with reaction varying from pH 7.6 to 8.0. It is rare in light alkaline or loam soils.

Disease symptoms

The symptoms of cotton disease include the discoloration of tissues and plugging of vessels by hyphae. In very stages of plant growth, vein clearing on cotyledonary and first leaves is also visible. Often the diseased plants are small with smaller leaves and bolls.

The causal organism

The disease is caused by *Fusariumoxysporum*f.sp. *Vasinfectum* (Atk.) Snyder & Hansen. The aerial mycelium of the fungus is white to grayish or bluish purple and purple and often forms a mat on the collar region of the stem near the ground level. The hyphae are both inter-and intra-cellular. The conidiophores are verticillately branched and developed in sporodochia, reduced poonnotes, or sometimes directly on the mycelium. Elliptical unicellular microconidia, averaging 5-12 x 2-3.5 μm in size, occur commonly. Sickle-shaped macroconidia are hyaline, mostly 3-septate, but sometimes 4- or 5 septate. They measure 40-50 x 3-4.5 μm in size. Chlamydospores are terminal or intercalary.

The fungus is soil borne surviving in the soil on stubbles of diseased plants. It has been found up to a depth of 1 metre or below the soil surface but infection is most severe in the first 40 centimetres. The disease is also seedborne. Infection occurs through the root system, the fungus entering the host when the plants are 1-3 weeks old and wilt symptoms become visible when the plants are 5-6 weeks old. The wilting occurs primarily due to excretory toxic substances produced by the fungus in vascular system and translocated through the plant the fungus produces fusaric acid, the fungus toxin, in the plant tissue, in the rhizosphere, as well as in the soil. The fungus also produces pectolytic enzymes to help in breakdown of the cell wall components.

Fusarium wilt of cotton is often inter-related closely with avenues of entrances into the host roots produced by various nematodes, the infestation of which results in loss of vitality in the plant and makes the host plant susceptible to infection by the wilt microorganisms. The optimum temperature for the development of cotton wilt is between 20 and 27 ^{o}C, the maximum being 31 ^{o}C. Most of the deaths in cotton occur when the soil temperature at 6-inch depth is between 22 and 25 ^{o}C and at 15-inch depth 24 and 25^{o}C. Moisture content of 80-90% saturation is best for disease development.

High potash applications alone and fairly high potash applications combination with nitrogen and phosphorus containing salts give definite control of cotton wilt. In sandy alluvial soils, containing nitrogen and phosphorus but lacking in potassium, the application of potash fertilizer reduces wilt. Applications of the trace elements zinc, lithium, aluminium, nickel, boron, cobalt, and manganese inhibits conidial germination of *Fusariumoxysporm*f.sp. v*asin*

fectum. However, boron, lithium and molybdenum are toxic to cotton plants.

Management of cotton fusarial wilt

Field sanitation, crop rotation, and mixed cropping are essential for reducing the wilt disease of cotton. Use of resistant varieties is the most effective control measure. Susceptible varieties of cotton should be replaced by resistant varieties. Proper application of micronutrients (such as zinc) and nitrogen and potash fertilizers reduces incidence of cotton fusarial wilts

CHAPTER SIX
MAJOR DISEASE AND INSECT PESTS OF VEGETABLE CROPS

6.1. DISEASES OF POTATO (*Solanumtuberosum*)

6.1.1. Potato late blight (*Phytophthorainfestans*)

Significance and distribution

Potato late blight is among the most destructive of all plant disease. Devastation caused by the disease in the late 1840s in Europe led to food shortages throughout Europe and gave rise to the Irish potato famine, leading to the deaths or emigration of an estimated three million Irish people. As an asexual organism, *Phytophthorainfestans* is a near-obligate parasite in nature. The

foliar phase of potato late blight limits production of tubers and because of infections on tubers destroy a potential food source.

Disease symptoms

The symptoms of late blight in the foliage of potato and tomato begin as hydrotic areas with indefinite margins at the tips or on the margins of leaflets. These lesions become necrotic and turn brown an almost black. The necrotic tissue dries and shrivels. Often, a chloranemic border develops around the necrotic areas; this yellow band is widest during wet weather. Necroses extend along the petioles and stems, which are girdled in wet weather. Necrotic areas on stems are not surrounded by yellow borders. The characteristic, moderately offensive odour that emanates from diseased plants is particularly noticeable when the disease id developing rapidly in the field. Slightly sunken hydrotic areas develop in the outer tissues of infected potato tubers. These areas become necrotic and turn brown to purple.

During wet weather, the signs of the pathogen-sporangiospores (conidiophores) and sporangia (conidia) – can be seen as a downy growth, particularly on the axial surfaces of leaflets.

Life history of the pathogen

The epidemiology of late blight depends on the occurrence or absence of sexual reproduction. In locations in which sexual reproduction is absent (only one mating type), the pathogen is essentially an obligate parasite and cannot survive for long periods in the absence of a susceptible host. In locations where sexual reproduction is possible, oospores may survive from one season to the next, regardless of host presence. Although sporangia can survive for days or weeks in moist soil, they do not survive for long periods and, especially, they do not survive drying and freezing. Mycelium in infected viable tubers survives well from one season to the next regardless of storage type. Sporangia produced from lesions on infected plants can be dispersed on wind currents or splashed in raindrops to neighboring plants and the secondary cycles are possible. Note that both foliage and tubers are susceptible to infection.

Pathogen dispersal

Infected seed tubers can be transported kilometers, hundreds of kilometers, or from on continent to another. It is also possible for oospores to be transported long distances. The very rapid

asexual generation time of 5-6 days under optimal conditions with potential for production large numbers of sporangia on potato foliage enables epidemics to develop even from very small levels of initial inoculums. The efficiency of infected tubers initiating epidemics may now be a function of pathogen genotypes as well as a function of host genotype and meteorological conditions.

Aerial dispersal comprises three distinct phases: escape from the canopy, transport through atmosphere and landing on plant tissues. Short-range transport of sporangia can be very important, especially under moist, cloudy conditions. Sporangia (usually produced during a wet period overnight) can be dispersed by wind during drying in the morning for some distance and distances of up to 11 km have been identified. Then they cause infection when the leaves are again wetted (by dew or rain).

Favourable conditions

Potato late blight is a classic polycyclic disease (many generations per season). It is well established that moderate temperatures (15-25^{o}C) and high moisture (leaf wetness from dew, irrigation, fog, mist, rain, etc) or high relative humidity (near 100%) favour epidemics of late blight. The sporangia can germinate well at 18 - 20^{o}C in a film of water. When the temperature is 20-21^{o}C, two to eight zoospores are released from each sporangium within three hours. The zoospores swim about in the water film on the surface of the leaf, lose or withdraw their flagella, and germinate by producing germ tubes. Appressoria form at the tips of elongating germ tubes and the cuticle of the leaf is penetrated directly by penetration pegs growing from appressoria.

When the temperature is 22-32^{o}C, a sporangium in a film of water germinates by producing a single germ tube. Thus, the sporangia function as conidia in warm weather. Apresoria form at the ends of germ tubes from sporangia (conidia), and the fungus penetrates directly through the uninjured cuticle beneath the appressoria. Occasionally, branches from germ tubes derived from sporangia enter the leaf through its stomata. The mycelium develops intercellularly in the diseased leaves of potato and tomato. Fingerlike branches from the intercellular hyphae penetrate cells of the suspect and act as feeding organs. Parasitized host cells die shortly after the tissues have been invaded by the fungus, but symptoms are not visible until two or three days after the onset of infection. The first symptoms are hydrolysis and spot. The typical blighting of diseased

foliage becomes apparent between five and seven days after ingress. Sporangiophores and sporangia, the signs of the fungus, protrude stomata approximately one week after infection has begun.

Sporangia (produced over a wide range of temperature, 3-26 ^{o}C and 91-100%RH) are the inocula for secondary cycles or the fungus. The phenomena of inoculation, prepenetration, penetration, and infection proceed as described. Tubers formed at or just below the surface of the soil are infected late in the season by spores that drop to the soil from diseased leaves.

Survival of *P. infestans*

Airborne sporangia are affected by temperature, relative humidity, and solar radiation. Sporangia can survive longer at high relative humidities that at low humidities and can do better at 15-20 ^{o}C than at 30 ^{o}C. Similarly the pathogen survives longer in moist soil than dry soils. Low pH, aluminium toxicity, low moisture and associated microbes will lessen survival of sporangia in soil.

Disease management

Management strategies are grouped into those that limit or reduce the inoculums and those that retard the pathogen growth. Elimination of infected potatoes reduces the initial pathogen population. Destruction of cull-piles ad volunteers, planting healthy seed tubers, and seed tuber treatment with fungicides are important components of a comprehensive management system. Utilization of resistant cultivars with field resistance and periodic application of protectant or effective systemic fungicide, E.g. metalaxyl (ridomil) is also recommended.

6.2. Diseases of carrot (*Daucuscarota*)

6.2.1. Carrot root knot (*Meloidogyne incognita*)

The nematode attacks many host crops including carrot, radish, turnip, cucurbits, potato, tomato, egg-plants, chillies, groundnut, okra, pineapple, pyrethrum, and many other crops.

Disease symptoms

The nematode infestation on the root appears as tiny tubercles but heavy and heavy localized infestations induce excessive cell division leading to gall formation. Affected plants become stunted with chlorotic appearance. They usually appear in patches within the fields. Location of these patches is generally the same from one season to the next.

Causal agent

The nematode *Meloidogyne incognita* is the causal agent. The nematode survives in the soil on crop debris. The life cycle consists of an egg, four larval stages, and an adult stage. Second stage larvae usually enter the root just behind the root-tips.

Disease control

Field fumigation with nemagon, nematox has been recommended as the effective means of control. Crop rotation, ploughing with wild marigold and use of resistant varieties are most useful.

6.3. DISEASE OF ONIONS (*Allium cepa* L.)

6.3.1. Onion downy mildew (*Peronospora destructor*)

Significance and distribution

Down mildew of onion occurs widely distributed in relatively cool and humid areas of the world especially in wet, low-lying and badly drained areas. It is known to be seed-borne disease. The hyphae also grow down into the leaf bases and may remain dormant in the bulb, from which infection may be initiated in the field. Oospores may remain viable in soil for years and constitute the main primary source of inoculum. The infected leaves die and bulbs fail to develop in severely attacked plants.

Disease symptoms

The disease symptoms first appear as pale yellowish spots on the upper half of the leaves. The fungus sporulates on the spots as bluish-grey, fuzzy mildew when the humidity is high. Spots enlarge andn coalesce each other and the affected leaves wither and die and the growth of the bulb is reduced. Sporangiophores bearing infective sporangia are produced on the lesions and

give a grayish-violet colour to the lesions. Local lesions, resulting from secondary infection are oval to cylindric in shape and often paler in colour. They may consist of alternating chlorotic and green layers of tissues on the leaf and stem.

Causal pathogen

Peronospora destructor is an obligate biotrophicphycomycetous and is the causal fungus of downy mildew of onion. It belongs to the family Peronosporaceae, order peronosporales in the class phycomycetes. The pathogen survives in the form of oospores in the soil and as a dormant mycelium in the bulbs. Infected bulbs (decaying onion debris) and oospores in the soil serve as a source of primary infection. Air dissemination of conidia is the chief means of secondary spread.

Disease control measures

Seed treatment with seed dressing fungicides and spraying with Bordeaux mixture (copper or organic fungicides) has been recommended. The use of disease-free bulbs in well-drained areas reduces the incidence of the disease. Clean cultivation and crop rotation are also advisable. Use of resistant varieties is the best means of control.

6.2.2. Onion purple blotch (*Alternariaporri*)

Significance and distribution

Purple blotch is a widespread disease on *Alliums* in the world but is most severe on onions grown in hot, humid climates. The fungus is a necrotroph asexual conidia-producing state of ascomycetes.

Disease symptom

The first symptoms on leaves are small water-soaked lesions that develop white centres and enlarge to become zonate and brown to purple in colour. The disease is characterized by the appearance of small, white, sunken lesions with purple centre on the leaves. Later the lesions enlarge rapidly, girdling leaf and the bulbs rotting follow in the storage. Severely affected foliage may die back and seed stalks may fail to produce seed heads. Older leaves are more susceptible than younger ones to infection

Causal fungus

Alternariaporri is the causal fungus and belongs to the family Dematicaceae in the order Moniliales and class Deuteromycetes.

Disease epidemiology

Alternariaporri is active over a higher temperature range, with an optimum of 25 ^{o}C. Conidia are formed at night on leaf lesions at relative humidities 90% or more and are fully mature after 15 hour of dew. Sporulation occurs at night at high relative humidities, and conidia are released between 0800 and 1400 hours as humidities decrease on calm days. The concentration of conidia in the air increases on windy days and also after rainfall, irrigation or spraying.

With this disease, the duration of conditions favourable for infection and subsequent RH is important in determining the type of lesion that is produced. Thus, the conidia formed under dew durations of 12 hour or less and low RH cause sterile leaf flecks, while those formed under dew conditions lasting for 16 hours or more with high RH cause typical lesions. Flecks may be caused by immature conidia that are not sufficiently aggressive to infect. About 96% of normal conidia can germinate at 25 ^{o}C within 24 hours of inoculation and that penetration of leaf tissue is direct or the through stomata.

6.3. Garlic Diseases

Garlic rust caused by *Pucciniaallii* is potentially damaging to garlic crops, has a wide distribution and limits production of the crop in many countries. Different disease management options are being used to reduce its effect. Cultural disease management methods such as rotation with non-host crops, optimum crop density, adequate supply of moisture, use of healthy planting materials and field sanitation with other crops can reduce the intensity of garlic rust by reducing available inoculum to initiate infection. Use of host plant resistance to manage diseases reported as it is economical, long-lasting, effective, easy to handle and environment-friendly. There are several fungicides which are effective against garlic rust, as a stop-gap measure or as an integral part of the crop management system. Integrated management of garlic rust is possible that a combination of control tactics is applied in many parts of the world. Such an integrated

approach does not depend on a single method, which in the case of monogenic host resistance could be non-durable, and should be more sustainable over time.

Symptoms

*Pucciniaallii*infects garlic at bulb formation stage. Garlic rust is readily identified by the earliest symptom of small, circular to elongate white flecks that occur on both sides of leaves, as the disease progresses, these small spots expand, and the leaf tissue covering the lesions ruptures and masses of orange, powdery spores (uredospores) become visible as pustules.

Management Options

Diseases caused by *Pucciniaallii* are controlled primarily through the use of chemical sprays with fungicides, and disease free or treated seeds, adequate nitrogen fertilizer were generally reduces the rate of infection by *Pucciniaallii*. Crop rotation, removal and burning of crop debris (if infected), and eradication of weed hosts help in reducing the inoculum potential for subsequent plantings of susceptible crops.

Host Plant Ranges and Pathogen Survival

Pucciniaallii causes rust diseases in onion (*Allium cepa* L.), scallion (*A. chinense*L.) spring onion or Japanese bunching onion (*A. fistulosum* L., porrumgroup: naganegi), garlic (*A. sativum* L. nin-niku), and Chinese chive (*A. tuberosum* L.) in Japan.

White rot of Garlic

White rot is a serious disease of garlic and other *Allium species including onion.* The disease is caused by soil born fungus *Sclerotiumcepivorum*Berk. It was first described by Berkerley in England in 1841. It is the most important, widespread and destructive fungal disease of garlic, onions and leek. Once introduced in the field, the disease can quickly spread within and between paddocks and can cause complete crop loss. The pathogen is particularly difficult to manage, as it can remain dormant in the soil for many years until the next crop is planted. White rot is a serious disease of garlic and other *Allium species.* The disease is caused by soil born fungus *Sclerotiumcepivorum*Berk. It was first described by Berkerley in England in 1841. It is the most important, widespread and destructive fungal disease o f garlic, onions and leek. Once introduced in the field, the disease can quickly spread within and between paddocks and can cause complete crop. The pathogen is particularly difficult to manage, as it can remain dormant in the soil for many years until the next crop is planted.

Management Options

White rot are controlled mainly by the use of chemical sprays with fungicides, and disease free or treated seeds, adequate nitrogen fertilizer were generally reduces the rate of infection. Crop rotation, removal and burning of crop debris (if infected) *by Sclerotiumcepivorum*, and eradication of weed hosts help in reducing the inoculum potential for succeeding plantings of vulnerable crops.

CHAPTER SEVEN

MAJOR DISEASE AND INSECT PESTS OF FRUIT AND PLANTATION CROPS

7.1. DISEASES OF BANANA (*Musa paradisiacal* subsp. *sapientum*Kunze)

7.1.1. Banana fusarium wilt or Panama disease (*Fusariumoxysporum*)

Disease significance and distribution

The disease was first recorded from Panama in 1890 and its presence has since been recorded from all over the world wherever banana is grown. It occurs in Central and South America, parts of Africa (Tanzania, Kenya, Uganda, etc.), Siri Lanka, Burma, Thailand, Malaysia, Indonesia, Hawai, Fiji, Australia, New Zealand, Phillipines, and India. The disease results in wilting of the banana, stunting of growth and withering of leaves and fruit ripening prematurely. Gros Michael is a very susceptible banana variety to the disease

Disease symptoms

Infection may take at any stage in very young suckers and growing adult plants through most apparent on plants at least 5 months old and 2-3 months old plants are also killed under highly favourable conditions for disease development.

The first symptom of the disease is faint yellow streak in the petiole of the oldest and lowest leaf. Two types of symptoms follow this stage:

1. In the yellowing type is progressive yellowing of the old leaves and eventually collapse at the petiole.
2. In the non-yellowing type, the leaf collapses at the petiole without leaf chlorosis.Often all the leaves but the youngest collapse, the heart alone remaining upright. Any new

leaves that are produced are blotchy and yellow, often with wrinkling of lamina. The pseudostem often shows a more or less conspicuous longitudinal splitting of the outer leaf sheaths that form the outer covering of the pseudostem. About 4-6 weeks after appearance of the streak on the petiole only dead trunk of trunk of the pseudostem remains.

Discoloured vascular strands varying from light-yellow to dark-brown are the distinguishing internal symptoms. Usually the discoloration appears in the outer or odestleafsheath and extends up to the pseudostem. Vascular discoloration is pronounced in the rhizome but is not common in the roots. However, roots of diseased rhizomes are frequently blackened and decayed. Longitudinal sections through diseased root-bases show characteristic red strands passing into the rhizome stele.

In summary, banana fusarium wilt first begins with conspicuous yellowing of the bottom leaves. Discoloration starts at the margin and spreads toward the midrib. Discolored leaves undergo rapid wilting followed by sucking of petioles with characteristic leaf drooping, withering and browning. Subsequently leaves are subject to a similar development until only the topmost leaf is left standing erect while green in color. The withered leaves form around the pseudostem of the plant. The top leaf eventually withers while the dead plant remains upright until it is blown over by wind.

Heavily infected plant becomes stunted in growth and develops abnormal bunches and fruits ripen prematurely. The fingers become bottlenecked and ripe irregularly and too rapidly while the pulp become pithy, yellow and acidic in taste. When the affected plants cut open show yellow, red or brown discoloration of vascular strands. This discoloration eventually turns purple to black. Diseased rhizomes possess a characteristic smell that becomes unpleasant as a result of putrification to the secondary rots. Roots of the disease plants often become black and start decay.

Causal pathogen

The causal pathogen is *Fusariumoxysporum*f.sp. *cubense*. The mycelium is mainly intercellular, being typically found in the vascular vessels, though intercellular hyphae may be observed in the cortex of roots and in parenchymatous tissue in proximity to the infection court. The sporodochia

(conidia-bearing bodies) appear at a late stage on the surface of the petioles and leaves of infected plants. The conidiophores are verticilliately branched and measure 70 μm in length. Conidia are borne at the apical end of main lateral branches. Microconidia are 0- and 1- septate, ovate or elongate and measure 5-7 x 2.5-3μm. macroconidia are sickle-shaped, pedicellate, mostly 3-septate and measure 22-36 x 4.4 μm. chlamydospores are oval or spherical and usually in pair. There are different physiological races.

Disease cycle

The fungus is a soil-inhabiting pathogen surviving in the soil mainly as chlamydospores formed by the hyphal and conidial cells. The pathogen penetrates the plant directly through roots and indirectly through the damaged rhizomes or through injured roots, e.g., nematodes. The fungus then produces mycelia that move upwards into the psedomstem and leaves through vascular bundles, resulting in wilting due mainly to toxic substances. Secondary spread is by means of wind-blown conidia, irrigation water or movement of infested soil by surface flood-water. The chief way of transmission is the movement of infected rhizomes or suckers to new places.

Disease epidemiology

Depth of soil penetration, survival and dispersal of the pathogen depends on soil conditions. Clay soils retard the spread and penetration is more in sandy soils. So light-textured of 30% is favourable for the pathogen.

Control measures

1) Eradication of diseased plants and quarantine
2) Use of disease free suckers
3) Crop rotation
4) Improvement of soil conditions by flooding the soil for six months or more to reduce inoculums by suffocation
5) Breeding and cultivation of resistant varieties
6) Use of antagonistic organisms or biological control e.g. actinomycetes and soil-bacteria capable of producing antibiotics such as musarin and monamycin

7) Applications of sodium nitrate and mercuric salts to the soil check fungal growth on small scale farming.

7.1.2. Enset bacterial wilt and Banana (*Xanthomonascampestrismusacearum* D. Yirgou& J. F. Bradbury)

Significance and distribution

This disease was first reported and described by DagnachewYirgou and Bradbury in 1986 who attributed it to *Xanthomonascapestrismusacearum*. Known to occur only in Ethiopia, the bacterium was later renamed according to accepted nomenclature, as *Xanthomonascapestris*pv. *musacearum* (Yirgou& Bradbury) Dye in 1980. It belongs to the Schizomycetes in the order Pseudomonadales and family Pseudomonadaceae. Ensete bacterial wilt is widespread having been observed in ensete plantation at 2650-2950 m altitudes. In a survey in the early 1980s involving 29 weredas (administrative districts) in the main ensete growing regions, the disease was severe in 23 weredas (Ashagari, 1985). This serious ensete wilt is encountered in Gurage and Wolayta districts. The bacterium has been reported to be pathogenic on hot pepper, tobacco, sesame, and *Daturastramonium* in inoculation tests. However, it has limited host range.

The disease also affects banana growing side by side with ensete in Sidama, and North Omo zones unde natural conditions. Even, a natural epidemic of the disease was reported on the banana in Kaffa province in 1974 Ensete bacterial wilt is very destructive as it kills ensete plants at all growth stage, including 4-7 years old plants ready for harvest. Devastated fields are sometimes abandoned and replaced with other crops. Other times, farmers are forced to rotate infested fields with other plants such as maize for at least two cropping seasons.

Disease symptoms

The disease produces symptoms similar to those produced by Moko disease of banana caused by *Pseudomonas solanacearum*. However, enset wilt is caused by *Xanthomonascampestris*pv. *musacearum.* The first symptom of the disease is wilting of the heartleaf or one of the inner leaves. The wilting progresses down the leaf until the leaf breaks at the petiole. One by one all the leaves on a plant wilt, break at the petiole and wither. A cut made through the petiole reveals browning of the vascular strands. Yellowish or grayish droplets of bacterial cells ooze out from

the cut ends of the vascular strands. Such droplets are also seen when sections are made through the pseudostem and the rhizome. The brown discoloration of the vascular strands is also evident in cross sections of the pseudostem and rhizome. The plant eventually dies and rots down to the ground.

Methods of transmission

The various methods of disease transmission include: mechanical means; implements used in ensete cultivation; cutting knives; animals (including insect vectors) and human beings; young shoots that may acquire the disease from infected rhizomes used for propagation. Mechanical transmission of the bacterium and the importance of wounds in disease initiation were demonstrated by cutting ensete leaves with contaminated knives and inoculation injured plants with suspensions of the bacterium. Transmission may occur through contaminated soil, but it is not known how long the bacterium may survive in the soil. Variation in resistance against the bacterium has been observed among ensete cultivars.

Ensete bacterial with control

The use of resistant clones offers the best approach to bacterial wilt control. It is estimated that there exist over 200 different ensete clones in Ethiopia. Sanitary measures that would prevent, reduce or eliminate the spread of the bacterium in the field include: (1) flaming the ensete cutting tools after using them on infected plants; (2) preventing animals from browsing and straying into infested plots (livestock should be kept out of the ensete grove by fencing the land); (3) fencing in the infested site after digging out the corm and roots (the land should be dug up 4-5 times after uprooting of diseased plants during the dry season); (4) knives should be sterilized by flaming or by dipping in a disinfectant such as formaldehyde or phenol before and after use; and (5) chopping them up together with the pseudostem and leaves. Soil and animal manure should be added to the pile to enhance the rotting of the plant materials. The infected plants may also be dug out and disposed off far from the ensete field; contact with diseased plant should be avoided; diseased plants should be chopped up and soil and animal dung should be added into the pile. All cutting and digging tools should be flamed. A local quarantine should be effected; if possible the infected site should be fenced in; movement of soil from infested site to uninfested site should be restricted.

Cultural practices which would reduce *XanthomonascampestrisPv. Musacearuminocula*in the field include: (1) deep tillage and turning over the soil to expose it under the sun, during the dry period prior to planting; (2) manuring the soils of planting holes; (3) spot rotations of infested sites in the field with non-host or non-reservoir plants of *X. campestris*pv. *musacearum* (e.g. fallow planting crops such as sorghum and maize for two years); and (4) replacing wilt susceptible ensete cultivars with resistant ones.

In summary, bacterial wilt may be effectively managed in the subsistence ensete farming system by integrating the use of clones with low levels of resistance (if clones with high levels of resistance are not available) with early detection, prompt destruction, and proper disposal of infected plants and sanitary and cultural management practices that would prevent, reduce or eliminate the spread of the bacterial inocula in the field. Do not allow the growth and distribution of *Canna orchoides* around ensete plantation since it an alternative host for the pathogen.

7.2.2. Sigatoka disease (*Mycosphaerellamusicola*Leach, anamorph: *Cercosporamusae*)

Significance and distribution

It first occurred in the Sigatoka Valley of Fiji Island in 1913 and destroyed almost all banana plantations, hence its name is sigatoka disease.

Disease symptoms

The symptoms first appear on the leaves as light-yellow spots. These spots enlarge and become oval and colour changes to dark brwon. The centre of these spots die, turning light-grey and surrounded by brown ring. The ring is also surrounded by a bright-yellow halo. On severe infection, spots coalesce, killing large portion of the leaf until the majority of the leaves are dried and scorched brown. Other characteristic symptoms include the formation of streaks on the petioles and lines of brown spots with a grayish centre running parallel to the midrib and a concentration of spots on the leaves causing leaves to die.

Causal pathogen

Mycosphaerellamusicola Leach is the causal fungus. The pathogen survives on the banana foliage in the soil in form of perithecia that liberate ascospores later. The fungus produces two

types of spores i.e. conidia and ascospores. Under favourable conditions each kind of spore produces its own typical symptoms. Conidia on conidiospores are water-borne and spread through the banana plantation during humid and windy weather. The ascospores are wind-borne and mainly infect expanded leaves.

The pertithecia are ostiolate, brownish black; asci eight-spored; ascospores 1-septate, hyaline, 20 -80 x 2-6µm. spermagonia also occur. Ascospores are well adapted for survival in the absence of rainfall in lower canopy of banana leaves; infectivity of ascospores dependent on expulsion from the perithecia into moving air and deposition on susceptible host surfaces.

Disease control methods

1) Spraying with medium viscosity refined mineral oils at 100-110 litres per hectare.
2) Spraying with the systemic fungicide calixin.

7.2. Disease of papaya (*Carica papaya* L.)

7.2.1. Powdery mildew of papaya (*Ovulariopsis papaya*Van der Biji)

Significance and distribution

The disease is found on the leaves and undoubtedly it does serious damage on the crop.

Disease symptoms

The disease symptoms appear in the form of large, irregular shaped, yellow blotches on the upper side of the leaves. On the lower side of the infected leaves develop a white ashy growth which later turns grey to black. In severe cases, the infected areas dry up and turn brown and whole the leaf curls upwards and dies.

Causal pathogen

Ovulariopsis papaya is the causal the causal agent. The fungus survives on the crop itself in the form of mycelium. Secondary infection is by means of wind-borne conidia. Warm and humid weather favour disease development.

Disease control

Spraying with karathane can control this disease.

7.3. Disease of mango (*Mangiferaindica* L.) and avocado (*Persea Americana* Mill.)

7.3.1. Mango Anthracnose (*Colletotrichumgloeosporioides*Penz.)

Significance and distribution

This is the most common and widespread fungus disease of mango. It occurs in Brazil, France, the Philippines, Indonesia, Trinidad, Peru, USA, Portugal, India and Hawaii. The disease attacks young as well as mature fruits. If early infection of fruits takes place they fall off. Mature fruits are blemished hence fetch low price in the market.

Disease symptoms

Anthracnose of ripening fruits is characterized by the development of black spots of various forms which may be slightly sunken or may show surface cracks. These spots may coalesce to form larger spots and ultimately the whole fruit may be involved. The spots are often concentric at the stem end, and sometimes in streaks towards one side of the fruit. This suggests that the disease has spread through spores washed down by rain-water from the stem end.

The leaf and twig anthracnose is characterized by leaf spots, wither tip and blossom blight. Numerous oval or irregular spots may appear on the leaf surface. Under humid conditions these spots increase rapidly and form irregular necrotic areas. Young leaves are most susceptible to this infection. Symptoms of wither tip or die-back appear at the tip of very young branches. Black necrotic areas are formed on the affected twigs that dry from the tip downwards, accompanied by defoliation of the branch. In blossom blight the inflorescence shows minute black spots on the flowers which dry and shed.

The causal organism

*Colletotrichumgloeosporioides*Penz., is a widely distributed fungus causing leaf spot and anthracnose on citrus, papaya, avocado, and etc.

The mycelium consists of rather narrow, sparsely separate hyphae that are at first hyaline but later take on a slightly dark colour. These develop at first as tangled sub-epidermal masses of hyphae from which arise the epidermis. From the apex of each conidiophore are developed one or conidia. Setae are common on twigs but not on fruits. The conidia remain embedded in a viscid fluid that swells in moist conditions, ruptures the epidermis and exposes the conidial mass to be disseminated by raindrop splashed and insects. The conidia in mass are pinkish but hyaline individually. They are broadly oval to oblong, with rounded ends, non- septate, and sometimes contain 1-2 globules. Their average size is about 12-16 x4-6 μm.

Diseased twigs and leaves which fall on the ground are a prolific source of perennation and fresh infection. The fungus has a long saprophytic survival ability on dead twigs. On the fruits most of the infection takes place from the start of the blossoming period until the fruits are more than half grown. The fungus enters the pores of the fruit while it is still green and develops in the flesh during ripening. Latent infection of mature fruits has also been reported to take through lenticels.

Humid and misty conditions are considered most favourable for infection. The fungus does not grow at relative humidities below 95%. An isolate of *C. gloeosporioides* from citrus is known to produce toxin (s) in liquid culture that may be responsible for die-back symptoms.

Control measures

Plant vigour keeps the infection of twigs away. Proper fertilization and watering during dry season must be done to maintain tree vigour. Tree sanitation is also important. Diseased twigs should be pruned and burnt along with fallen leaves. Pruning should be followed by spraying with suitable fungicides such as 6:6:100 Bordeaux mixture, 0.15% Cuprocide, Fytolan or Blitox-50, etc. Four to five sprayings give satisfactory control of the disease. Combination of captan with zined for spraying the trees has been found very effective against this disease. When treating mango tree with zined or Bordeaxu mixture, two sprayings should be given at the time of flowering and subsequent sprayings may be started just before the onset of the rainy season and continued until harvest at an interval of 14 days. For control of fruit rot, post –harvest fruit treatment in hot water. Fruits may be dipped in hot water at 50^0C for 15 minutes. Hare chemical treatments are not effective.

7.3.2. Powdery mildew of mango (*Uncinulanecator* (Schw.) Burr.)

Significance and distribution

Powdery mildew of grapevine occurs in mild or severe form in Americas, Europe, India, parts of Africa, and Australia. French wine industry suffered huge losses due to epidemics of this disease during 1850-1855. The disease is much more serious than the downy mildew of the same host and is more dangerous than any other powdery mildews of different crops.

Disease symptoms

The disease attacks the vines at any stage of their growth. As in other powdery mildews, the characteristic symptom of the disease is the appearance of white, powdery patches on affected parts. All aerial parts including fruits are attacked. On young leaves small whitish patches appear on the upper, or sometimes on the lower, surface of the leaf. These patches grow in size and finally coalesce to cover large areas on the lamina. Similar floury patches are formed on the stem, tendril, flowers, and young fruit bunches. The powdery growth gradually turns grey and finally dark colour. Malformation and discoloration of the affected leaves are common symptoms. Diseased vines have a wilted appearance and remain dwarf. The stems turn brown in colour. If the attached fruits are nearing maturity they become irregular in form and only few of them ripen, though remaining undersized. If the disease has appeared early, the fruits may not develop at all. Necrosis of the penetrated epidermal cells and even of adjacent cells is a characteristic reaction of resistance in host.

The causal pathogen

Powdery mildew of grapevine is caused by the fungus *Uncinulanecator* (Schw.) Burr. (syn. *Oidiumtuckeri*). The mycelium is entirely superficial on the surface of parts attacked to which it adheres by means of appressoria. The hyphae are very slender, and septate, turning darker when formation of conidia is over. The conidiophores arising from the mycelium are simple and erect. They bear a chain of 3-4 conidia. The conidia are oval in shape and measure 25-30 x 15-17 μm. They are resistant to desiccation.

The cleistothecia are found embedded in the superficial mycelium on the leaves, or on shoots, chiefly at the nodes or on buds among the scales and hairs. They are black when fully mature,

almost round with a flattened top, and measure 75- 100 μm in diameter. The peridium is covered with 8-25 septtate appendages that are coiled at the distal end. Each fruit contains 2-8 ovoid asci measuring 48-60 x 37-45 μm. In each ascus there are four to six ascospores that are oval in shape and measure 20 x 31 μm.

Disease cycle

In localities where cleistothecia are formed they are believed to carry the fungus from season to season. Environmental factors are very much responsible for their formation. It has been reported that conducive climatic conditions are decisive for the development of the disease. Warm, dry weather with just enough humidity is very favourable. The disease development is retarded in sunshine.

Control measures

Pruning after shedding of the leaves, thinning out and cutting back of laterals, removal and destruction of all diseased parts are important parts of clean cultivation. Dusting vines with sulphur (300 mesh) gives effective control. The first dusting should be done when new shoots are 3-6 inches long, second dusting during or just before blossoming. A third application can be made 40-50 days later.

7.3.2. Avocado Root rot

- *Two groups of causal agents account for most of the damage done to avocado trees by root rots.*
- *The main group includes various species of Phytophthora, of which P.cinamomi is the most widespread.*
- *Phytophthora root root- caused byPhythophthora spp. especially P.cinnamomi identified as the most serious cause of losses in avocado orchards worldwide.*
- ***Present in nearly all the areas where avocado isgrowing.***
- *No cultivar yet has been found to confer total resistance- control is therefore costly and difficult.*

Epidemiology / disease development

- Soil temperature has a considerable influence on the development of the disease, and on its seriousness.
- ***At 15^0c the disease does little damage, butdoses become very high at temperatures of 27 to 30^0c.***
- Severe root infections- with in a soil PH range of 4.5-7.5. Reducing the PH to 3.5 strongly curtails infection, but this level also inhibits tree growth.
- Soil moisture directly affects the severity of root attacks.
- The disease invariably develops faster and is more serious in poorly drained, shallow soils with a high water- holding capacity.
- Poor irrigation management can also aggravate the disease.
- P. cinnamomi primarily attacks vigorous roots with out lesions.

Note***: In avocado unlike in other fruit species, affected roots have little chance of subsequently recovering.***

Symptoms

- Of two kinds based on plant parts infected

Root infection

- Leaves of affected trees small and yellowish green, lacking in vigor
- Frequently fall from the tree and not replaced
- The whole canopy appears sparse, and the ends of branches are bare and desiccated
- The tree often bears a large number of fruits but of small size.
- Most main and intermediate roots are dark brown in color. Much of the root tissue is rotten and crumbles between the fingers.
- In the final stages of the disease almost no new living roots can be found and the tree is unable to feed itself
- The tree dies out and collapses appealing to die of thirst while the soil around it remains most.

Control / management of the disease

- The use of disease free plants in the nursery

Harvest from the tree the fruits being used to supply seed, not to pick them up from the ground.

- Careful selection of orchard site - consider any factors favoring the disease development
- The use of resistant root stocks.
- Chemicals

CHAPTER EIGHT
STORED GRAIN DISEASES AND INSECT PESTS

Fungi and bacteria responsible for in-storage decay often originate in the field. When penetration into the host takes place in the field, the pathogen, which is then in its early or quiescent stages of infection, will get to the storeroom within the host tissue without eliciting any symptoms of decay. Yet, even when preharvest infection has not taken place, there are always fungal spores and bacterial cells, which are typical components of the airborne microorganism population, on the fruit and the vegetable during their growth. This cargo of spores and cells is transferred to storage with the harvested crops. Examination of the fungal spore population on the surface of stems, leaves, flower parts, crops and other plant organs, after harvest, reveals the presence of many important airborne fungi such as species of *Cladosporium, Alternaria, Stemphylium, Penicillium, Aspergillus,Rhizopus, Mucor, Botrytis, Fusarium*and others.

Many of the airborne fungi are among the most important decay agents which affect harvested crops and, given the right conditions, develop and cause decay. Many postharvest pathogens perpetuate on crop debris in the field and can, under suitable conditions, develop and produce abundant new spores. These fungal spores are easily carried by air currents, winds and rain, or dispersed by insects, to the flowers and the young crops, at various stages of development, and form a potential source of infection.

Soil, irrigation water and plant debris form an important source of infection of various vegetables. Soil-residing fungi and bacteria can attack the bulb, tuber, root and other vegetal parts, while these are still attached to the parent plant, through a tight contact with the soil, by lifting of soil particles by winds, rains or irrigation, through growth, or by arriving in storage with soil residues attached to the vegetable. Some soil microorganisms, such as species of the fungi *Botrytis, Sclerotinia*and *Fusarium*or the bacterium *Erwiniacarotovora,* are among the main decay agents in stored vegetables. Host infection can occurpreharvest, during harvest or during

any of the postharvest handling stages. Harvesting instruments, containers, packing houses with their installations, the hands of the packers or selectors, the atmosphere of the storage rooms - are all bountiful sources of fungal spores.

Despite the diversity of microorganisms that the crops carry with them into storage, only a few species can naturally attack them, while other species that reside on the surface, at times in large quantities, will not penetrate and will not cause any decay. The development of disease during storage depends, primarily, on the existence of the appropriate microorganisms alongside a given host. However, in order for the fungal spores or bacterial cells that have reached the suitable host to be capable of infecting, they have to encounter the appropriate conditions for germination on the surface of the host, to penetrate into the host tissues and to develop there.

FACTORS AFFECTING DISEASE DEVELOPMENT

A. Preharvest factors, harvesting and handling
B. Inoculum level
C. Storage conditions
D. Host-pathogen interactions

Management options

1. Preharvest chemical treatments
2. Modified and controlled atmospheres
3. Calcium application
4. Postharvest chemical treatment
5. Heat treatments
6. Biological control

SOME MAJOR INSECT PESTS OF CEREAL CROPS, VEGETABLES, FRUITS, INDUSTRIAL CROPS AND THEIR DISTRIBUTION, NATURE OF DAMAGE, LIFE CYCLE AND THEIR MANAGEMENT OPTIONS

1. African Army worm (*Spodoptera exempta)*

- Order—Lepidoptera
- family—Noctuidae

1.1 <u>Host plants</u>:- The most important cereal crops attacked by army worm in Ethiopia include, Maize, Sorghum, Millet, wheat, Teff, Barley, Rice and as alternate hosts different grasses and sugar cane. The range of host plants is restricted to cereals and different grasses (including pasture and wild grasses).

1.3 Life cycle / Biology and Recognition: – the army worm has four stages of development with complete metamorphosis. These are the adult moth, the egg, larva and pupa. The life cycle of armyworm depending on environmental conditions might take 2-5 days for egg, 11-24 days for larva, 7-12 days for pupa and 2-13 days for moth (pre-oviposition period). Generally the life cycle take between 22 and 54 days with an average of 37 days.

Egg:- the eggs are laid in groups of 100-400. A female lays several egg masses until a total of up to more than 1000 eggs. The eggs are laid in single or multi layers on the leaves. They are covered with black hair scales. The individual eggs are conical, with slightly rounded apex. Each egg is about 0.5 mm in diameter and is yellowish (or pale yellow) in color when newly laid. As the eggs develop, they darken until just before hatch. Hatching takes place, after 2-5 days.

Larva :- Translucent larvae, which have black head capsules, feed on their egg shell for about one hour before spinning silken threads on which they are dispersed by the wind. There are six larval instars. On hatching the larvae are colorless or whitish. They have black heads. As they feed the colour becomes green. During the first three instars the caterpillars remain green. After this they turn black when there are many caterpillars together (gregarious form) or they will remain in various shades of green or brown when there are only few caterpillars together (solitary form). In a typical army-worm outbreak the caterpillars are velvety black on top with pale lines on each side. The underside of the body is green or greenish yellow. There is a pale stripe along the top of the body. The caterpillars do not have obvious hairs. The head is shiny black and shows a v-shaped white mark. The upper side of the first segment behind the head is black and has three narrow white stripes. A fully-grown Caterpillar is 25-35mm (aver .30mm) long. The larval stages take 11-24 days.

Pupa: -During pupation if there is rain (the soil is dump or moist) the mature caterpillars burrow in to the soil (under ground) and pupate in constructed silk lined chambers 2-3 cm below the surface. The pupa is deep red brown or brown or black in colour. It is about 17mm long. The pupal stage may last 7-12 days depending on the environment.

Adult (moth) :– the adult is a grey-brown night-flying moth. Both sexes have white hind wings with dark veins running through them. The fore wings show an over all dull grey-brown appearance. They have two spots. The inner (orbicular) spot is elongate and pale. The outer (reniform) spot is kidney-shaped and more clearly visible in males than in females. The wing span is 25-35 mm for males and 22-37 mm for females.

1.4 Damage symptoms and pattern – newly hatched larvae are only capable of feeding on the young leaves of their host plant. They do so by rasping the epidermis and the effect of feeding by the young larvae is skeletonization of the leaf of the food plant, making a pattern on the leaves descriptively termed << Windowing >>. The older stages of the caterpillars devour the leaves of their hosts completely or leave only the midrib.

1.5. Outbreaks and Migration :- Outbreak describes the sudden appearance and the explosive increase in numbers of the armyworm larvae. There are two outbreak conditions. These are:-

- A primary out break which is an outbreak derived from low-density populations and it is believed to be from the low-density populations of solitarious larvae and
- A secondary outbreak which is an out break derives from earlier out break and successive outbreaks.

Primary outbreak most often occur in central Tanzania and costal areas of Kenya and these first outbreak which initiate the onset and spread of subsequent secondary outbreaks through out eastern Africa as far to Yemen. The army worm moths are able to travel hundreds of K.ms down wind during the hours of darkness. Also the generation of army worm produced monthly show a gradual north ward movement following the trend of the long rains.

1.6 Army worm forecasting: – There are two basic types of traps being in use for forecasting army worm moths. These are light traps and pheromone traps. Light traps have been widely in use in the last two decades, but it has several disadvantages. On the other hand pheromone trap is advantageous by:-

- simple for use,
- don't require electricity and
- generally catch only male army worm moths.

1.7 Control/management measures

- **Traditional control**: – Methods practiced by farmers include:-
  - Digging small trenches between the adjacent infested crop field and hitting the larvae with branches, twigs of trees of bushes inside the trenches.
  - Moving herds of cattle on infested grass land (pasture).
  - Collecting larvae and put them in the trench and cover them with soil.
- **Biological Control**
- **Chemical Control**

The following chemicals can be used.

- Malathion (Ethiolathian) 50% EC 2 lit/ha
- " " 95% ULV 1.25-1.5 lit/ha
- Fenitrothion (sumithion) 50% E.C 1.3 lit/ha
- " " 96% ULV 1lit/ha
- Carbaryl (Sevin) 85% Wp 1.2—1.5 Kg/ha
- Cypermethrin 5% E.C or 6% E.C 1.0 lit/ha
- " 2.5% ULV 1.0 lit/ha
- Trichiorophon 50% EC 1.0 lit/ha
- " 80% WP 1.0 Kg/ha

2. **Stalk borer**

There are four stem borers of cereals, sugar cane and other wild hosts recorded in Ethiopia. These include maize stalk borer (*Busseola fusca*), pink stalk borer *(Sesamia calamistis*), spotted stalk borer (*Chilo partellus*) and S*esamia cretica*. From all, maize stalk borer is the most serious pests of cereals in Ethiopia.

2.1. **Maize stalk borer *(Busseola fusca)***

Order—Lepidoptera

Family—Noctuidae

2.1.1. Host plants: - The maize stalk borer is a major pests of maize and sorghum. Most stalk borer alternatively attack other cereals, many species of grasses and sugar cane.

2.13. Life cycle/Biology and morphology

The Adult emerges from pupae in the late afternoon and early evening and are active at night. During the day they rest on plants and plant debris and are seldom seen unless disturbed. During the 3-4 nights following emergence, females lay eggs in batches of 30-100 under the inner surfaces of leaf sheaths, a single moth may lay a total of 200-400 eggs. The larvae hatched about 10-15 days later, the incubation period varying according to temperature and relative humidity (the increment of both, decrease the incubation period). Hatched larvae initially disperse over plants before they enter the leaf whorls and start to feed on leaves. Once established in their host plants, they bore into stem of the plant and cause dead hearts. The larvae feed inside the stem until it gets mature. The whole larval period may lasts in 35 to 60 days. They then pupate in the

tunnels, often after excavating emergence windows to facilitate the exit of adult moths. The adults emerge 15-20 days after pupation. During dry and/or cold weather, larvae enter a diapause of 6 months or more in stems, stubble, and other plant residues before pupating during the next favorable period.

Egg – The eggs are hemispherical or globular and about 1mm in diameter. They are white first, but they turn darker when they get older.

Larvae – The fully grown caterpillars measure 35 to 40 mm in length. Their color varies from light or dark violet to pinkish-white; also young larvae are deep purple or black in color. Along the sides of the body are rows of small black spots. The head is dark brown and the pro-thorax is yellowish-brown.

Pupae- pupae are brown or dark brown or shiny yellow-brown in color, but their colour may varies with location and its length is about 18-25 mm long.

Adult- Adult moths are seldom seen in farmers' fields as they are inactive during day light and are cryptically coloured. The adult wingspan is about 20-40 mm, with females generally larger than male. The forewings are reddish brown with dark markings, the hind wings whitish to greyish-brown. There is much seasonal and geographic variation; moths developing in colder, wetter conditions tend to be darker in color.

2.1.4 **Damage symptoms**- the newly hatched caterpillars feed on tender leaves of the host plants initially by scrapping in leaf whorls of growing plants. The fully developed leaves show irregular << pinholes >> (holes) and windows caused by the feeding effect of the larvae. The developed larvae bore into the stems and may kill the central leaves and growing points, producing dead hearts. Also in maize the caterpillars bore in to the maize cobs and feed on the developing grains. Early attacked plants are stunted in growth (have poor growth), reduced yield and are more susceptible to wind damage and secondary infection.

2.1.5 **Control/management measures for stalk borer**

- **Cultural control**
  - ✓ Destruction or appropriate disposal of crop residues (all dried stalks must be removed and burnt after harvest).

- ✓ Early sowing and simultaneous sowing over a large area prevent population build-up.
- ✓ Eradication of the alternative hosts and volunteer crop plant
- ✓ Horizontal laying of maize or sorghum stem on direct sun light to destroy the larvae and can be used for construction.
- ✓ Crop rotation: - rotating maize or sorghum with leguminous or root crops will help to reduce the stem borer population build up.
- ✓ Fertilizer application.
- ✓ Tillage
- ✓ Inter cropping

❖ Host-plant resistance

- ✓ A wide range of mechanisms were involved in stem borer resistance in maize and sorghum, including non-preference for oviposition, reduced feeding, reduced tunneling & tolerance of plants to leaf damages, dead heart and stem tunneling.

❖ **Biological control**

Biological control measures applied by using naturally occurring bio control agents such as parasites, parasitoids, predators and disease, includes

- o Parasitoids such as Trichogramma sp. (egg)
- o Parasites such as Braconid wasp. (as larval parasites)
- o Disease such as Bacillus thuringiensis

❖ **Botanical control**

Using botanical extracts (act as repellent or phagodeterrent or may kill the pest directly) to protect crops from the attacks of pests. This includes.

- o Using neem extractions.
- o Application of cow or cattle dung and urine
- o Application of wood ashes, etc.

❖ **Pheromonal control**

The female sex pheromone can be used for population monitoring and control by mass trapping and mating disruption.

- **Chemical control**

  Chemical control is as last alternatives. If more than 5% of the plants show leaf damage, chemical control may be carried out. Insecticides, which have been found effective as sprays or granules or dusts are carbofuran, Diazinon, Carbaryl, Fenitrothion, Endosulfan 35% EC, synthetic Pyrethroids, Cypermethrin 10% granules, Endosulfan 1% or 3% granule and Endosulfan 4% dust.

- **IPM**

When the above single control option is insufficient, one may try to exploit the interaction of different control strategies in a pest management system.

3. <u>African boll worm</u> *(Helicoverpa armigera)*

Order—Lepidoptera

Family—Noctuide

Species—*Helicoverpa armigera (= Heliothis armigera)*

3.1. <u>Host plants</u> – In Ethiopia it attacks sorghum, maize, cotton, Beans, Tomato, Tobacco, Many legumes, some vegetables and other plants.

3.3. **<u>Life-cycle/Biology and morphology</u>**

African boll worm posses four stage of development (complete metamorphosis). This includes, egg, larvae, pupa and adult.

<u>Egg:</u> - eggs are laid singly on young buds, stocks, leaves, flowers or fruits. In the case of maize this coincides with the flowering period. Each female moth may lay 1000-1500 eggs. Eggs are spherical, 0.5 mm in diameter. They are first yellow, later turning brown. The egg stage takes 2-5 days according to temperature.

<u>Larva</u>: - The colour of the larva is varies from black to green, brown, reddish-brown, whitish and orange. The fully-grown caterpillars are up to 40mm (4 cm) in length and the body has typical weavy and pale longitudinal strips on yellow to dark-green background. There are six larval instars. The total larval period is 14-24 days, but up to 50 days at low temperatures. After 14-30 days the larvae made their way to the ground where they pupate.

<u>Pupae</u> :-pupation takes place in the soil. The shiny brown pupa is about 16 mm long. The pupal period (development) takes 10 to 14 days or more according to the temperature.

<u>Adult</u>:-The adult brown nocturnal moth has a wing span of 32-40mm and 16-18mm body length. The forewings are yellowish brown or greyish to brown, with a broad slightly darker band and a

small dark spot. The hind wings are pale with a broad dark grey or brown marginal band with two lighter spots on it.

The complete life cycle may /can be as short as about 28 days and there are several generations each season.

3.4. **Damage symptoms and Pattern**

–African boll worm caterpillars are extremely polyphagous and cause heavy damage to flower buds and bolls. They bore in to the bolls often with the hind part of the body exposed outside the boll. In sorghum the caterpillars feed on the head when the grains are in the milky stage. In maize the developing cobs are attacked. In other food crops and pulses they bore in to suitable part of the fruit or pods. Its damage is recognized by clean circular holes, which are bored in flower buds, bolls, fruits and pods. If younger bolls / fruits/ pods are attacked they normally show a yellowish colour and the bracteoles opened out. Excreta may be seen on the plant or under it. The infested bolls and buds drop pre-maturely. Also secondary fungus infection often occurs after an infestation of the bollworm. One caterpillar can damage a number of bolls and buds or pods (2-3 bolls or fruits or pods/night) by moving from one to the other. Generally African bollworm can be considered as the most important pests in Ethiopia.

3.5. Management and control of African Bollworm.

Monitoring-

- The host plants should be inspected regularly for young caterpillars for apply chemical control.
- In the case of maize and sorghum, particularly when they are in a milk ripe condition or stage.

Cultural Control

- Crop rotation
- Early planting
- Intercrop with crops that host beneficial insects in strips or plant catch or trap border stripes

  Eg- sun flower
- Destroy alternate hosts/weeds
- All crop residues must be burnt after harvest.
- For sorghum the use of varieties with open panicles is recommended.

- Ploughing the field will exterminate the pupae in the ground.

Chemical control

Chemical controls are used as a last option (alternative) for chemical control, apply insecticides or spraying at red egg stage or newly hatched larvae

(When caterpillars are small).Some times, since *Helicoverpa armigera* is known for showing resistance to many insecticides, mixtures of insecticides with different mode of action are used.

An insecticide used to control ABW includes.

- Cypermethrin 25% Ec 1.5 lit/ha
- Endosulfan 35% Ec 2 lit/ha
- Sumithion 50% Ec 1.5-2 lit/ha
- Diazinon 60% Ec 2 lit/ha
- Carbaryl (sevin) 85% Wp 1.5 Kg/ha
- Cypermethrin (cymbush) 5% Ec 1.68 lit/ha

4. Grass hopper

Order-Orthoptera

Family-Acrididae

4.1. Host plants:- sorghum, finger millet, rice, maize and Teff.

Alternate hosts:-wild grass.

4.3. **Life cycle (Biology and morphology)**

There are three stages in the grass hopper life cycle. This includes: -

The egg- nymph (= larva) - Adult.

Egg-The female lays eggs in the soil and surrounds the eggs with a frothy liquid that hardens to form a protective structure or "pod ". Each female may lay 56-900 eggs (the number of egg pods deposited may range from 7 to 30, and the number of eggs per pod may vary from 8 to 30) .

Nymphs:-Grasshopper nymphs undergo through five instars or developmental stages. After each instar, they shed their cuticle and grow larger. Nymphs normally reach the adult stage in five to six weeks.

Adult:-Adult is the only stage to have wings, readily move out of hatching areas and begins laying one to two weeks after becoming adults. They have two pairs of wings and the hind legs are enlarged and used for jumping. The mouth parts are of the chewing-biting type. Adults live two to three months.

4.4. Occurrence and population- Grass hopper damage occurs most frequently in areas with low rain fall. Grass hopper populations fluctuate in cycles. High or very high populations may be present for two to four years followed by low to moderate populations for several years.

4.5. Management/Control measures

Cultural Control

- Eliminate weeds and any susceptible plant from around crop field.
- Early planting
- Ploughing the field to expose the egg to natural enemy and sun, and eliminating potential egg laying site.
- Exposing grass hopper to birds.
- Planting the periphery of the field with non preferred crops such as sunflower or crucifers.
- In small holding, regular hand-picking and subsequent mechanical extermination can be practiced.

Natural/Biological Control

- There are several natural enemies of grass hopper.

Chemical control

The best time to control grass hoppers is during the 3rd and 4 th instars of nymphal stage. Adult grass hoppers are difficult to control with insecticides due to their size and less susceptibility to the insecticides.

Recommended insecticides are

- Carbaryl (sevin) 85% Wp 1.5 Kg/ha
- Fenitrothion (Sumithion) 50% Ec 1.5-2 lit/ha
- Malathion 50% Ec 1-2 lit/ha
- Ethiozinon 60% Ec 1-2 lit/ha
- Ethiodemethrin 2.5% Ec 150ml-400ml/ha

5. Lady bird beetles (Teff Epilachna)

Order – Coleoptera

Family – Coccinellidae

Species – *Epilachna similis*

The Lady bird beetles family (Coccinellidae) contains some species with vegetarian feeding habits, but most of them are predaceous on other insects, particularly on aphids, mealy bugs and

scale insects. Larvae and adults devour the pests and are there fore considered as beneficial insects. Even though most lady bird beetles considered as beneficial insects, in contrary some lady bird beetles especially Epilachna species cause considerable damage on crop plants and leads to high yield losses.

5.1. **Host plants**:-The range of host plants include a number of cereals such as wheat, barley, sorghum, Teff, maize, grasses and several other plants.

5.3. **Life cycle (Biology and morphology)**

There are four stages in *Epilachna similis* life cycle, the same as other coleopteran insects, which includes the egg -larva (grubs) -pupa - adult

Egg - The eggs are about 0.5 mm long and pale yellow in colour. They are elongate oval and have a hexagonal sculpturing. The eggs are laid in clusters of 20-50 eggs on the underside of the leaves in a vertical position.

Larva:-Mature grubs are 6-7 mm long and yellowish or dark yellow in colour. They are broad, with a dark head and bear dark coloured strong branched spines. But the young larvae are pale yellow and covered with delicate spines.

Pupa-Pupation takes place in the leaves of host plants. Mostly pupa is dark yellow in colour.

Adult – The adult beetles are oval or hemispherical in shape and about 6-8 mm in length. Adult *Epilachna similis* are reddish to reddish brown or brownish yellow in colour with black spots on the wing covers (elytra). Thorax and wing cases are covered with short, light-coloured hairs and generally dull appearance (not shiny). These beetles are very good fliers.

5.4. **Damage symptoms and patterns** -Both larvae (grubs) and adults feed on the leaves, usually on the underside, leaving the upper epidermis and veins intact (This type of damage is called “windowing “). Heavily attacked Leaves are skeletonized and dry up. In certain years this lady bird is found in great numbers and causes much damage.

5.5. Management/ Control methods.

Cultural control

- Early planting or planting at the exact time.
- Crop rotation
- Destroy weeds/alternate hosts from the field.

Monitoring

- It needs early and regular inspection of lady bird beetles insect pest occurrence, to control the pest before it makes sever damage.

Chemical control

-Chemical control is used as a last option (alternatives)

-Insecticide used to control lady bird beetle larvae and adult are

- ✓ Trichlorphon 95% sp 1 Kg/ha
- ✓ Fenitrothion 50% Ec 1.5 lit/ha
- ✓ Thiodan (endosulfan) 35% Ec 2 lit/ha
- ✓ Sevin (carbaryl) 85% Wp 1.5 Kg/ha
- ✓ Malathion 50% Ec 1.5-2 lit/ha

6. Sorghum Shoot fly

Order—Diptera

Family—Muscidae

Species—*Atherigona soccata*

6.1. Host plants

Main host:-sorghum

Alternate hosts:-maize, finger millet, Rice, wheat, and several species of grasses.

6.3. Life cycle (Biology and morphology of the insect)

Egg-the eggs are either deposited at the base of young shoots near soil surface or on the underside of the leaves of seedlings and on young tillers. One to three eggs are laid per leaf. The white eggs have an elongate shape, and measure 0.8X0.2mm. Hatching takes 2-3 days.

Larvae:-The full grown maggot is whitish or yellowish in colour and up to 8 to 10mm in length. The mouth hooks and the two spiracles projecting at the hind end of the body are stained black. The young maggots crawl down inside the sheath and then they bore into the base of the young shoot, Killing the growing point and youngest leaf. This leaf turns brown and withers. The larval period takes 7-12 days

- Maggot (larva) is the damaging stage.

Pupa-pupation usually takes place in the base of the dead shoot, but some times in the soil. The pupal period takes about 7 days.

Adult-The adult insect is 3-4 mm in length, the Head and thorax is grey (or pale grey) in colour, the abdomen is yellowish with paired brown patches. The male is more blackish. The wings are transparent. It looks like a small house fly.

- The total life cycle takes from 17-21 days depending up on weather conditions.

6.4. Damage symptoms and pattern- The maggot feed on the growing point of the shoot of the seedling and cause the characteristic << dead heart>> effect. When good growing conditions prevail the young plants are usually able to compensate the damage by producing new tillers which may partly escape attack, but later the ripening of the ear heads will be unequal. When the out break is severe also tillers may be attacked.

Management/Control methods

- **Cultural control**
  - Early sowing
  - Use resistant varieties when available.
  - Destroy voluntarily plants and grass weeds
- **Biological control (natural enemies)**
  - parasitoids—Trichogramma species
  - predators—spider
- **Chemical control**
  - Seed dressing – by using Marshall and carbofenothion
  - Recommended insecticide
    - Trichlorphon 95% sp at 1.5 kg/ha
    - Fenitrothion 50% Ec at 1 lit/ha
    - Dimethoate 40% Ec at 1-2 lit/ha

7. Aphids

Order-Homoptera

Family—Aphididae

7.1. Host plants:-Many cereals, Vegetables, Coffee, Sugar cane, Cotton, grasses, garden herbs and wild plants are attacked by different types of Aphid species.

7.3. Biology and Morphology of Aphids .

Aphids exhibit a number of colours. They are soft-bodied, more or less pear-shaped, small polyphagous insects, the largest species not more than 4 mm in length. The mouth parts are needle-sharp stylets for piercing and sucking. The rear end of the body bears two characteristic projections called cornicles/siphunculi. Most aphids are very host specific and Adult of the same species may have both apterous (wingless) and alate (winged) females, which reproduce asexually by vivipary except when unfavorable conditions (such as the long dry season in Ethiopia) may trigger sexual reproduction, some times associated with migration to a woody perennial host. Winged forms are mainly produced if the host plant is over crowded. There are three immature instars, all capable of walking. The life cycle of aphids can last as short as two weeks under favorable conditions. Warm and dry weather is especially favorable for rapid increase of aphid populations.

7.4. Damage pattern and symptoms

Aphids live in colonies on different parts of the plant, mostly on young shoots and leaves. The sugary, sticky excrements, called << honey dew >> accumulate on the upper surface of leaves and << Sooty mould >> fungus develops on them. The honey dew is a favorite food of some ant species. The ants protect the aphids against their natural enemies (lady bird beetles, Lacewing larvae, parasitic wasps, hover fly larvae, etc). The host plants are harmed by the extraction of sap, the injection of toxic saliva, the transmission of virus diseases, and the production of honey dew associated with sooty mould. The symptom of damage on host plants are-

- ✓ **On leaves** – yellowing, spotting, mottling, deformation and leaf curl
- ✓ **On flower/Inflorescences** – Inflorescences can be sterile and stagnation of flower and fruit production.
- ✓ **On plants**- New growth remains dwarfed or stunted. Heavy attack may cause the plant to wilt and die.

7.5. Management/Control measures.

- ❖ **Cultural control**
  - ✓ Choosing the appropriate sowing time.
  - ✓ Designing the appropriate cropping system. (such as intercropping with some botanicals having insecticidal properties)
  - ✓ Burn crop stubbles after harvest.

- ✓ Avoid alternate hosts, which serve as reservoir.
- ✓ Observe a closed season.
- ✓ Use of resistance Varieties when available.

- ❖ **Biological Control.**

Encourage natural enemies such as predators (lady bird beetles, spiders, larvae of syrphids, lace wings, etc…) and parasitoids by enhancing diversity.

- ❖ **Use sprays of natural products (botanicals) another solutions**
  - ✓ Chili, Garlic, neem, tobacco,… etc, some times we used more than one botanical
  - ✓ cow urine
  - ✓ soap solution
  - ✓ etc
- ❖ **Chemical control (as last options/alternatives)**
  - ✓ Primiphos-methyl (Actellic) 50% Ec at 0.5 lit/ha
  - ✓ Dimethoate 50% Ec 1.5 lit/ha
  - ✓ Thiometon 15% ULV at 1.2 lit/ha
  - ✓ Endosulfan (thiodan) 35% Ec 1.5 lit/ha
  - ✓ Malathion 50% Ec—1.5 lit/ha
  - ✓ Sumithion 50% Ec 1 lit/ha

8. Termites (white Ants).

Order—Isoptera

8.1. **Hosts**- Teff, Barley, Maize, Wheat, Millet, Sorghums, Cotton, and other several crops, trees and traditional built house.

8.2. Distribution- Important pests all over tropics (including tropical Africa) and a major pest of grass lands below 1500m in parts of Africa. In Ethiopia many species are found through out the country.

8.3. Life History and cycle

Termites are social insects, polymorphic, living in large communities, sometimes in elaborate nests both above and below ground. More than 2000 species of termites, all of which social and form colonies from several hundred individuals to more than a million (in some of the highly

specialized species). In Ethiopia so far, 61 species belonging to 25 genera and four families have been recorded (crop protection department, 2005).

Termites posses three stage of development (egg, nymph (= Larva), adult) which is called Incomplete metamorphosis. Termites are more closely related to cockroaches than to ants and live in colonies which consist of reproductive forms (Queens and Kings), Workers and soldiers.

- Reproductive forms (Queens and Kings) – The King and queens can reproduce all the castes, including others like them selves which at certain seasons swarm out of the nest in enormous numbers. Their wings are long, narrow, whitish, or semitransparent, with many indistinct veins, and the two pairs are almost exactly alike in size and appearance. At the beginning of rainy season the colony produce a number of sexual matured, winged males and females who leave the nest. After swarming these queens and kings loose their wings and start new colonies. These swarming, winged kings and queens are the only termites that normally appear in the open air. Among the termites, the kings (males) live a long time and mate repeatedly with the queens and all termite eggs are fertilized.
    - ✓ **Queens**:-The queen develops a very large abdomen (in some species as much as about 10 cm in length) filled with ovaries and lays eggs continuously through out her life. The queens are larger than other members of their colony. The eggs are laid at prodigals rate that totals about 30000 a day in some species.
    - ✓ **Kings**:-winged reproductive which usually fly after the rains.

- **Worker:-**The workers which form the majority of the population are wingless and sterile. The workers build nests, supply food, care for the eggs, feed the extremely young nymphs, the queens and kings, and perform others duties for the colony, except reproduction and defense.

- **Soldiers;** - The wingless soldiers are recognized by their modified mouthparts (power full jaws) and large brownish heads. The mandibles of the solder become enormously enlarged. Soldiers defend the colony and foraging workers from predators and other enemies.

8.4 Nest of termites:- Some large termite species live in big mounds which are called termitaria, which are constructed by them selves. The mounds constructed from 0.2cm to

more than 1m below the soil surface, inter connected by underground passages (complex network of passage ways).Each hive is a dome- shaped cavity about 0.5m in diameter, some are full of termites in all stages of development; others are used largely for the storage of cut grass or other crops.

8.5 DAMAGE:- Termites have biting-chewing mouth parts. They are considered as important pests all over the tropics attacking living plants as well as dry wood. The termites damage stem and roots, which is especially serious in seedlings often they cut the base of the plant at the ground level. Tree trunks or plant stems are covered with runways composed of plant fragments, soil and saliva. Underneath this protecting cover they feed on the bark. Trees may be ring-barked. Small plants may be killed. Losses in crops and trees due to pest species are often over 15% and in serious condition can reach 100%. The extent and type of damage vary with the presence of different termite species.

8.6 Control measures

- **Cultural control**
  - ✓ Planting early to avoid severe damage.
  - ✓ Crop rotation (do not plant two susceptible crops in consecutive years).
  - ✓ Ploughing after harvest.
  - ✓ Mechanical weeding
  - ✓ Remove stubbles immediately after harvest.
  - ✓ Removal of the queen or dig out and destroy the central located royal chamber where the king and queen are to be found (the remaining termites treated with chemicals).
- **Biological control**
  - ✓ Exposing termites living out in mounds by digging out and allowed to be eaten up by the animals.
- **Chemical control**
  - ✓ For termites on soil surface or in top soil

    -spray the affected area or water it by means of watering can, or broad cast an insecticide as a dust or wettable powder formulation and work it in to the soil 10-20 cm deep about two weeks before planting.
  - ✓ For termites in nests or mounds

pore the emulsion with a watering can in to the channel.

- ✓ Ethiozinone 6o% E.C 20-30cc/mound.

- **Other methods**
  - ✓ Use of wood ash
  - ✓ Spray cow dung

9. Cut worms (*Turnip moth*)

Order -Lepidoptera

Family –Noctuidae

Species –*Agrotis segetum*

9.1 Host plant:-It can be attack a wide range of plants, but the common host plants are vegetables, root crops, coffee seedlings, maize, tobacco, cotton, … etc.

9.3 Life cycle (biology and morphology of cut worm)

Eggs:--Eggs are laid singly or in small grouped on the stalks or leaves of the host plant or in the near by ground or in moist soil and on weeds. A single female may lays between 1000-1300eggs. These are ribbed and globular; they start off cream colored and turn reddish yellow to blackish before hatching. The caterpillars hatch 10-14 days.

Larvae: - the young larvae are pale, yellowish-green with a blackish head. But the mature caterpillars are grey, brown, greenish-grey and greenish-brown, or blackish in color and 4-5 cm in length. The larvae molt six times and its development periods vary from 3-4 weeks and influenced by the temperature and adequacy of diet. Third to last instars (6^{th}) become negatively phototoxic and they are nocturnal, hiding in the day time in the soil. Larvae construct burrows or tunnels in the soil about 2.5-5 cm deep. During the photophase in all these habitats, the larvae will usually seek a soil depth of 2.5-10 cm, where a moisture line exists. Often the larvae are found curled in the soil during the day time. Pupation takes place in the ground.

Pupation:-Pupae are about 15-25 mm in length and 5-6 mm in width. It is dark-brown or smooth and shiny reddish-brown with two dark spines at the tip of the abdomen. Pupa appears almost black in color just before the moth emerges.

Adult (moth):-The adult is medium sized moth, about 22 mm long with a wingspan of 40-50mm. The fore wings are greyish-brown with black lines or kidney shaped marking along the side margins. Hind wings are pearly white with brownish margins and veins. The males antennae are plumose (feathered), and the female antennae are filiform.

9.4 Damage pattern and symptoms:-Cut worms are polyphagous pests and the mature larvae cause the most damage. During the first one or two weeks of their life they feed on the young leaves of seedlings and can create perforations (shot holes) on leaves, later they migrate to the ground where they hide by the day near their hosts. At night they appear on the soil surface and attack the seedlings near ground level by biting holes in the stem or cutting them completely, often causing death of seedlings and some times wilting is observed because of partial cutting. Relatively small population of cut worms are capable of destroying entire stands of some crops such as maize and seedlings in a nursery, because of a cut worms larva will often cut one plant and quickly move on to other plants and continue cutting.

9.5 Control /Management Methods

- Cultural control
    - ✓ Ploughing(deep ploughing) a month before sowing turns the caterpillars or pupae up to the surface where they shrivel in sun or eaten by birds
    - ✓ Early avoidance of weeds reduces the egg laying sites
    - ✓ Avoid planting susceptible crops in fields with a known history of cut worm problems.
    - ✓ In small area removing by hand at early in the morning and after noon.
    - ✓ Flooding of soils induces caterpillars to leave their hiding places during the day time and thus become exposed to predators and adverse environmental conditions.
- ***Biological control***
    - ✓ Ants
    - ✓ Several bird species (including hens)
- ***Other solutions***
    - ✓ Treat the seed bed with wood ash or a mixture of wood ash and chalk
- ***Chemical control***
    - ✓ As a last alternative, insecticide like Chlorpyrifos, Diazinone, (Ethiozinone) 60% E.C and Ethiodemetrin 2.5% E.C used to control cut worms.

CHAPTER NINE

AGRICULTURAL PESTICIDE AND ITS APPLICATION

A pesticide is any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest.

- **NB**. Herbicides, Insecticides, Nematicides, Fungicides and Rodenticides are a part of pesticide. Those pesticides are very important to manage pest

Nomenclature of Pesticide

- There is often more than one formulation of a particular pesticide. This can make selection and application of various products somewhat confusing. Each pesticide has a trade name (sometimes more than one), a common name, and a chemical name.

Any approved herbicide is known by three names.

1. Common name

- Short form of the chemical is called common name commonly accepted short name. Common names are agreed upon by a committee on terminologies.

2. Chemical name

- It gives full molecular structure.

3. Trade name

- Trade name is the name offered by the manufacturer. These names are approved by different organizations or institutes which are considered authority on this subject

Role of pesticides

- Pesticide should be applied only based on the need, i.e. if pest reaches ETL.

2. It should be judiciously combined with other components of IPM and pesticides should be used as last resort.

3. When pest population approaches ETL, pesticides are the only means of preventing economic damage.

4. Pesticides are available in easy and ready to use packings.

5. Easy to apply and large area can be covered.

6. A range of insecticides are available depending on crop, insect and nature of damage.

7. Pesticides which are cost effective (High benefic/cost ratio) and safe (High benefit/risk ratio)

Proper Handling Procedures

General *Pesticide Handling Rules*

- Always read and follow pesticide label instructions. It is against the law to use a pesticide in any manner not specifically listed on the label or contrary to label instructions and requirements.
- Always wear the protective clothing specified on the manufacturer's label. Use of protective clothing should be in compliance with federal, local and state laws.
- Handle pesticide containers carefully to prevent accidental ruptures or spills. Pesticide containers should never be dropped or thrown. Be aware of proper cleanup procedures in case of an accident.

Open pesticide containers carefully to prevent spills and make resealing easier.

- Always measure pesticides carefully. Pesticides should only be used as directed on the product label.
- Measure and pour pesticides *below eye level to* prevent spills or splashes

Never spray pesticides in a manner that creates drift.

Classification of pesticide

Groups of pesticides: The pesticides are generally classified into various groups based on pest organism against which the compounds are used, their chemical nature, mode of entry and mode of action.

1. **Based on organisms**

a)Insecticides: Chemicals used to kill or control insects **eg**. endosulfan, malathion

b) **Rodenticides**: Chemicals exclusively used to control rats **eg**. Zinc phosphide

c) **Nematicides**:- Chemicals used to control nematodes **eg**. Ethylene dibromide

d) **Fungicides**: Chemicals used to control plant diseases caused by fungi (eg.) Copper oxy cholirde

e) **Herbicide** : Chemicals used to control weeds (eg.) 2,4,-D

f) **Bactericide**: Chemicals used to control the plant diseases caused by bacteria (eg.) Streptomycin sulphate

g) **Avicides**: Chemicals used to repel the birds (eg.) Anthraquionone

h) **Molluscicides**: Chemicals used to kill the snails and slugs (eg.) Metaldehyde

2. Based on mode of entry

a) **Stomach poison**: The insecticide applied in the leaves and other parts of the plant when ingested, act in the digestive system of the insect and bring about kill

(eg.) Malathion.

b) **Fumigant**: Toxicant enter in vapour form into the tracheal system (respiratory poison) through spiracles (eg.) Aluminium phosphide

c) **Protoplasmic poison**: Toxicant responsible for precipitation of protein (eg.) Arsenicals.

d) **Nerve poison** : Chemicals inhibit impulse conduction (eg.) Malathion.

e) **Chitin inhibition** : Chemicals inhibit chitin synthesis (eg.) Diflubenzuron

f) **Contact Poison** : The toxicant which brings about death of the pest species by means of contact (eg.) Fenvalerate.

g) **Systemic poison**: Chemicals when applied to plant or soil are absorbed by foliage (or) roots and translocated through vascular system and cause death of insect feeding on plant. (eg.) Dimethoate.

Classification based on chemical nature

- **Inorganic chemicals**

Eg. Arsenic, Fluorine, Sulphur, lime sulphur (Insecticides) zinc phosphide (Rodenticide)

II. **Organic pesticides** Organic compounds (constituted by C, H, O and N mainly)

a. **Hydrocarbon oil (or) Petroleum oi**l – eg. Coal tar oil, kerosine etc.,

b. **Animal origin:**– eg. Nereistoxin extracted from marine annelids – commercially available as cartap, padan.

c. **Plant origin**: Nicotine from tobacco plants, pyrethrum from Chrysanthemum flowers, Rotenoids from roots of Derris and Lonchocarpus Neem – azadirachtin, Pongamia glabra, Garlic etc.,

d. **Synthetic organic compounds: These organic chemicals are synthetically produced in laboratory.**

i. Chlorinated hydrocarbon (or) organochlorines Eg. DDT, HCH, Endosulfan, Lindane, Dicofol (DDT, HCH banned)

ii. Cyclodienes Eg. Chlordane, Heptachlor (Banned chemicals)

iii Organophosphates : (Esters of phosphoric acid) Eg. Dichlorvos, Monocrotophos, Phospamidon, Methyl parathion, Fenthion, Dimethoate, Malathion, Acephate, Chlorpyriphos

Basedonchemicalformulation

A, Chlorinated hydrocarbons

- This group are broad spectrum pesticides, include DDT, Aldrin, Dieldrin, Toxaphene, Lindane, Chlordane, Methooxychlor, Kepone
- Their persistence in the environmental media range between 2 and15 years

B, **Organophosphate**

- include broad and narrow spectrum pesticides such as Malathion, Parathion, Methyl parathion, Diazinon.
- their persistence in the environment is generally low to moderate (normally1to12 weeks). Some can last several years. Organophosphate can easily contaminate water supply because they are water-soluble.
- don't have characteristics of bioaccumulation and amplification.
- highly toxic to human and other animals.

e.g. Malathion is less toxic to mammals than other Organophosphates

C, Carbamates

- are broad and narrow spectrum agents. Examples include: carbaryl, (sevin), propoxur carbo furan, aldicarb, maned zineb and methomyl.
- are less persistence (days to weeks)
- don't bioaccumulate and are not biologically amplified
- their toxicity for humans and other animals are from low to high unfortunately they are highly toxic to honeybees.

D, Botanical

- Are group of broad and narrow spectrum agents produced naturally by plants or by chemical modifications of such natural substances. Examples of natural botanicals include rotenone, pyrethrum and camphor.
- Are low persistence (day to week) are effective at a low dose, do not bio accumulate, are not biologically amplified.
- Have low to moderate toxicity to humans and animals. Most are expensive.

Microbotanicals

- these are diverse array of microorganisms, including bacteria , fungi, and protozoan,
- selectively kill particular pest , usually by producing certain toxins. Examples include Bacillus, Popilliae (beetles)

- have low persistence (day to weeks) are effective at a low dose,
- do not bioaccumulate, are not biologically amplified have low toxicity to humans and other animals

Classification of pesticides according to hazard *(Based on WHO Classification, 1996)*

- According to the World Health Organization (WHO) pesticides have been classified into four categories: That are extremely hazardous, highly hazardous. moderately hazardous, slightly hazardous, depending on their acute oral and dermal LD50%
- A value for solids in both oral and dermal tests is always lower in all four categories of acute toxicity levels. In the same way solid pesticides have lower acquit toxicity than liquid pesticides.

LD50% Based hazardous classification

Class LD 50% for the rat (mg/Kg body weight)

		Oral	**Oral**	**Dermal**	**Dermal**
		solids	**Liquids**	**Slids**	**Liquids**
1a	Extremely hazardous	5 or less	20 or less	10 or less	40 or less
1b	Highly hazardous	5-50	20-200	10-100	40-100
II	Moderately hazardous	50-500	200-2000	100-1000	100-4000
III	Slightly hazardous	500-2000	2000-3000	Over 1000	Over 4000