

## **LECTURE NOTES**

Degree and Diploma Programs  
For Health Science Students

# ***Medical Parasitology***



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## **PREFACE**

This lecture note is useful to students of health science, medicine and other students and academicians. It is believed to provide basic knowledge to students on medical parasitology. It also serves as a good reference to parasitologists, graduate students, biomedical personnel, and health professionals. It aims at introducing general aspects of medically important parasites prevalent in the tropics and in Ethiopia in particular. It is our belief that this note will contribute much in alleviating the shortage of Parasitology texts.

Students preparing to provide health care in their profession need solid foundation of basic scientific knowledge of etiologic agents of diseases, their diagnosis and management. To face the fast growing trends of scientific information, students require getting education relevant to what they will be doing in their future professional lives. Books that are of manageable size are increasingly important in helping students learn the seemingly overwhelming amount of information they must absorb.

## ACKNOWLEDGEMENTS

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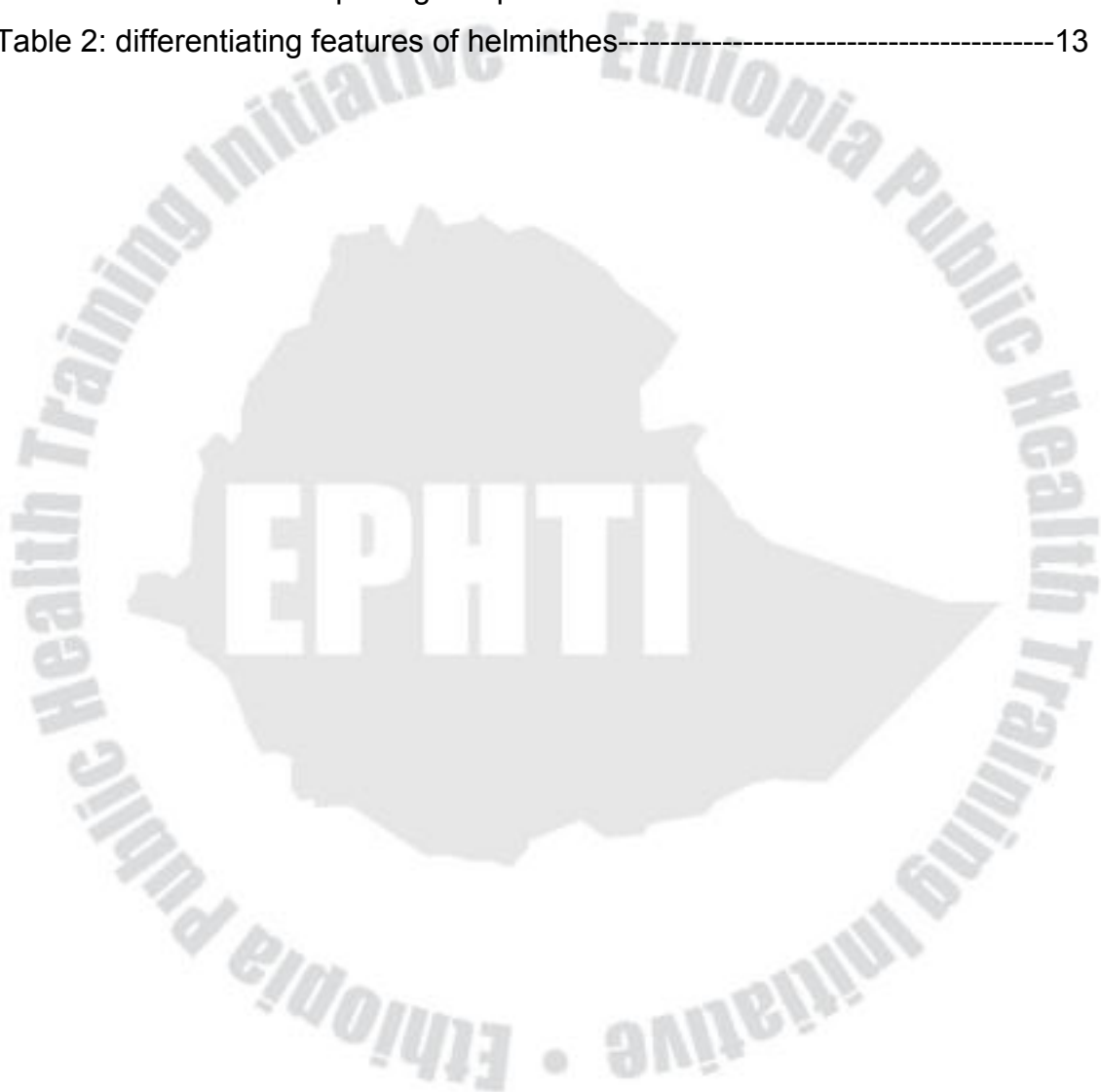
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## ABBREVIATIONS AND ACRONYMS

CNS: Central nervous system

CSF: Cerebro-spinal fluid

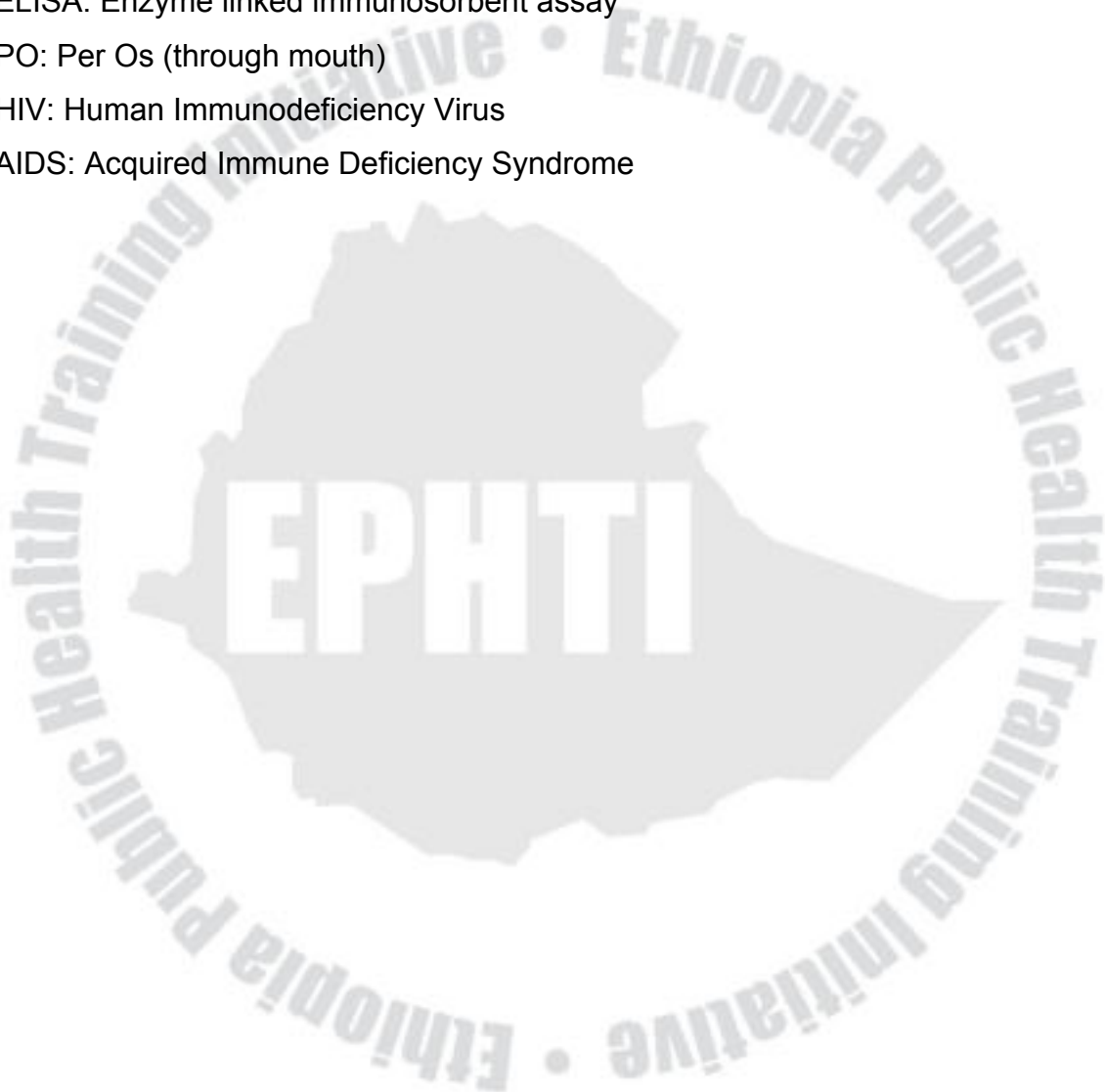
DEC: Diethyl carbamazine

ELISA: Enzyme linked immunosorbent assay

PO: Per Os (through mouth)

HIV: Human Immunodeficiency Virus

AIDS: Acquired Immune Deficiency Syndrome



# UNIT ONE

## GENERAL PARASITOLOGY

### LEARNING OBJECTIVES

**At the end of this section the student is expected to:**

- Discuss the various types of parasites and hosts.
- Explain the relationship between a parasite and the host and their effects.
- Discuss in detail the classification of medically important parasites.
- Explain the difference between the Cestodes, Nematodes, Trematodes and protozoa

### INTRODUCTION

Man and other living things on earth live in an entangling relationship with each other. They don't exist in an isolated fashion. They are interdependent; each forms a strand in the web of life. Medical parasitology is the science that deals with organisms living in the human body (the host) and the medical significance of this host-parasite relationship.

### ASSOCIATION BETWEEN PARASITE AND HOST

**A parasite** is a living organism, which takes its nourishment and other needs from a host; the host is an organism which supports the parasite. The parasites included in medical parasitology are protozoa, helminthes, and some arthropods. (See box 1 for broader classification of parasites). The hosts vary depending on whether they harbor the various stages in parasitic development. (See box 2)

## BOX 1. DIFFERENT KINDS OF PARASITES

- **Ectoparasite** – a parasitic organism that lives on the outer surface of its host, e.g. lice, ticks, mites etc.
- **Endoparasites** – parasites that live inside the body of their host, e.g. *Entamoeba histolytica*.
- **Obligate Parasite** - This parasite is completely dependent on the host during a segment or all of its life cycle, e.g. *Plasmodium* spp.
- **Facultative parasite** – an organism that exhibits both parasitic and non-parasitic modes of living and hence does not absolutely depend on the parasitic way of life, but is capable of adapting to it if placed on a host. E.g. *Naegleria fowleri*
- **Accidental parasite** – when a parasite attacks an unnatural host and survives. E.g. *Hymenolepis diminuta* (rat tapeworm).
- **Erratic parasite** - is one that wanders in to an organ in which it is not usually found. E.g. *Entamoeba histolytica* in the liver or lung of humans.

Most of the parasites which live in/on the body of the host do not cause disease (non-pathogenic parasites). In Medical parasitology we will focus on most of the disease causing (pathogenic) parasites. However, understanding parasites which do not ordinarily produce disease in healthy (immunocompetent) individuals but do cause illness in individuals with impaired defense mechanism (opportunistic parasites) is becoming of paramount importance because of the increasing prevalence of HIV/AIDS in our country.

## BOX 2. DIFFERENT KINDS OF HOSTS

- **Definitive host** – a host that harbors a parasite in the adult stage or where the parasite undergoes a sexual method of reproduction.
- **Intermediate host** - harbors the larval stages of the parasite or an asexual cycle of development takes place. In some cases, larval development is completed in two different intermediate hosts, referred to as first and second intermediate hosts.
- **Paratenic host** – a host that serves as a temporary refuge and vehicle for reaching an obligatory host, usually the definitive host, i.e. it is not necessary for the completion of the parasites life cycle.
- **Reservoir host** – a host that makes the parasite available for the transmission to another host and is usually not affected by the infection.
- **Natural host** – a host that is naturally infected with certain species of parasite.
- **Accidental host** – a host that is under normal circumstances not infected with the parasite.

There is a dynamic equilibrium which exists in the interaction of organisms. Any organism that spends a portion or all of its life cycle intimately associated with another organism of a different species is considered as Symbiont (symbiote) and this relationship is called symbiosis (symbiotic relationships).

The following are the three common symbiotic relationships between two organisms:

**Mutualism** - an association in which both partners are metabolically dependent upon each other and one cannot live without the help of the other; however, none of the partners suffers any harm from the association. One classic example is the relationship between certain species of flagellated protozoa living in the gut of termites. The protozoa, which depend entirely on a carbohydrate diet, acquire their nutrients from termites. In return they are capable of synthesizing and secreting cellulases; the cellulose digesting enzymes, which are utilized by termites in their digestion.

**Commensalism** - an association in which the commensal takes the benefit without causing injury to the host. E.g. Most of the normal floras of the humans' body can be considered as commensals.

**Parasitism** - an association where one of the partners is harmed and the other lives at the expense of the other. E.g. Worms like *Ascaris lumbricoides* reside in the gastrointestinal tract of man, and feed on important items of intestinal food causing various illnesses.

Once we are clear about the different types of associations between hosts and parasites, we can see the effect the parasite brings to the host and the reactions which develop in the host's body due to parasitic invasion.

## **EFFECT OF PARASITES ON THE HOST**

The damage which pathogenic parasites produce in the tissues of the host may be described in the following two ways;

### **(a) Direct effects of the parasite on the host**

- Mechanical injury - may be inflicted by a parasite by means of pressure as it grows larger, e.g. Hydatid cyst causes blockage of ducts such as blood vessels producing infraction.
- Deleterious effect of toxic substances- in *Plasmodium falciparum* production of toxic substances may cause rigors and other symptoms.
- Deprivation of nutrients, fluids and metabolites -parasite may produce disease by competing with the host for nutrients.

### **(b) Indirect effects of the parasite on the host:**

Immunological reaction: Tissue damage may be caused by immunological response of the host, e.g. nephritic syndrome following Plasmodium infections. Excessive proliferation of certain tissues due to invasion by some parasites can also cause tissue damage in man, e.g. fibrosis of liver after deposition of the ova of *Schistosoma*.



## BASIC CONCEPTS IN MEDICAL PARASITOLOGY

In medical parasitology, each of the medically important parasites are discussed under the standard subheadings of morphology, geographical distribution, means of infection, life cycle, host/parasite relationship, pathology and clinical manifestations of infection, laboratory diagnosis, treatment and preventive/control measures of parasites. In the subsequent section some of these criteria are briefly presented.

**Morphology** - includes size, shape, color and position of different organelles in different parasites at various stages of their development. This is especially important in laboratory diagnosis which helps to identify the different stages of development and differentiate between pathogenic and commensal organisms. For example, *Entamoeba histolytica* and *Entamoeba coli*.

**Geographical distribution** - Even though revolutionary advances in transportation has made geographical isolation no longer a protection against many of the parasitic diseases, many of them are still found in abundance in the tropics. Distribution of parasites depends upon:

- a. The presence and food habits of a suitable host:
  - Host specificity, for example, *Ancylostoma duodenale* requires man as a host where *Ancylostoma caninum* requires a dog.
  - Food habits, e.g. consumption of raw or undercooked meat or vegetables predisposes to Taeniasis
- b. Easy escape of the parasite from the host- the different developmental stages of a parasite which are released from the body along with faeces and urine are widely distributed in many parts of the world as compared to those parasites which require a vector or direct body fluid contact for transmission.
- c. Environmental conditions favoring survival outside the body of the host, i.e. temperature, the presence of water, humidity etc.
- d. The presence of an appropriate vector or intermediate host – parasites that do not require an intermediate host (vector) for transmission are more widely distributed than those that do require vectors.



Once we are clear about the geographical distribution and conditions favoring survival in relation to different parasites, effective preventive and control measures can more easily be devised and implemented.

**Life cycle of parasites** - the route followed by a parasite from the time of entry to the host to exit, including the extracorporeal (outside the host) life. It can either be simple, when only one host is involved, or complex, involving one or more intermediate hosts. A parasite's life cycle consists of two common phases one phase involves the route a parasite follows inside the body. This information provides an understanding of the symptomatology and pathology of the parasite. In addition the method of diagnosis and selection of appropriate medication may also be determined. The other phase, the route a parasite follows outside of the body, provides crucial information pertinent to epidemiology, prevention, and control.

**Host parasite relationship** - infection is the result of entry and development within the body of any injurious organism regardless of its size. Once the infecting organism is introduced into the body of the host, it reacts in different ways and this could result in:

- a. Carrier state - a perfect host parasite relationship where tissue destruction by a parasite is balanced with the host's tissue repair. At this point the parasite and the host live harmoniously, i.e. they are at equilibrium.
- b. Disease state - this is due to an imperfect host parasite relationship where the parasite dominates the upper hand. It can result either from lower resistance of the host or a higher pathogenicity of the parasite.
- c. Parasite destruction – occurs when the host takes the upper hand.

**Laboratory diagnosis** – depending on the nature of the parasitic infections, the following specimens are selected for laboratory diagnosis:

- a) **Blood** – in those parasitic infections where the parasite itself in any stage of its development circulates in the blood stream, examination of blood film forms one of the main procedures for specific diagnosis. For example, in malaria the parasites are found inside the red blood cells. In Bancroftian and Malayan filariasis, microfilariae are found in the blood plasma.

b) **Stool** – examination of the stool forms an important part in the diagnosis of intestinal parasitic infections and also for those helminthic parasites that localize in the biliary tract and discharge their eggs into the intestine.

In protozoan infections, either trophozoites or cystic forms may be detected; the former during the active phase and the latter during the chronic phase. Example, Amoebiasis, Giardiasis, etc.

In the case of helminthic infections, the adult worms, their eggs, or larvae are found in the stool.

c) **Urine** – when the parasite localizes in the urinary tract, examination of the urine will be of help in establishing the parasitological diagnosis. For example in urinary Schistosomiasis, eggs of *Schistosoma haematobium* are found in the urine. In cases of chyluria caused by *Wuchereria bancrofti*, microfilariae are found in the urine.

d) **Sputum** – examination of the sputum is useful in the following:

- In cases where the habitat of the parasite is in the respiratory tract, as in Paragonimiasis, the eggs of *Paragonimus westermani* are found.
- In amoebic abscess of lung or in the case of amoebic liver abscess bursting into the lungs, the trophozoites of *E. histolytica* are detected in the sputum.

e) **Biopsy material** - varies with different parasitic infections. For example spleen punctures in cases of kala-azar, muscle biopsy in cases of Cysticercosis, Trichinelliasis, and Chagas' disease, Skin snip for Onchocerciasis.

f) **Urethral or vaginal discharge** – for *Trichomonas vaginalis*

Indirect evidences – changes indicative of intestinal parasitic infections are:

- a. **Cytological changes in the blood** – eosinophilia often gives an indication of tissue invasion by helminthes, a reduction in white blood cell count is an indication of kala-azar, and anemia is a feature of hookworm infestation and malaria.
- b. **Serological tests** – are carried out only in laboratories where special antigens are available.

**Treatment** – many parasitic infections can be cured by specific chemotherapy. The greatest advances have been made in the treatment of protozoal diseases.

For the treatment of intestinal helminthiasis, drugs are given orally for direct action on the helminthes. To obtain maximum parasitocidal effect, it is desirable that the drugs administered should not be absorbed and the drugs should also have minimum toxic effect on the host.

**Prevention and control** - measures may be taken against every parasite infecting humans. Preventive measures designed to break the transmission cycle are crucial to successful parasitic eradication. Such measures include:

- Reduction of the source of infection- the parasite is attacked within the host, thereby preventing the dissemination of the infecting agent. Therefore, a prompt diagnosis and treatment of parasitic diseases is an important component in the prevention of dissemination.
- Sanitary control of drinking water and food.
- Proper waste disposal – through establishing safe sewage systems, use of screened latrines, and treatment of night soil.
- The use of insecticides and other chemicals used to control the vector population.
- Protective clothing that would prevent vectors from resting in the surface of the body and inoculate pathogens during their blood meal.
- Good personal hygiene.
- Avoidance of unprotected sexual practices.

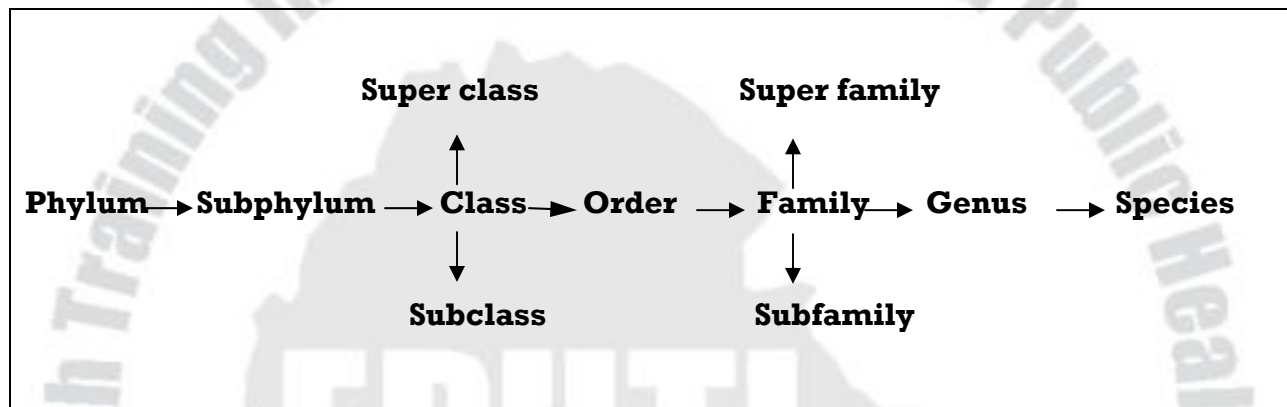
## **CLASSIFICATION OF MEDICAL PARASITOLOGY**

Parasites of medical importance come under the kingdom called protista and animalia. Protista includes the microscopic single-celled eukaryotes known as protozoa. In contrast, helminthes are macroscopic, multicellular worms possessing well-differentiated tissues and complex organs belonging to the kingdom animalia. Medical Parasitology is generally classified into:

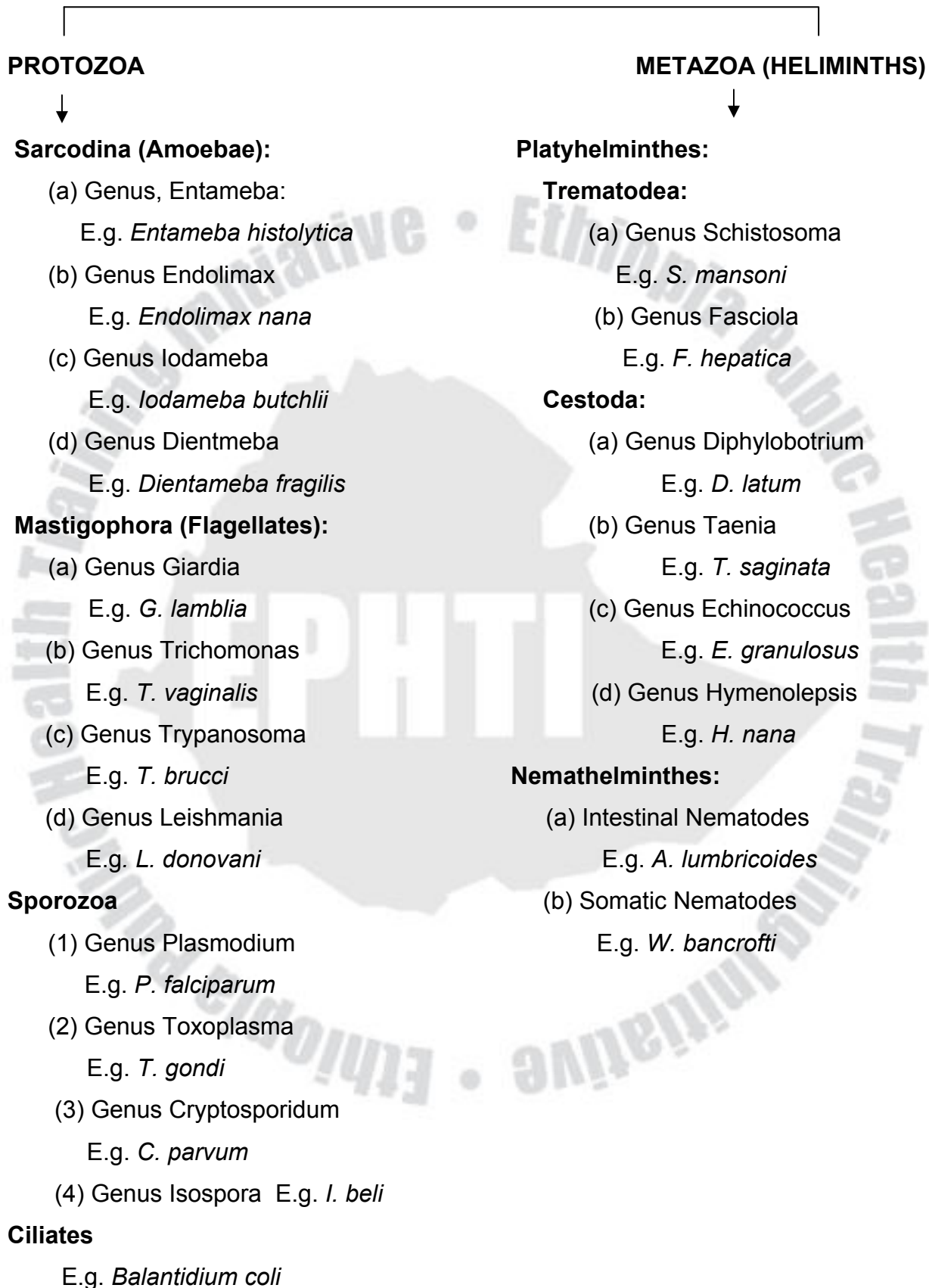
- **Medical Protozoology** - Deals with the study of medically important protozoa.

- **Medical Helminthology** - Deals with the study of helminthes (worms) that affect man.
- **Medical Entomology** - Deals with the study of arthropods which cause or transmit disease to man.

Describing animal parasites follow certain rules of zoological nomenclature and each phylum may be further subdivided as follows:



**FIGURE 1. CLASSIFICATION OF MEDICALLY IMPORTANT PARASITES**



## GENERAL CHARACTERISTICS OF MEDICALLY IMPORTANT PARASITES

Medically important protozoa, helminthes, and arthropods, which are identified as causes and propagators of disease have the following general features. These features also differ among parasites in a specific category.

### (1) PROTOZOA

Protozoan parasites consist of a single "cell-like unit" which is morphologically and functionally complete and can perform all functions of life. They are made up of a mass of protoplasm differentiated into cytoplasm and nucleoplasm. The cytoplasm consists of an outer layer of hyaline ectoplasm and an inner voluminous granular endoplasm. The ectoplasm functions in protection, locomotion, and ingestion of food, excretion, and respiration. In the cytoplasm there are different vacuoles responsible for storage of food, digestion and excretion of waste products. The nucleus also functions in reproduction and maintaining life.

The protozoal parasite possesses the property of being transformed from an active (trophozoite) to an inactive stage, losing its power of motility and enclosing itself within a tough wall. The protoplasmic body thus formed is known as a cyst. At this stage the parasite loses its power to grow and multiply. The cyst is the resistant stage of the parasite and is also infective to the human host.

**Reproduction** – the methods of reproduction or multiplication among the parasitic protozoa are of the following types:

1. Asexual multiplication:

- (a) Simple binary fission – in this process, after division of all the structures, the individual parasite divides either longitudinally or transversely into two more or less equal parts.
- (b) Multiple fission or schizogony – in this process more than two individuals are produced, e.g. asexual reproduction in Plasmodia.



## 2. Sexual reproduction:

- (a) Conjugation – in this process, a temporary union of two individuals occurs during which time interchange of nuclear material takes place. Later on, the two individuals separate.
- (b) Syngamy – in this process, sexually differentiated cells, called gametes, unite permanently and a complete fusion of the nuclear material takes place. The resulting product is then known as a zygote.

Protozoa are divided into four types classified based on their organs of locomotion. These classifications are: amoebas, ciliates, flagellates, and sporozoans.

**TABLE 1. CLASSIFICATION OF THE PATHOGENIC PROTOZOA:**

| PROTOZOA                         | ORGAN OF LOCOMOTION                         | IMPORTANT HUMAN PATHOGENS                            |
|----------------------------------|---------------------------------------------|------------------------------------------------------|
| 1. Rhizopoda<br>(Amoeba)         | Pseudopodia                                 | <i>Entamoeba histolytica</i>                         |
| 2. Mastigophora<br>(Flagellates) | Flagella                                    | Trypanosomes<br>Leishmania<br>Trichomonas<br>Giardia |
| 3. Sporozoa                      | None, exhibit a slight<br>Amoeboid movement | Plasmodium.Spp                                       |
| 4. Ciliates                      | Cilia                                       | <i>Balantidium coli</i>                              |

## (2) HELMINTHS:

The helminthic parasites are multicellular, bilaterally symmetrical animals having three germ layers. The helminthes of importance to human beings are divided into three main groups with the peculiarities of the different categories described in table 2.

**TABLE 2. DIFFERENTIATING FEATURES OF HELMINTHES**

|                         | <b>CESTODE</b>            | <b>TREMATODE</b>                                                     | <b>NEMATODE</b>        |
|-------------------------|---------------------------|----------------------------------------------------------------------|------------------------|
| <b>Shape</b>            | Tape like, segmented      | Leaf like, Unsegmented                                               | Elongated, Cylindrical |
| <b>Sexes</b>            | Not separate (monoecious) | Not separate (monoecious)<br>Except blood flukes which are dioecious | Separate. (diecious)   |
| <b>"Head" End</b>       | Suckers: with hooks       | Suckers: no hooks                                                    | No suckers, and hooks  |
| <b>Alimentary canal</b> | Absent                    | Present but incomplete                                               | Present and complete   |
| <b>Body cavity</b>      | Absent                    | Absent                                                               | Present                |



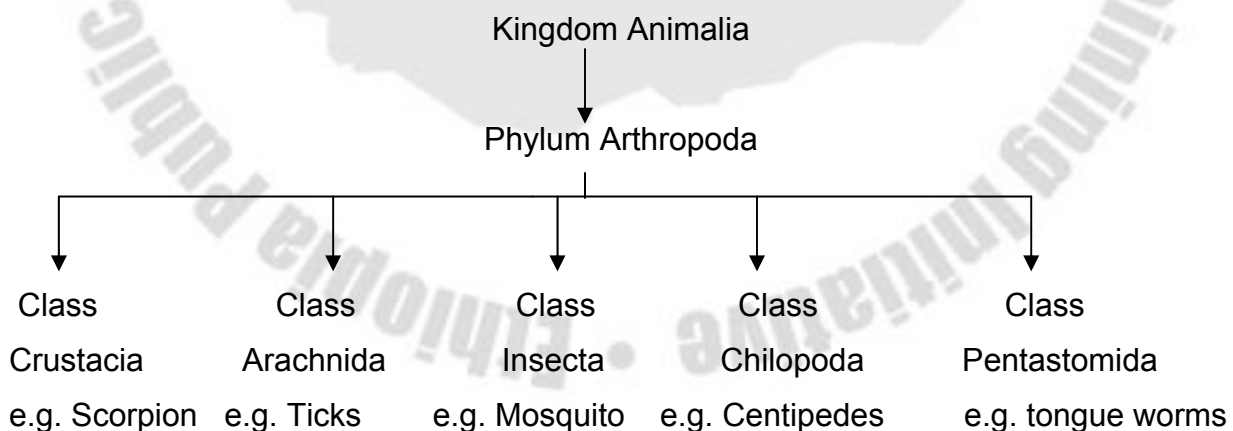
### (3) ARTHROPODS

Arthropods, which form the largest group of species in the animal kingdom, are characterized by having a bilaterally symmetrical and segmented body with jointed appendages. They have a hard exoskeleton, which helps enclose and protect the muscles and other organs. An open circulatory system, with or without a dorsally situated heart pumps the blood (hemolymph) via arteries to the various organs and body tissues. Blood is returned to the heart through body spaces known as hemocoeles. In addition, respiratory, excretory, and nervous systems are present.

Arthropods affect the health of humans by being either direct agents for disease or agents for disease transmission.

The arthropods of medical importance are found in Classes Insecta, Arachnida, and Crustacea which have their own distinguishing features. In Class insecta the body is divided into head, thorax, and abdomen, with one pair of antennae. Diseases like malaria, yellow fever, onchocerciasis, and trypanosomiasis are primarily transmitted by insects.

**FIGURE 2. CLASSIFICATION OF ARTHROPODS**



**N.B.** Crustacea, Arachnida, and Insecta are the three most common classes of arthropods of medical significance, which need closer attention

## SUMMARY

A parasite is an organism which lives in/on the body of a host. A host is that which harbors the parasite. There is usually some association such as mutualism, commensalisms, or parasitism between the parasite and the host. This association may produce a variety of effects and the host usually tends to react to it.

Understanding the various structural and behavioral components of parasites assists classification. In general, the protozoa, helminthes and arthropods are the most commonly studied and the most important parasites in medical parasitology. They are further sub classified considering many parameters.



## REVIEW QUESTIONS

1. Explain briefly the various types of parasites and hosts.
2. Explain the three types of symbiotic relationships and give examples.
3. Discuss the mechanisms by which parasites impose their effect on the host.
4. Give examples of reactions that occur in the body of the host following parasitic invasion.

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## **UNIT TWO**

### **MEDICAL PROTOZOLOGY**

#### **LEARNING OBJECTIVES:**

At the end of the lesson, the student should be able to:

- Discuss the classification of medically important protozoa.
- Discuss the pathogenesis and clinical aspects of infections.
- Describe the general epidemiological aspects and transmission patterns of diseases caused by protozoa.
- Identify the methods and procedures of laboratory diagnosis of pathogenic protozoa in clinical specimens.
- Discuss treatment options for protozoan infections.
- Implement the preventive and control measures of protozoan infection.

#### **INTRODUCTION**

Protozoa (singular, protozoan), from the Greek 'protos' and 'zoon' meaning "first animal", are members of eukaryotic protists. They may be distinguished from other eukaryotic protists by their ability to move at some stage of their life cycle and by their lack of cell wall.

#### **Occurrence of protozoa**

Protozoa are found in all moist habitats. They are common in sea, in soil and in fresh water. These organisms occur generally as a single cell. Colonies of protozoa might also occur in which individual cells are joined by cytoplasmic threads and form aggregates of independent cells.

However, distinct types of protozoa, include a resistant cyst (non-motile) stage to survive adverse environmental conditions, such as desiccation, low nutrient supply, and even anaerobiosis. For example, the soil amoeba, *Naegleria* is a resistant cyst in dry

weather, a naked amoeba in moist soil, and becomes flagellated when flooded with water.

## **Morphology of protozoa**

Protozoa are predominantly microscopic, ranging in size from 2 to more than 100 $\mu$ m. Morphologically, they are within a mass of protoplasm, consisting of a true membrane – bound nucleus and cytoplasm.

The nucleus contains clumped or dispersed chromatin and central nucleolus or karyosome, which are useful structures to distinguish protozoan species from one another based on the shape, size and distribution of these structures.

## **Importance of protozoa**

Protozoa serve as an important link in the food chain and ecological balance of many communities in wetland & aquatic environments. They are also important in biological sewage treatment, which involves both anaerobic digestion and/or aeration. In addition, protozoa are important laboratory organisms in research areas, by which their asexual reproduction enables clones to be established with the same genetic make-up. These are useful in the study of cell cycles and nucleic acid biosynthesis during cell division.

## **Medical concern of protozoa**

Protozoa are ubiquitous in moist areas, including the human alimentary canal. From an ecological standpoint, protozoa may be divided into free-living forms and symbiotic forms. Some of the symbiotic ones are parasitic and may cause disease.

Although most amoebas are free-living, several are found as commensal inhabitants of the intestinal tract in humans. One of these organisms *Entamoeba histolytica* may invade tissue and produce disease. The majority of ciliates are free living and seldom parasitize humans. Flagellates of the genus Trypanosomes and Leishmania are capable of invading the blood & tissue of humans, where they produce severe chronic illness. Others such as *Trichomonas vaginalis* and *Giardia lamblia*, inhabit the

urogenital and gastrointestinal tracts and initiate disease characterized by mild to moderate morbidity but no mortality.

Sporozoan organisms, in contrast, produce two of the most potentially lethal diseases of humankind: malaria and toxoplasmosis. With the advent of HIV a new and important chapter has been opened; i.e. 'opportunistic' parasitosis. Most of the parasitic incidents belong to endocellular protozoa of different genera or species.

## **Reproduction and regeneration of protozoa**

As a general rule, protozoa multiply by asexual reproduction. This is not to say that sexual processes are absent in the protozoa. Some parasitic forms may have an asexual phase in one host and a sexual phase in another host. (refer to page 18 for details on reproduction of protozoans)

## **Transmission**

In most parasitic protozoa, the developmental stages are often transmitted from one host to another within a cyst. The reproduction process is also related to the formation of the cyst. Asexual reproduction of some ciliates and flagellates is associated with cyst formation, and sexual reproduction of Sporozoa invariably results in a cyst. Pathogenic protozoa can spread from one infected person to another by:

- Faecal – oral transmission of contaminated foods and water.
- Insect bit inoculums or rubbing infected insect faeces on the site of bite.
- Sexual intercourse

## **Pathogenesis**

Protozoan organisms are virtually always acquired from an exogenous source, and as such, they have evolved numerous ways to enter the body of the human host. Factors that are important for pathogenicity include:

- Attachment to the host tissue followed by replication to establish colonization.

- Toxic products released by parasitic protozoa.
- Shifting of antigenic expression to evade the immune response and inactivate host defences.

## Antiprotozoal agents

Generally the antiprotozoal agents target relatively rapidly proliferating, young, growing cells of the parasite. Most commonly, these agents target nucleic acid synthesis, protein synthesis, or specific metabolic pathways (e.g. folate metabolism) unique to the protozoan parasites.

## CLASSIFICATION OF PROTOZOA

Protozoa of medical importance are classified based on their morphology and locomotive system as described below:

Amoebas - *Entamoeba histolytica*

Flagellates - *Giardia lamblia*, *Trichomonas vaginalis*, *Trypanosoma* spp, *Leishmania* spp

Ciliophora - *Balantidium coli*

Coccidian - *Isospora belli*, *Cryptosporidium parvum*, *Toxoplasma gondii*, *Plasmodium* species

Protozoan pathogens can also be grouped according to the location in the body where they most frequently cause disease.



**Table-1 Important pathogenic protozoa and commonly caused diseases.**

| Type and location | Species                                                                                                                                                                        | Disease                                                                                                                                  |
|-------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------|
| Intestinal tract  | <i>Entamoeba histolytica</i><br><i>Giardia lamblia</i><br><i>Cryptosporidium parvum</i><br><i>Balantidium coli</i><br><i>Isospora belli</i><br><i>Cyclospora cayentanensis</i> | Ambiasis<br>Giardiasis<br>Cryptosporidiosis<br>Balantidiasis<br>Isosporiosis<br>Cyclosporiasis                                           |
| Urogenital tract  | <i>Trichomonas vaginalis</i>                                                                                                                                                   | Trichomoniasis                                                                                                                           |
| Blood and tissue  | Plasmodium species<br><i>Toxoplasma gondii</i><br>Trypanasoma species<br>Leishmania species<br>Naegleria species<br>Acanthamoeba species<br>Babesia microti                    | Malaria<br>Toxoplasmosis<br>Trypanosomiasis<br>Leishmaniasis<br>Amoebic Meningoencephalitis<br>Amoebic Meningoencephalitis<br>Babesiosis |



## UNIT THREE

### AMOEBIASIS

#### INTRODUCTION

Amoebas primitive unicellular microorganisms with a relatively simple life cycle which can be divided into two stages:

- Trophozoite – actively motile feeding stage.
- Cyst – quiescent, resistant, infective stage.

Their reproduction is through binary fission, e.g. splitting of the trophozoite or through the development of numerous trophozoites within the mature multinucleated cyst. Motility is accomplished by extension of pseudopodia (“false foot”)

#### 1.1. *Entamoeba histolytica*

##### Morphological features

###### (a) Trophozoites

Viable trophozoites vary in size from about 10-60µm in diameter. Motility is rapid, progressive, and unidirectional, through pseudopods. The nucleus is characterized by evenly arranged chromatin on the nuclear membrane and the presence of a small, compact, centrally located karyosome. The cytoplasm is usually described as finely granular with few ingested bacteria or debris in vacuoles. In the case of dysentery, however, RBCs may be visible in the cytoplasm, and this feature is diagnostic for *E.histolytica*.

###### (b) Cyst

Cysts range in size from 10-20µm. The immature cyst has inclusions namely; glycogen mass and chromatoidal bars. As the cyst matures, the glycogen completely disappears; the chromatoidals may also be absent in the mature cyst.

## Life cycle

Intestinal infections occur through the ingestion of a mature quadrinucleate infective cyst, contaminated food or drink and also by hand to mouth contact.

It is then passed unaltered through the stomach, as the cyst wall is resistant to gastric juice.

In terminal ileum (with alkaline pH), excystation takes place.

Trophozoites being actively motile invade the tissues and ultimately lodge in the submucous layer of the large bowel. Here they grow and multiply by binary fission.

Trophozoites are responsible for producing lesions in amoebiasis.

Invasion of blood vessels leads to secondary extra intestinal lesions.

Gradually the effect of the parasite on the host is toned down together with concomitant increase in host tolerance, making it difficult for the parasite to continue its life cycle in the trophozoite phase.

A certain number of trophozoites come from tissues into lumen of bowel and are first transformed into pre-cyst forms.

Pre-cysts secrete a cyst wall and become a uninucleate cyst. Eventually, mature quadrinucleate cysts form. These are the infective forms.

Both mature and immature cysts may be passed in faeces. Immature cysts can mature in external environments and become infective.

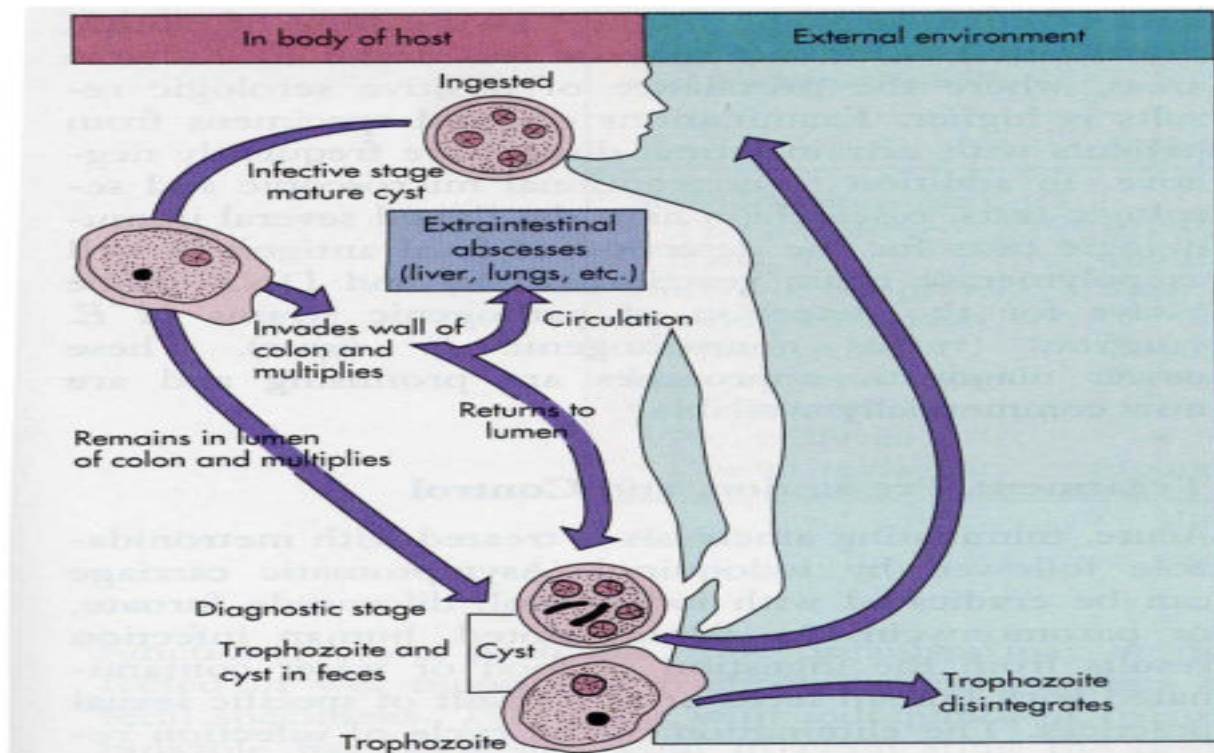


Figure-1 life cycle of *Entamoeba histolytica*

## Pathogenesis

Trophozoites divide and produce extensive local necrosis in the large intestine. Invasion into the deeper mucosa with extension into the peritoneal cavity may occur. This can lead to secondary involvement of other organs, primarily the liver but also the lungs, brain, and heart. Extraintestinal amebiasis is associated with trophozoites. Amoebas multiply rapidly in an anaerobic environment, because the trophozoites are killed by ambient oxygen concentration.

## Epidemiology

*E. histolytica* has a worldwide distribution. Although it is found in cold areas, the incidence is highest in tropical and subtropical regions that have poor sanitation and contaminated water. About 90% of infections are asymptomatic, and the remaining produces a spectrum of clinical syndrome. Patients infected with *E. histolytica* pass non-

infectious trophozoites and infectious cysts in their stools. Therefore, the main source of water and food contamination is the symptomatic carrier who passes cysts. Symptomatic amebiasis is usually sporadic. The epidemic form is a result of direct person-to-person faecal-oral spread under conditions of poor personal hygiene.

### **Clinical features**

The outcome of infection may result in a carrier state, intestinal amebiasis, or extraintestinal amebiasis. Diarrhoea, flatulence, and cramping are complaints of symptomatic patients. More severe disease is characterised by the passing of numerous bloody stools in a day. Systemic signs of infection (fever, leukocytosis, rigors) are present in patients with extraintestinal amebiasis. The liver is primarily involved, because trophozoites in the blood are removed from the blood by the portal veins. The right lobe is most commonly involved, thus pain over the liver with hepatomegaly and elevation of the diaphragm is observed.

### **Immunity**

*E.histolytica* elicits both the humeral and cellular immune responses, but it is not yet clearly defined whether it modulates the initial infection or prevents reinfection.

### **Laboratory diagnosis**

In intestinal amoebiasis:

- Examination of a fresh dysenteric faecal specimen or rectal scraping for trophozoite stage. (Motile amoebae containing red cells are diagnostic of amoebic dysentery).
- Examination of formed or semiformed faeces for cyst stage. (Cysts indicate infection with either a pathogenic *E.histolytica* or non-pathogenic *E.dispar*.)

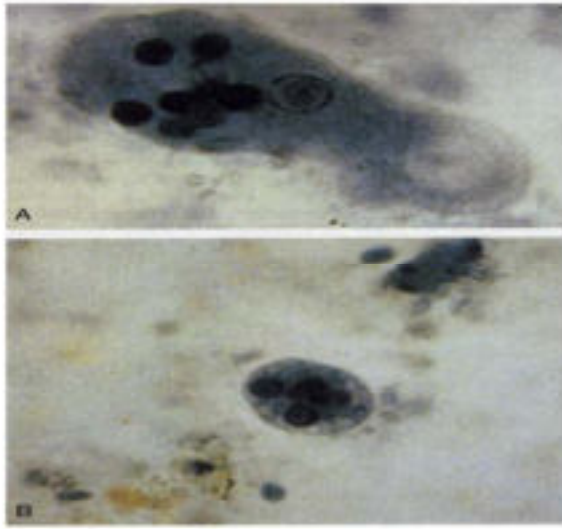


Figure 2-, *E.histolytica* trophozoite (A) *E. histolytica* Cyst (B)

### Extraintestinal amoebiasis

- Diagnosed by the use of scanning procedures for liver and other organs.
- Specific serologic tests, together with microscopic examination of the abscess material, can confirm the diagnosis.

### Treatment

Acute, fulminating amebiasis is treated with metronidazole followed by iodoquinol, and asymptomatic carriage can be eradicated with iodoquinol, diloxanide furoate, or paromomycin. The cysticidal agents are commonly recommended for asymptomatic carriers who handle food for public use.

Metronidazole, chloroquine, and diloxanide furoate can be used for the treatment of extra intestinal amoebiasis.

### Prevention

Introduction of adequate sanitation measures and education about the routes of transmission.

Avoid eating raw vegetables grown by sewerage irrigation and night soil



## 1.2. OTHER AMEBAE INHABITING THE ALIMENTARY CANAL

Most of these amoebae are commensal organisms that can parasitize the human gastrointestinal tract.

***Entamoeba hartmanni*** in all of its life-cycle stage, *E.hartmanni* resembles *E.histolytica* except in size, yet there is a slight overlap in the size range. The trophozoites do not ingest red blood cells, and their motility is generally less vigorous than that of *E.histolytica*. As in other amoebae, infection is acquired by ingestion of food or water contaminated with cyst-bearing faeces. Identification is based on examination of small amoebae in unstained or iodine-stained preparations. Usually no treatment is indicated, measures generally effective against faecal-borne infections will control this amoebic infection.

***Entamoeba coli*** the life cycle stages include; trophozoite, precyst, cyst, metacyst, and metacystic trophozoite. Typically the movements of trophozoites are sluggish, with broad short pseudopodia and little locomotion, but at a focus the living specimen cannot be distinguished from the active trophozoite of *E.histolytica*. However, the cysts are remarkably variable in size. *Entamoeba coli* is transmitted in its viable cystic stage through faecal contamination. *E.coli* as a lumen parasite is non-pathogenic and produces no symptoms. The mature cyst (with more than four nuclei) is the distinctive stage to differentiate *E.coli* from the pathogenic *E.histolytica*. Specific treatment is not indicated since this amoeba is non-pathogenic. The presence of *E.coli* in stool specimen is evidence for faecal contamination. Prevention depends on better personal hygiene and sanitary disposal of human excreta.

***Entamoeba polecki***- arelatively cosmopolitan parasite of hog and monkey. It can cause human disease but is rarely isolated. The disease is manifested as mild, transient diarrhoea. The diagnosis of *E.polecki* infection is confirmed by the microscopic detection of cysts in stool specimens. Treatment is the same as for *E.histolytica* infection. Prevention is achieved by good personal hygiene.

***Endolimax nana*** is a lumen dweller in the large intestine, primarily at the cecal level, where it feeds on bacteria. The life cycle is similar to *E.histolytica*. Motility is typically sluggish (slug-like) with blunt hyaline pseudopodia, Projects shortly. Human infection results from ingestion of viable cysts in polluted water or contaminated food. Typical ovoid cysts of *E.nana* are confirmative. Rounded cysts and living trophozoites are often confused with *E.hartmanni* and *E.histolytica*. No treatment is indicated for this non-pathogenic infection. Prevention can be achieved through personal cleanliness and community sanitation.

***Iodamoeba buetschlii***: - the natural habitat is the lumen of the large intestine, the principal site probably being the caecum. The trophozoite feeds on enteric bacteria; it is a natural parasite of man and lower primates. It is generally regarded as a non-pathogenic lumen parasite. No treatment is ordinarily indicated. Prevention is based on good personal hygiene and sanitation in the community.

***Entamoeba gingivalis*** - only the trophozoite stage presents, and encystation probably does not occur. *E.gingivalis* is a commensal, living primarily on exudate from the margins of the gums, and thrives best on unhealthy gums. No specific treatment is indicated. However the presence of *E.gingivalis* suggests a need for better oral hygiene. The infection can be prevented by proper care of the teeth and gums.

***Blastocystis hominis***- is an inhabitant of the human intestinal tract previously regarded as non-pathogenic yeast. Its pathogenecity remains controversial. The organism is found in stool specimen from asymptomatic people as well as from people with persistent diarrhoea. *B.hominis* is capable of pseudopodia extension and retraction, and reproduces by binary fission or sporulation. The classic form that is usually seen in the human stool specimen varies tremendously in size, from 6-40µm. There are thin – walled cysts involved in autoinfection, and thick–walled cysts responsible for external transmission via the faecal-oral route. The presence of large numbers of these parasites (five or more per oil immersion microscopic field) in the absence of other intestinal pathogens indicates disease. The organism may be detected in wet mounts or trichome –stained smears of faecal specimens. Treatment with iodoquinol or metronidazole has



been successful in eradicating the organism from intestine and alleviating symptoms. However, the definitive role of *B.hominis* in disease remains to be demonstrated. The incidence and apparent worldwide distribution of the infection indicates preventive measures to be taken, which involve improving personal hygiene and sanitary conditions.



**Table 2: Morphology of Trophozoites of intestinal Amoebae**

| Species                      | Size (diameter or length)                                                               | Motility                                                 | Nucleus                                      |                                                               |                                                                                    | Cytoplasm                |                                                                        |
|------------------------------|-----------------------------------------------------------------------------------------|----------------------------------------------------------|----------------------------------------------|---------------------------------------------------------------|------------------------------------------------------------------------------------|--------------------------|------------------------------------------------------------------------|
|                              |                                                                                         |                                                          | Number                                       | Peripheral Chromatin                                          | Karyosomal chromatin                                                               | Appearance               | Inclusions                                                             |
| <i>Entamoeba histolytica</i> | 10-60 $\mu$ m: usual range. 15-20 $\mu$ m- commensal form over 20 $\mu$ m-invasive form | Progressive with hyaline, finger-like pseudopods         | One: not visible in unstained preparations   | Fine granules: usually evenly distributed and uniform in size | Small, discrete: usually centrally located, but occasionally eccentrically located | Finely granular          | Erythrocytes occasionally: non-invasive organisms may contain bacteria |
| <i>Entamoeba hartmanni</i>   | 5-12 $\mu$ m: usual range, 8-10 $\mu$ m                                                 | Usually non progressive: may be progressive occasionally | One: not visible in unstained preparations   | Similar to E. histolytica                                     | Small, discrete, often eccentrically located                                       | Finely granular          | Bacteria                                                               |
| <i>Entamoeba coli</i>        | 15-50 $\mu$ m: usual range, 20-25 $\mu$ m                                               | Sluggish, non progressive, with blunt pseudopods         | One: often visible in unstained preparations | Coarse granules, irregular in size and distribution           | Large, discrete, usually eccentrically located                                     | Coarse, often vacuolated | Bacteria yeasts, other materials                                       |

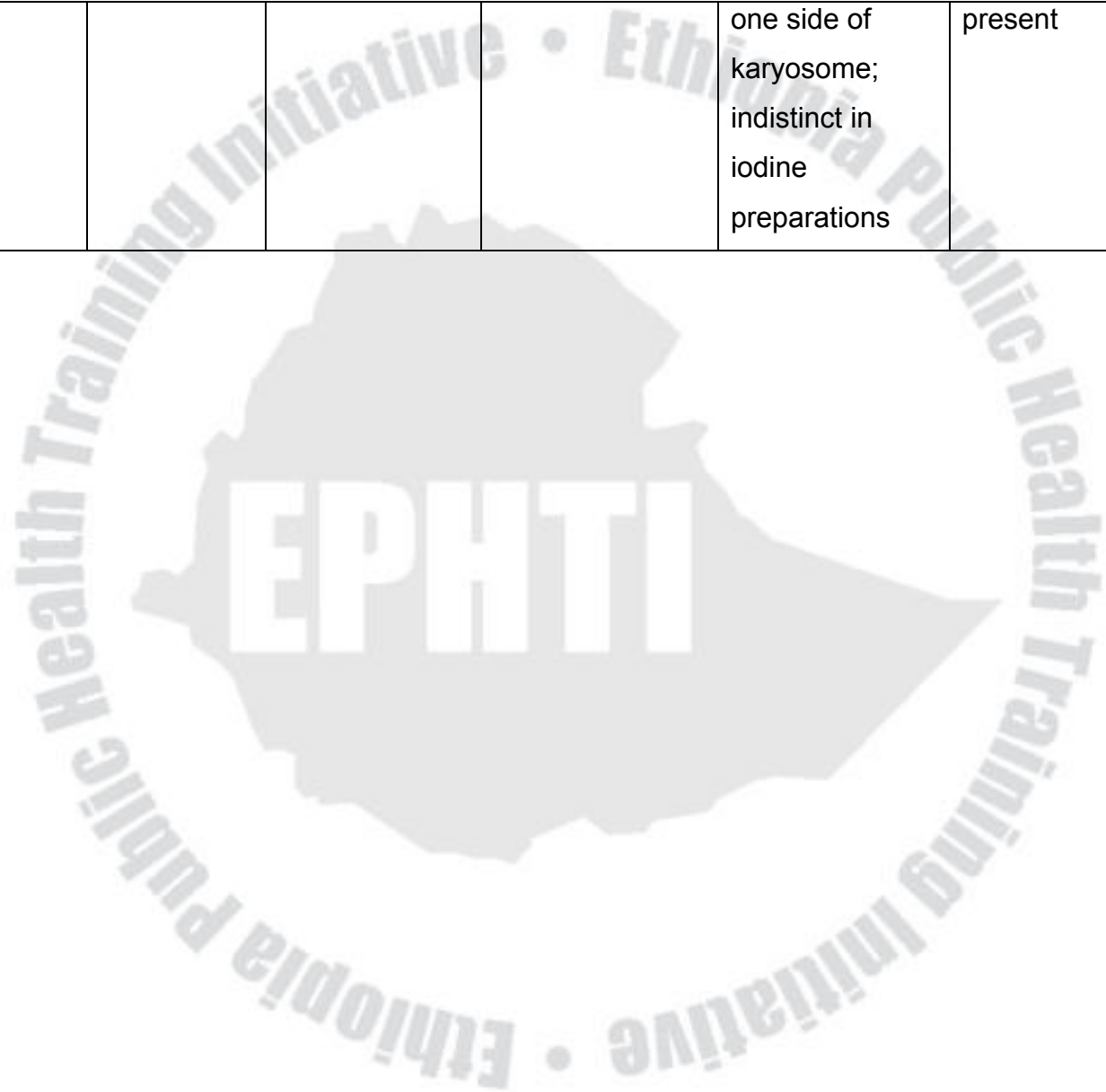
|                             |                                |                                                                                              |                                                                                                                       |                                                                                                                          |                                                                                                                                     |                                                    |                                     |
|-----------------------------|--------------------------------|----------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------|-------------------------------------|
| <i>Entamoeba ploecki</i>    | 10-25µm: usual range, 15 -20µm | Usually sluggish, similar to E.coli; occasionally in diarrheic specimens, may be progressive | One: may be slightly visible in unstained preparations: occasionally distorted by pressure from vacuoles in cytoplasm | Usually fine granules evenly distributed, occasionally irregularly arranged, chromatin sometimes in plaques or crescents | Small, discrete, eccentrically located: occasionally large, diffuse, or irregular                                                   | Coarsely granular, may resemble E.coli; vacuolated | Bacteria yeasts                     |
| <i>Endolimax nana</i>       | 6-12µm: usual range, 8 -10µm   | Sluggish, usually non- progressive, with blunt pseudopods                                    | One: visible occasionally in unstained preparations                                                                   | None                                                                                                                     | Large, irregularly shaped, blotlike                                                                                                 | Granular, vacuolated                               | Bacteria                            |
| <i>Iodamoeba buetschlii</i> | 8-20µm: usual range, 12 -15µm  | Sluggish usually non-progressive                                                             | One: not usually visible in unstained preparations                                                                    | None                                                                                                                     | Large, usually centrally located, surrounded by refractile, achromatic granules: granules often not distinct even in stained slides | Coarsely granular, vacuolated                      | Bacteria, yeasts, or other material |

**Table 3: Morphology of cysts of intestinal Amoebae**

| Species                      | Size                                        | Shape                                                | Nucleus                                                                      |                                                                                   |                                                                             | Cytoplasm                                                                                        |                                                                                           |
|------------------------------|---------------------------------------------|------------------------------------------------------|------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|
|                              |                                             |                                                      | Number                                                                       | Peripheral Chromatin                                                              | Karyosomal chromatin                                                        | Chromatoid bodies                                                                                | Glycogen and other features                                                               |
| <i>Entamoeba histolytica</i> | 10-20 $\mu$ m: usual range, 12 –15 $\mu$ m  | Usually spherical                                    | Four in mature cyst: immature cysts with 1 or 2 occasionally seen            | Peripheral chromatin present: fine, uniform granules, evenly distributed          | Small, discrete, usually centrally located                                  | Present: elongated bars with bluntly rounded ends                                                | Usually diffuse: concentrated mass often in young cysts; stains reddish brown with iodine |
| <i>Entamoeba histolytica</i> | 5-10 $\mu$ m: usual range, 6 –8 $\mu$ m     | Usually spherical:                                   | Four in mature cyst: immature cysts with 1 or 2 often seen                   | Similar to E. histolytica                                                         | Similar to E. histolytica                                                   | Present: elongated bars with bluntly rounded ends; may be rounded and grapelike                  | Similar to E. histolytica                                                                 |
| <i>Entamoeba coli</i>        | 10-35 $\mu$ m: usual range, 15 – 25 $\mu$ m | Usually spherical: sometimes oval, triangular, or of | Eight in mature cyst: occasionally, super nucleate cysts with 16 or more are | Peripheral chromatin present: coarse granules irregular in size and distribution, | Large, discrete, usually eccentrically, but occasionally centrally, located | Present, but less frequently seen than in E. histolytica; usually splinterlike with pointed ends | Usually diffuse, but occasionally well-defined mass in immature cysts; stains reddish     |

|                             |                                            |                                                    |                                                                   |                                                   |                                                                          |                                                                                                             |                                                                                                  |
|-----------------------------|--------------------------------------------|----------------------------------------------------|-------------------------------------------------------------------|---------------------------------------------------|--------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------|
|                             |                                            | another shape                                      | rarely seen; immature cysts with 2 or more occasionally seen      | but often appear more uniform than in trophozoite |                                                                          |                                                                                                             | brown with iodine                                                                                |
| <i>Endolimas nana</i>       | 5-10 $\mu$ m: usual range, 6 -8 $\mu$ m    | Spherical, ovoid, or ellipsoidal                   | Four in mature cysts: immature cysts with less than 4 rarely seen | None                                              | Large (blotlike), usually centrally located                              | Granules occasionally or small oval masses present, but bodies as seen in Entamoeba species are not present | Usually diffuse; concentrated mass occasionally in young cysts; stains reddish brown with iodine |
| <i>Iodamoeba buetschlii</i> | 5-20 $\mu$ m: usual range, 10 – 12 $\mu$ m | Ovoid ellipsoidal, triangular, or of another shape | One in mature cyst                                                | None                                              | Large, usually eccentrically located; refractile, achromatic granules on | Granules occasionally present, but bodies as seen in Entamoeba species are not                              | Compact, well-defined mass; stains dark brown with iodine                                        |

|  |  |  |  |                                                                      |         |  |
|--|--|--|--|----------------------------------------------------------------------|---------|--|
|  |  |  |  | one side of<br>karyosome;<br>indistinct in<br>iodine<br>preparations | present |  |
|--|--|--|--|----------------------------------------------------------------------|---------|--|



### 1.3. PATHOGENIC FREE-LIVING AMOEBAE

Among the numerous free-living amoebae of soil and water habitats, certain species of *Naegleria*, *Acanthamoeba* and *Balamuthia* are facultative parasites of man. Most human infections of these amoebae are acquired by exposure to contaminated water while swimming. Inhalation of cysts from dust may account for some infections.

***Naegleria fowleri***- the trophozoites occur in two forms. Amoeboid forms with single pseudopodia and flagella forms with two flagella which usually appear a few hours after flooding water or in CSF.

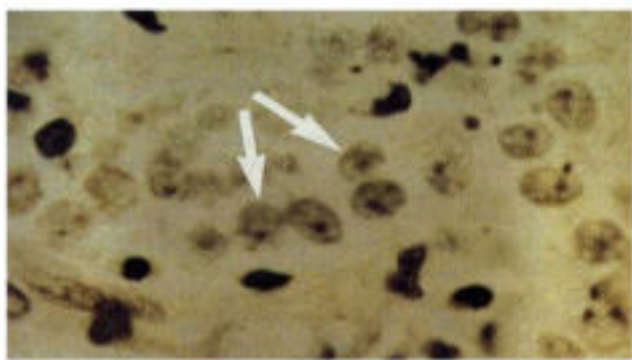


Figure 3. *Naegleria* trophozoites in a section of spinal cord from a patient with amoebic meningoencephalitis

***Acanthamoeba species***- the trophozoites have an irregular appearance with spine-like pseudopodia, and acanthopodia.

***Balamuthia species***- the trophozoite extends a broad, flat lamellipodia or sub pseudopodia from it. The trophozoite may be bi-nucleated. Unlike most amoebae the nuclear envelope breaks down during mitosis.

*Naegleria*, *Acanthamoeba*, *Balamuthia* organisms are opportunistic pathogens. *Naegleria fowleri* causes acute primary amoebic meningoencephalitis. *Acanthamoeba* & *Balamuthia* organisms are responsible for granulomatous amoebic encephalitis and single or multiple brain abscesses, primarily in immunocompromised individuals. Keratitis (eye) and skin infection by *Acanthamoeba* may also occur. For the diagnosis of *Naegleria*, *Acanthamoeba*, and *Balamuthia* infections, specimens of nasal



discharge and cerebrospinal fluid; and in cases of eye infections corneal scraping should be collected. The clinical specimen can be examined with saline wet-preparation and Iodine stained smear. Treatment of free-living amoebic infections is largely ineffective. These infections are rare in Ethiopia.



## UNIT FOUR

### PATHOGENIC FLAGELLATES

#### INTRODUCTION

Flagellates are unicellular microorganisms. Their locomotion is by lashing a tail-like appendage called a flagellum or flagella and reproduction is by simple binary fission.

There are three groups of flagellates:

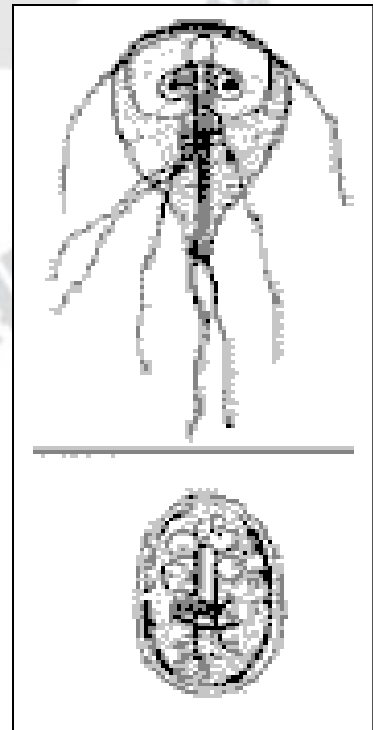
- Luminal flagellates
  - Giardia lamblia*
  - Dientamoeba fragilis*
- Hemoflagellates
  - Trypanosoma species.
  - Leishmania species.
- Genital flagellates
  - Trichomonas vaginalis*

#### 2.1. Luminal flagellates

##### 2.1.1. Giardia lamblia

**Important features** – the life cycle consists of two stages, the trophozoite and cyst. The trophozoite is 9-12  $\mu\text{m}$  long and 5-15  $\mu\text{m}$  wide anteriorly. It is bilaterally symmetrical, pear-shaped with two nuclei (large central karyosome), four pairs of flagella, two axonemes, and a suction disc with which it attaches to the intestinal wall. The oval cyst is 8-12  $\mu\text{m}$  long and 7-10  $\mu\text{m}$  wide, thick-walled with four nuclei and several internal fibers. Each cyst gives rise to two trophozoites during excystation in the intestinal tract.

Transmission is by ingestion of the infective cyst.



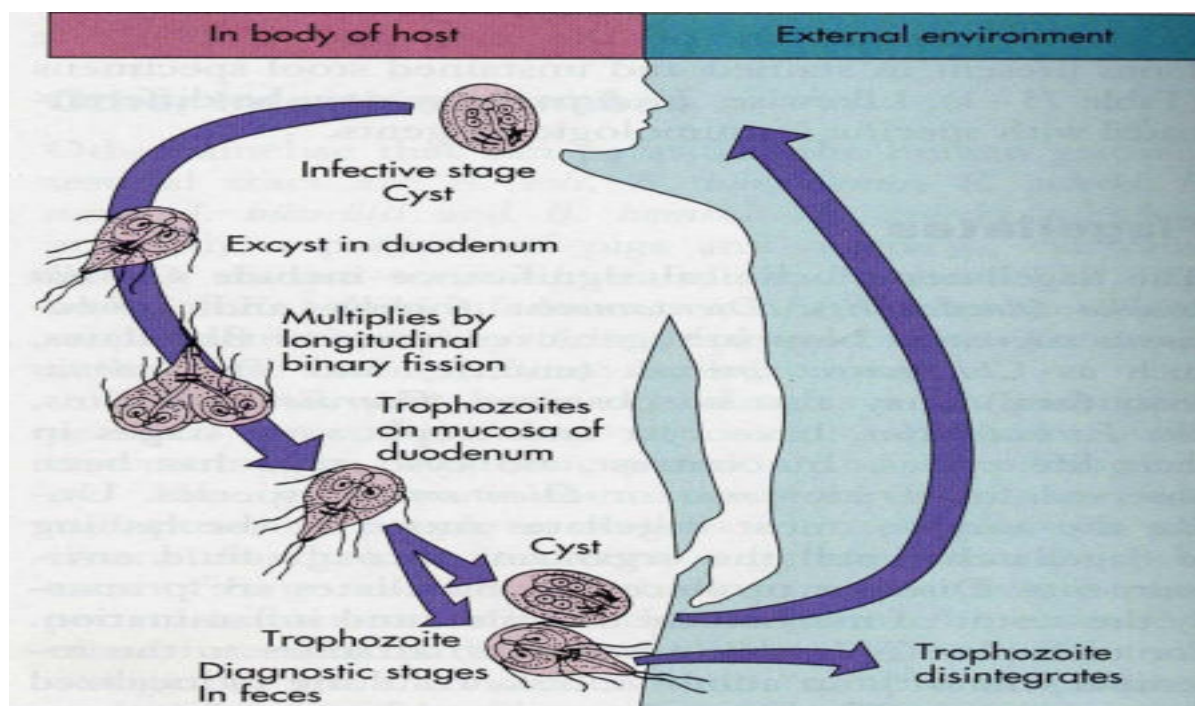


Figure 4; Life cycle of *Giardia lamblia*.

## Pathogenesis

Infection with *G.lamblia* is initiated by ingestion of cysts. Gastric acid stimulates excystation, with the release of trophozoites in duodenum and jejunum. The trophozoites can attach to the intestinal villi by the ventral sucking discs without penetration of the mucosa lining, but they only feed on the mucous secretions. In symptomatic patients, however, mucosa-lining irritation may cause increased mucous secretion and dehydration. Metastatic spread of disease beyond the GIT is very rare.

## Epidemiology

*Giardia lamblia* has a worldwide distribution, particularly common in the tropics and subtropics. It is acquired through the consumption of inadequately treated contaminated water, ingestion of contaminated uncooked vegetables or fruits, or person-to-person spread by the faecal-oral route. The cyst stage is resistant to chlorine in concentrations used in most water treatment facilities. Infection exists in 50% of symptomatic carriage, and reserves the infection in endemic form.

## **Clinical features**

### *Clinical disease:* Giardiasis

Symptomatic giardiasis ranges from mild diarrhea to severe malabsorption syndrome. Usually, the onset of the disease is sudden and consists of foul smelling, watery diarrhea, abdominal cramps, flatulence, and streatorrhoea. Blood & pus are rarely present in stool specimens, a feature consistent with the absence of tissue destruction.

## **Immunity**

The humoral immune response and the cellular immune mechanism are involved in giardiasis. Giardia – specific IgA is particularly important in both defense against and clearance of parasite.

## **Laboratory diagnosis**

Examination of diarrhoeal stool- trophozoite or cyst, or both may be recovered in wet preparation. In examinations of formed stool (e.g. in asymptomatic carriers) only cysts are seen. Giardia species may occur in “showers”, i.e. many organisms may be present in the stool on a given day and few or none may be detected the next day. Therefore one stool specimen per day for 3 days is important.

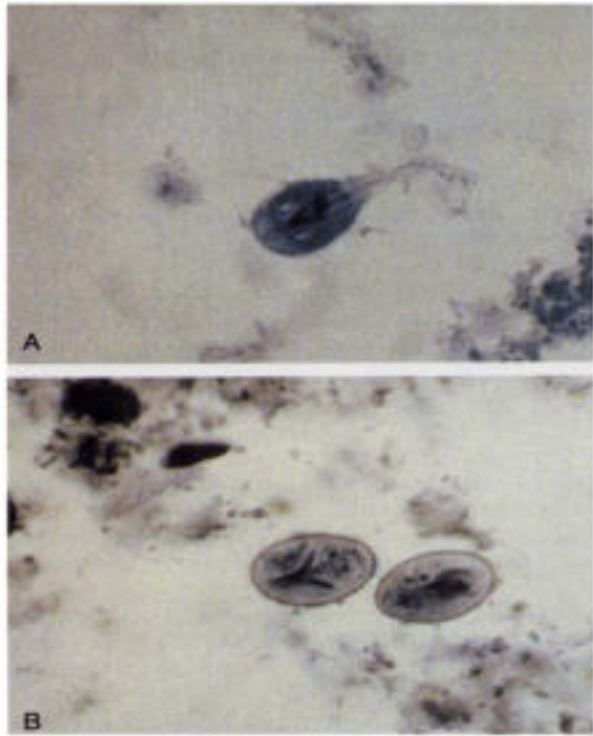


Figure 5; *Giardia lamblia* trophozoite (A), cyst (B)

If microscopic examination of the stool is negative in a patient in whom giardiasis is highly suspected duodenal aspiration, string test (entero-test), or biopsy of the upper small intestine can be examined.

In addition to conventional microscopy, several immunologic tests can be implemented for the detection of parasitic antigens.

### **Treatment**

For asymptomatic carriers and diseased patients the drug of choice is quinacrine hydrochloride or metronidazole.

### **Prevention**

- Asymptomatic reservoirs of infection should be identified & treated.
- Avoidance of contaminated food and water.
- Drinking water from lakes and streams should be boiled, filtered and/or iodine-treated.
- Proper waste disposal and use of latrine.



### 2.1.2. *Trichomonas vaginalis*

**Important features-** it is a pear-shaped organism with a central nucleus and four anterior flagella; and undulating membrane extends about two-thirds of its length. It exists only as a trophozoite form, and measured 7-23µm long & 5-15µm wide. Transmission is by sexual intercourse.

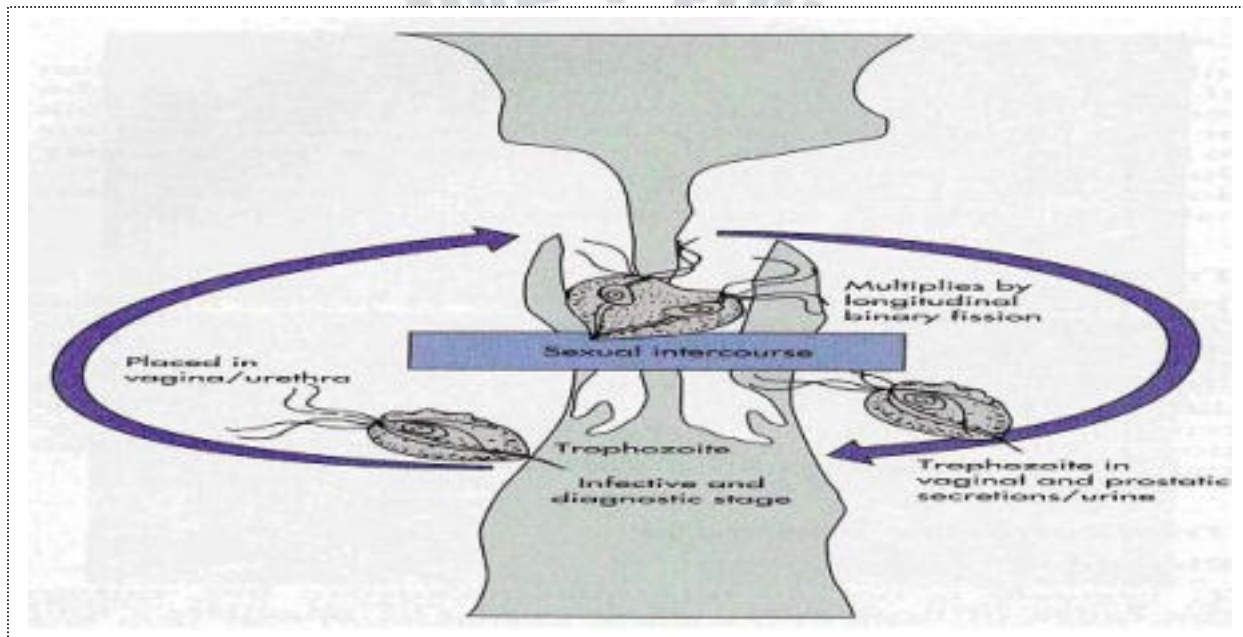


Figure 6; Life cycle of *Trichomonas vaginalis*

### Pathogenesis

The trophozoite is found in the urethra & vagina of women and the urethra & prostate gland of men. After introduction by sexual intercourse, proliferation begins which results in inflammation & large numbers of trophozoites in the tissues and the secretions. The onset of symptoms such as vaginal or vulval pruritus and discharge is often sudden and occurs during or after menstruation as a result of the increased vaginal acidity. The vaginal secretions are liquors, greenish or yellowish, sometimes frothy, and foul smelling. Infection in the male may be latent, with no symptoms, or may be present as self limited, persistent, or recurring urethritis.

## **Epidemiology**

This parasite has worldwide distribution, and sexual intercourse is the primary mode of transmission. Occasionally, infections can be transmitted by fomites (toilet articles, clothing), although this transmission is limited by liability of the trophozoite. Rarely Infants may be infected by passage through the mother's infected birth canal. The prevalence of this flagellate in developing countries is reported to be 5% –20% in women and 2% –10% in men.

## **Clinical features**

*Clinical disease* - trichomoniasis.

Most infected women at the acute stage are asymptomatic or have a scanty, watery vaginal discharge. In symptomatic cases vaginitis occurs with more extensive inflammation, along with erosion of epithelial lining, and painful urination, and results in symptomatic vaginal discharge, vulvitis and dysuria.

## **Immunity**

The infection may induce humoral, secretory, and cellular immune reactions, but they are of little diagnostic help and do not appear to produce clinically significant immunity.

## **Laboratory diagnosis**

- In females, *T.vaginalis* may be found in urine sediment, wet preparations of vaginal secretions or vaginal scrapings.
- In males it may be found in urine, wet preparations of prostatic secretions or following massage of the prostate gland.
- Contamination of the specimen with faeces may confuse *T.vaginalis* with *T.hominis*.



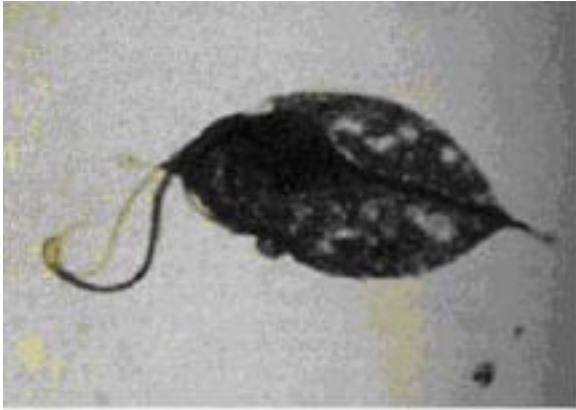


Figure 7; *Trichomonas vaginalis*

### Treatment

Metronidazole is the drug of choice. If resistant cases occur, re-treatment with higher doses is required.

### Prevention

- Both male & female sex partners must be treated to avoid reinfection
- Good personal hygiene, avoidance of shared toilet articles & clothing.
- Safe sexual practice.

#### 2.1.3. *Dientamoeba fragilis*

*Dientamoeba fragilis* was initially classified as an amoeba; however, the internal structures of the trophozoite are typical of a flagellate. No cyst stage has been described. The life cycle and mode of transmission of *D. fragilis* are not known. It has worldwide distribution. The transmission is postulated, via helminthes egg such as those of *Ascaris* and *Enterobius* species. Transmission by faecal- oral routes does occur. Most infection with *D. fragilis* is asymptomatic, with colonization of the cecum and upper colon. However, some patients may develop symptomatic disease, consisting of abdominal discomfort, flatulence, intermittent diarrhea, anorexia, and weight loss. The therapeutic agent of choice for this infection is iodoquinol, with tetracycline and

paramomycine as acceptable alternatives. The reservoir for this flagellate and lifecycle are unknown. Thus, specific recommendation for prevention is difficult. However, infection can be avoided by maintenance of adequate sanitary conditions.

#### 2.1.4. Other flagellates inhabiting the alimentary canal

**Trichomonas hominis** – The trophozoites live in the caecal area of the large intestine and feed on bacteria. It is considered to be non-pathogenic, although it is often recovered from diarrheic stools. Since there is no known cyst stage, transmission probably occurs in the trophic form. There is no indication of treatment.

**Trichomonas tenax** – was first recovered from the mouth, specifically in tartar from the teeth. There is no known cyst stage. The trophozoite has a pyriform shape and is smaller and more slender than that of *T.hominis*. Diagnosis is based on the recovery of the organism from the teeth, gums, or tonsillar crypts, and no therapy is indicated.

**Chilomastix mesnli** – has both a trophozoite and cyst stage. It normally lives in the cecal region of the large intestine, where the organism feeds on bacteria and debris. It is considered to be a non-pathogenic, and no treatment is recommended.

**Table 4: Morphology of Trophozoites of intestinal Flagellates**

| Species                      | Length                         | Shape                                                                                      | Motility       | Number of Nuclei                                                                                                  | Number of Flagella        | Other features                                                                                                                                                                              |
|------------------------------|--------------------------------|--------------------------------------------------------------------------------------------|----------------|-------------------------------------------------------------------------------------------------------------------|---------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <i>Dientamoeba fragilis</i>  | 5-15µm: usual range, 9 - 12µm  | Ameboid; pseudopodia are angular, serrated, or broad-lobed and hyaline, almost transparent | Sluggish       | 1 or 2; in approximately 40% of organisms only 1 nucleus is present; nuclei not visible in unstained preparations | None                      | Karyosome usually in form of cluster of 4-8 granules; no peripheral chromatin; cytoplasm is finely granular, vacuolated, and may contain bacteria; organism formerly classified as an ameba |
| <i>Trichomonas hominis</i>   | 8-20µm: usual range, 11 - 12µm | Pear-shaped                                                                                | Rapid, jerking | 1; not visible in unstained mounts                                                                                | 3-5 anterior; 1 posterior | Undulating membrane extending length of body                                                                                                                                                |
| <i>Trichomonas vaginalis</i> | 7-23µm: usual range, 10 - 15µm | Pear-shaped                                                                                | Rapid, jerking | 1; not visible in unstained mounts                                                                                | 3-5 anterior; 1 posterior | Undulating membrane extends ½ length of body; no free posterior flagellum; does not live in intestinal                                                                                      |

|                            |                                |             |                 |                                    |                                |                                                                                                                  |
|----------------------------|--------------------------------|-------------|-----------------|------------------------------------|--------------------------------|------------------------------------------------------------------------------------------------------------------|
|                            |                                |             |                 |                                    |                                | tract; seen In vaginal smears and urethral discharges                                                            |
| <i>Chilomastix mesnili</i> | 6-24µm: usual range, 10 -15µm  | Pear-shaped | Stiff, rotary   | 1; not visible in unstained mounts | 3 anterior; 1 cytostome        | Prominent cytostome extending $\frac{1}{3}$ – $\frac{1}{2}$ length of body; spiral groove across ventral surface |
| <i>Giardia lamblia</i>     | 10-20µm: usual range, 12 –15µm | Pear-shaped | “ Falling leaf” | 2;not visible in unstained mounts  | 4 lateral; 2 ventral; 2 caudal | Sucking disk occupying $\frac{1}{2}$ - $\frac{3}{4}$ of ventral surface                                          |

## 2.2. Haemoflagelates

### 2.2.1. Leishmania Species

- Clinical disease**
- Visceral leishmaniasis
  - Cutaneous leishmaniasis
  - Mucocutaneous leishmaniasis

The species of leishmania exist in two forms, amastigote (aflagellar) and promastigote (flagellated) in their life cycle. They are transmitted by certain species of sand flies (Phlebotomus & Lutzomyia)

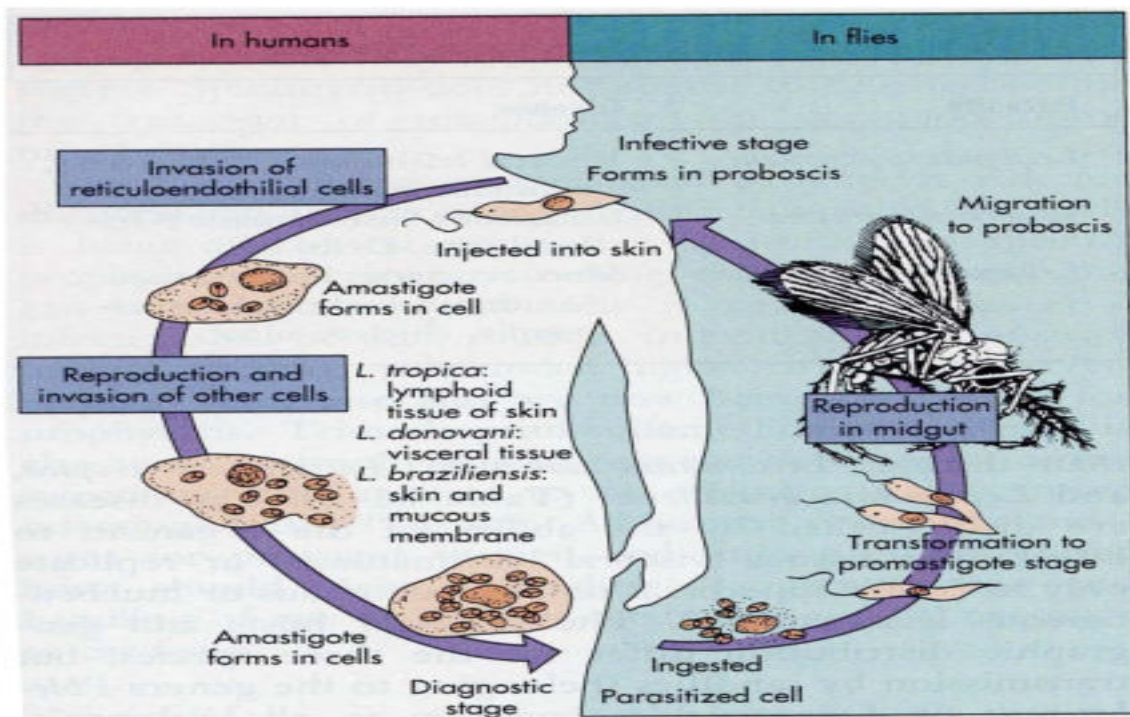


Figure 8; Life cycle of *Leishmania* species

#### 2.2.1.1. Visceral leishmaniasis

##### *Leishmania donovani*

**Important features-** the natural habitat of *L.donovani* in man is the reticuloendothelial system of the viscera, in which the amastigote multiplies by

simple binary fission until the host cells are destroyed, whereupon new macrophages are parasitized. In the digestive tract of appropriate insects, the developmental cycle is also simple by longitudinal fission of promastigote forms. The amastigote stage appears as an ovoidal or rounded body, measuring about 2-3µm in length; and the promastigotes are 15-25µm lengths by 1.5-3.5µm breadths.

### **Pathogenesis**

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anaemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anaemia and lymphadenopathy also occurs. Increased production of globulin results in hyperglobulinemia, and reversal of the albumin-to-globulin ratio.

### **Epidemiology**

*L. donovani donovani*, infection of the classic kala-azar ("black sickness") or dum-dum fever type occurs in many parts of Asia, Africa and Southeast Asia. Kala-azar occurs in three distinct epidemiologic patterns. In Mediterranean basin (European, Near Eastern, and Africa) and parts of China and Russia, the reservoir hosts are primarily dogs & foxes; in sub-Saharan Africa, rats & small carnivores are believed to be the main reservoirs. In India and neighboring countries (and Kenya), kala-azar is anthroponosis, i.e. there is no other mammalian reservoir host other than human. The vector is the *Phlebotomus* sand fly. Other variants of *L. donovani* are also recognized: *L. donovani infantum* with similar geographical distribution, reservoir host and vector; with *L. donovani donovani*. *L. donovani chagasi* is found in South America, Central America, especially Mexico, and the West Indies. Reservoir hosts are dogs, foxes, and cats, and the vector is the *Lutzomyia* sand fly.



### **Clinical features**

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate & invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia, and emaciation occurs. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis, occurs.

Untreated visceral leishmaniasis is nearly always fatal as a result of secondary infection.

### **Immunity**

Host cellular and humoral defence mechanisms are stimulated.

### **Laboratory diagnosis**

- Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).
- The amastigotes appear as intracellular & extra cellular L. donovan (LD) bodies.

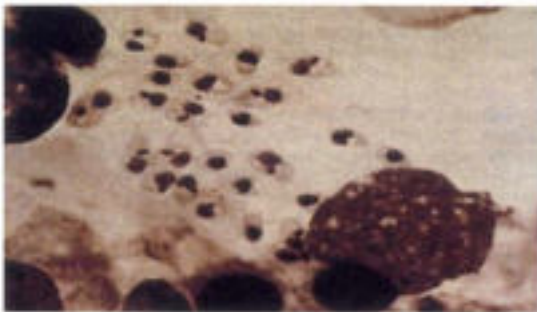


Figure 9; Giemsa-stained amastigotes (LD bodies)

- Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.
- Serologic testing is also available.



## **Treatment**

The drug of choice is sodium stibogluconate, a pentavalent antimonial compound.

Alternative approaches include the addition of allopurinol and the use of pentamidine or amphotercin B.

## **Prevention**

- Prompt treatment of human infections and control of reservoir hosts.
- Protection from sand flies by screening and insect repellents.

### **2.2.1.2. Old World Cutaneous Leishmaniasis (Oriental sore)**

#### **Clinical disease**

*L.tropica minor* - dry or urban cutaneous leishmaniasis

*L.tropica major* - wet or rural cutaneous leishmaniasis

*L.aethiopica* - cutaneous leishmaniasis

#### **Important features**

These are parasites of the skin found in endothelial cells of the capillaries of the infected site, nearby lymph nodes, within large mononuclear cells, in neutrophilic leukocytes, and free in the serum exuding from the ulcerative site. Metastasis to other site or invasion of the viscera is rare.

#### **Pathogenesis**

In neutrophilic leukocytes, phagocytosis is usually successful, but in macrophages the introduced parasites round up to form amastigote and multiply. In the early stage, the lesion is characterized by the proliferation of macrophages that contain numerous amastigotes. There is a variable infiltration of lymphocytes and plasma cell. The overlying epithelium shows acanthosis and hyperkeratosis, which is usually followed by necrosis and ulceration.

## **Epidemiology**

Cutaneous leishmaniasis produced by *L.tropica* complex is present in many parts of Asia, Africa, Mediterranean Europe and the southern region of the former Soviet Union. The urban Cutaneous leishmaniasis is thought to be an anthroponosis while the rural cutaneous leishmaniasis is zoonosis with human infections occurring only sporadically. The reservoir hosts in *L. major* are rodents. *L.aethiopica* is endemic in Ethiopia and Kenya. The disease is a zoonosis with rock & tree hyraxes serving as reservoir hosts. The vector for the old world cutaneous leishmaniasis is the Phlebotomus sand fly.

## **Clinical features**

The first sign, a red papule, appears at the site of the fly's bite. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate. Gradually the ulcer becomes hard and crusted and exudes a thin, serous material. At this stage, secondary bacterial infection may complicate the disease. In the case of the Ethiopian cutaneous leishmaniasis, there are similar developments of lesions, but they may also give rise to diffuse cutaneous leishmaniasis (DCL) in patients who produce little or no cell mediated immunity against the parasite. This leads to the formation of disfiguring nodules over the surface of the body.

## **Immunity**

Both humoral and cell mediated immunity (CMI) are involved

## **Treatment**

The drug of choice is sodium stibogluconate, with an alternative treatment of applying heat directly to the lesion. Treatment of *L.aethiopica* remains to be a problem as there is no safe and effective drug.

## **Prevention**

- Prompt treatment & eradication of ulcers
- Control of sand flies & reservoir hosts.

### **2.2.1.3. New World Cutaneous and Mucocutaneous Leishmaniasis (American cutaneous leishmaniasis)**

#### **Clinical disease:**

*Leishmania mexicana* complex- Cutaneous leishmaniasis.

*Leishmania braziliensis* complex- mucocutaneous or cutaneous leishmaniasis

#### **Important features:**

The American cutaneous leishmaniasis is the same as oriental sore. But some of the strains tend to invade the mucous membranes of the mouth, nose, pharynx, and larynx either initially by direct extension or by metastasis. The metastasis is usually via lymphatic channels but occasionally may be the bloodstream.

#### **Pathogenesis**

The lesions are confined to the skin in cutaneous leishmaniasis and to the mucous membranes, cartilage, and skin in mucocutaneous leishmaniasis. A granulomatous response occurs, and a necrotic ulcer forms at the bite site. The lesions tend to become superinfected with bacteria. Secondary lesions occur on the skin as well as in mucous membranes. Nasal, oral, and pharyngeal lesions may be polypoid initially, and then erode to form ulcers that expand to destroy the soft tissue and cartilage about the face and larynx. Regional lymphadenopathy is common.

#### **Epidemiology**

Most of the cutaneous & mucocutaneous leishmaniasis of the new world exist in enzootic cycles of infection involving wild animals, especially forest rodents. *Leishmania mexicana* occurs in south & Central America, especially in the Amazon

basin, with sloths, rodents, monkeys, and raccoons as reservoir hosts. The mucocutaneous leishmaniasis is seen from the Yucatan peninsula into Central & South America, especially in rain forests where workers are exposed to sand fly bites while invading the habitat of the forest rodents. There are many jungle reservoir hosts, and domesticated dogs serve as reservoirs as well. The vector is the *Lutzomyia* sand fly.

### **Clinical features**

The types of lesions are more varied than those of oriental sore and include Chiclero ulcer, Uta, Espundia, and Disseminated Cutaneous Leishmaniasis.

### **Laboratory diagnosis**

- Demonstration of the amastigotes in properly stained smears from touch preparations of ulcer biopsy specimen.
- Serological tests based on fluorescent antibody tests.
- Leishman skin test in some species.

### **Immunity**

The humoral and cellular immune systems are involved

### **Treatment**

The drug of choice is sodium stibogluconate.

### **Prevention**

- Avoiding endemic areas especially during times when local vectors are most active.
- Prompt treatment of infected individuals.

## **2.2.2. Trypanosomiasis**

### **Etiologic agents**

*Trypanosoma brucei complex* – African trypanosomiasis (sleeping sickness)

*Trypanosoma cruzi* – American trypanosomiasis (Chagas' disease)

## Important features

These species may have amastigote, promastigote, epimastigote, and trypomastigote stages in their life cycle. In human trypanosomes of the African form, however, the amastigote and promastigote stages of development are absent. Typical trypanosome structure is an elongated spindle-shaped body that more or less tapers at both ends, a centrally situated nucleus, a kinetoplast posterior to nucleus, an undulating membrane arising from the kinetoplast and proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end.

### 2.2.2.1. African trypanosomiasis

*Trypanosoma gambiense* & *Trypanosoma rhodesiense* are causative agents of the African trypanosomiasis, transmitted by insect bites. The vector for both is the tsetse fly.

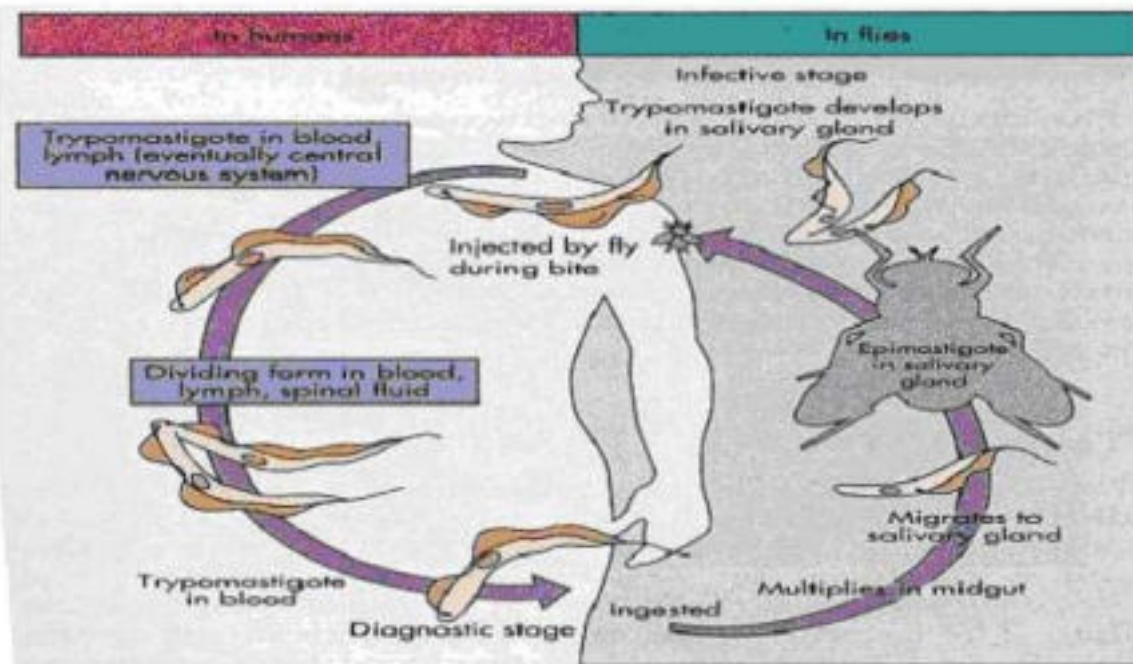


Figure 10; Life cycle of *Trypanosoma brucei*



## Pathogenesis

The trypomastigotes spread from the skin through the blood to the lymph node and the brain. The typical somnolence (sleeping sickness) usually progresses to coma as a result of demyelinating encephalitis. In acute form, cyclical fever spike (approximately every 2 weeks) occurs that is related to antigenic variation. As antibody mediated agglutination and lysis of the trypomastigotes occurs, the fever subsides. With a few remains of antigenic variants new fever spike occurs and the cycle repeats itself over a long period.

## Epidemiology

*T.burcei gambiense* is limited to tropical west and central Africa, correlating with the range of the tsetse fly vector. The tsetse flies transmitting *T.b. gambiense* prefer shaded stream banks for reproduction and proximity to human dwellings. People who work in such areas are at greatest risk of infection. An animal reservoir has not been proved for this infection.

*T.burcei rhodeseinse* is found primarily in East Africa, especially the cattle-raising countries, where tsetse flies breed in the brush rather than along stream banks. *T.b. rhodeseines* also differs from *T.b. gambiense* in that domestic animal hosts (cattle and sheep) and wild game animals act as reservoir hosts. This transmission and vector cycle makes the organism more difficult to control than *T.b. gambiense*.

## Clinical features

Although both species cause sleeping sickness, the progress of the disease is different. *T.gambiense* induced disease runs a low-grade chronic course over a few years. One of the earliest signs of disease is an occasional ulcer at the site of the fly bite. As reproduction of organisms continues, the lymph nodes are invaded, and fever, myalgia, arthralgia, and lymph node enlargement results. Swelling of



the posterior cervical lymph nodes is characteristic of Gambian sleeping sickness and is called winterbottom's sign.

Chronic disease progresses to CNS involvement with lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration. In the final stages, convulsions, hemiplegia, and incontinence occur. The patient becomes difficult to arouse or obtain a response from, eventually progressing to a comatose state. Death is the result of CNS damage and other infections, such as pneumonia.

In *T. rhodesiense*, the disease caused is a more acute, rapidly progressive disease that is usually fatal. This more virulent organism also develops in greater numbers in the blood. Lymphadenopathy is uncommon, and early in the infection, CNS invasion occurs, resulting in lethargy, anorexia, and mental disturbance. The chronic stages described for *T. gambiense* are not often seen, because in addition to rapid CNS disease, the organism produces kidney damage & myocarditis, leading to death.

### **Immunity**

Both the humoral and cellular immunity involve in these infections. The immune responses of the host to the presence of these parasites, however, is faced with antigenic variation, in which organisms that have changed their antigenic identity can escape the host immune response and initiate another disease process with increased level of parasitemia.

### **Laboratory**

Examination of thin and thick films, in concentrated anticoagulated blood preparations, and in aspiration from lymph nodes and concentrated spinal fluid.

Methods for concentrating parasites in blood may be helpful approaches including centrifugation of heparinized samples and an ion-exchange chromatography.

Levels of parasitosis vary widely, and several attempts to visualize the organism over a number of days may be necessary.



Figure 11; Trypomastigote stage of *Trypanosoma burcei* complex

### **Treatment**

The same treatment protocol is applied for these parasites. For the acute stages of the disease the drug of choice is suramin with pentamidine as an alternative. In chronic disease with CNS involvement, the drug of choice is melarsoprol. Alternatives include trypars amide combined with suramin.

### **Prevention**

- Control of breeding sites of tsetse flies and use of insecticides.
- Treatment of human cases to reduce transmission to flies.
- Avoiding insect bite by wearing protective clothing & use of screen, bed netting and insect repellants.

#### **2.2.2.2 American trypanosomiasis**

*Trypanosoma cruzi* is a pleomorphic trypanosome that includes an additional form of amastigote in its life cycle. The vector for transmission are reduviid bugs.

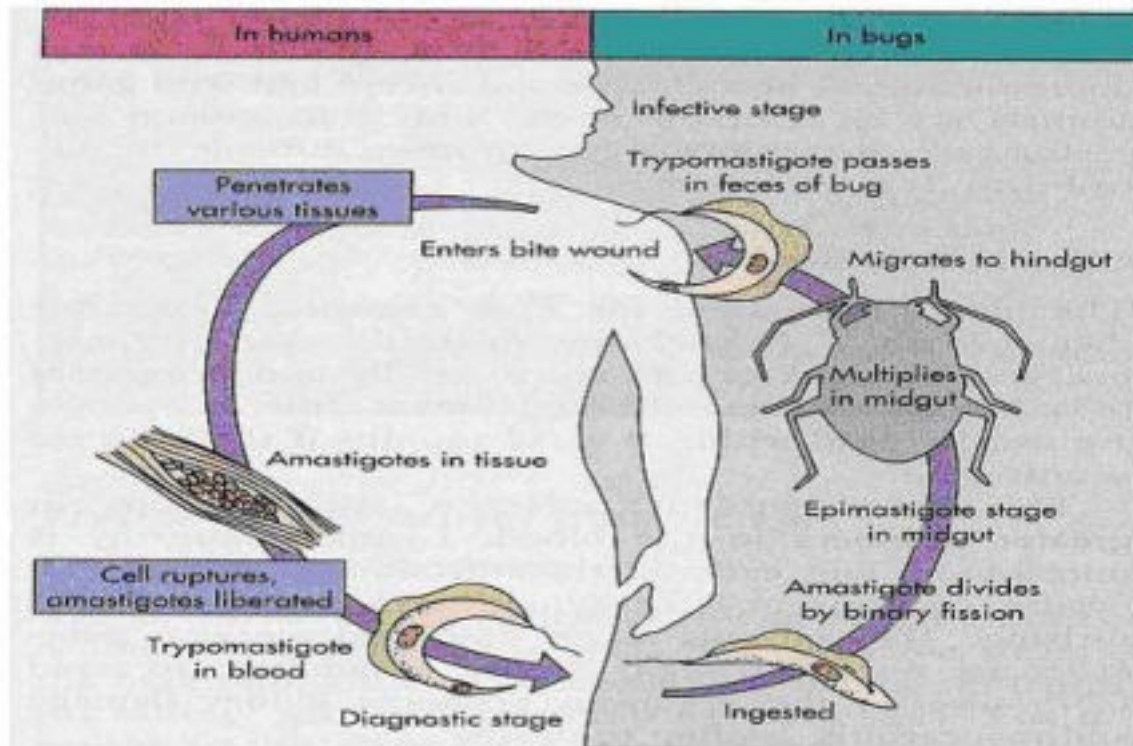


Figure 12; Life cycle of *Trypanosoma cruzi*

### Pathogenesis

During the acute phase, the organism occurs in blood as a typical trypomastigote and in the reticuloendothelial cells as a typical amastigote. The amastigotes can kill cells and cause inflammation, consisting mainly of mononuclear cells. Cardiac muscle is the most frequently and severely affected tissue. In addition, neuronal damage leads to cardiac arrhythmias and loss of tone in the colon (megacolon) and esophagus (megaesophagus). In the chronic phase, the organism persists in the amastigote form.

### Epidemiology

*T. cruzi* occurs widely in both reduviid bugs and a broad spectrum of reservoir animals in North, Central, and South America. Human disease is found most often among children in South and Central America, where there is direct correlation

between infected wild animal reservoir hosts and the presence of infected bugs whose nests are found in human dwellings.

### **Clinical features**

Chagas' disease may be asymptomatic acute or chronic disease. One of the earliest signs is development at the site of the bug bite of an erythematous and indurated area called a chagoma. This is often followed by a rash and edema around the eyes and face; in young children frequently an acute process with CNS involvement may occur. Acute infection is also characterized by fever, chills, malaise, myalgia, and fatigue. The chronic Chagas' disease is characterized by hepatosplenomegaly, myocarditis, and enlargement of the esophagus and colon as a result of the destruction of nerve cells (E.g. Auerbach's plexus) and other tissues that control the growth of these organs. Involvement of the CNS may produce granulomas in the brain with cyst formation and a meningoencephalitis. Death from chronic Chagas' disease results from tissue destruction in the many areas invaded by the organisms, and sudden death results from complete heart block and brain damage.

### **Laboratory diagnosis**

Examine thin or thick stained preparations for trypomastigotes. Wet preparations should also be examined to look for motile organisms that leave the blood stream and become difficult to find. Biopsy of lymph nodes, liver, spleen, or bone marrow may demonstrate organisms in amastigote stage.

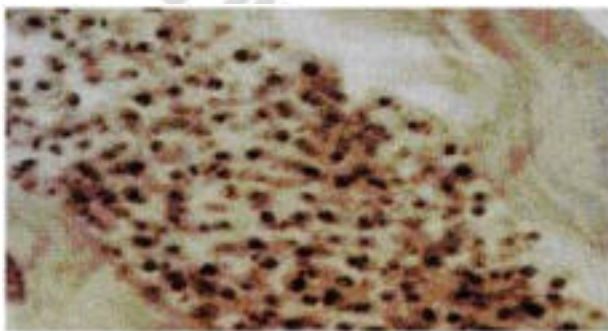


Figure 13; Amastigote stage of *Trypanosoma cruzi* in skeletal muscle

Xenodiagnosis - which consists of allowing an uninfected, laboratory-raised reduviid bug to feed on the patient and, after several weeks, examining the intestinal contents of the bug for the organism.

### **Immunity**

Unlike African trypanosomiasis, the antigenic variation is less common in *T.cruzi* infection. Therefore, the humoral and cellular immune responses function in the immune system.

### **Treatment**

The drug of choice is nifurtimox. Alternative agents include allopurinol & benzimidazole.

### **Prevention**

- Bug control, eradication of nests
- Treating infected person & exclusion of donors by screening blood.
- Development of vaccine.



## UNIT FIVE

### MEDICALLY IMPORTANT CILIATES

#### **Balantidiasis**

The intestinal protozoan *Balantidium coli* is the only member of the ciliate group that is pathogenic for humans. Disease produced by *B. coli* is similar to amebiasis, because the organisms elaborate proteolytic and cytotoxic substances that mediate tissue invasion and intestinal ulceration.

#### **Life cycle**

The life cycle of *B. coli* is simple, involving ingestion of infectious cysts, excystation, and invasion of trophozoites into the mucosal lining of the large intestine, caecum, and terminal ileum. The trophozoite is covered with rows of hair like cilia that aid in motility. Morphologically more complex than amebae, *B. coli* has a funnel-like primitive mouth called a cytostome, a large (macro) nucleus and a small (micro) nucleus involved in reproduction.

#### **Epidemiology**

*B. coli* are distributed worldwide. Swine and (less commonly) monkeys are the most important reservoirs. Infections are transmitted by the faecal-oral route; outbreaks are associated with contamination of water supplies with pig faeces. Person-to-person spread, including through food handlers, has been implicated in outbreaks. Risk factors associated with human disease include contact with swine and substandard hygienic conditions.

#### **Clinical features**

As with other protozoan parasites, asymptomatic carriage of *B. coli* can exist. Symptomatic disease is characterized by abdominal pain, tenderness, tenesmus,



nausea, anorexia, and watery stools with blood and pus. Ulceration of the intestinal mucosa, as with amebiasis, can be seen; a secondary complication caused by bacterial invasion into the eroded intestinal mucosa can occur. Extra intestinal invasion of organs is extremely rare in balantidiasis.

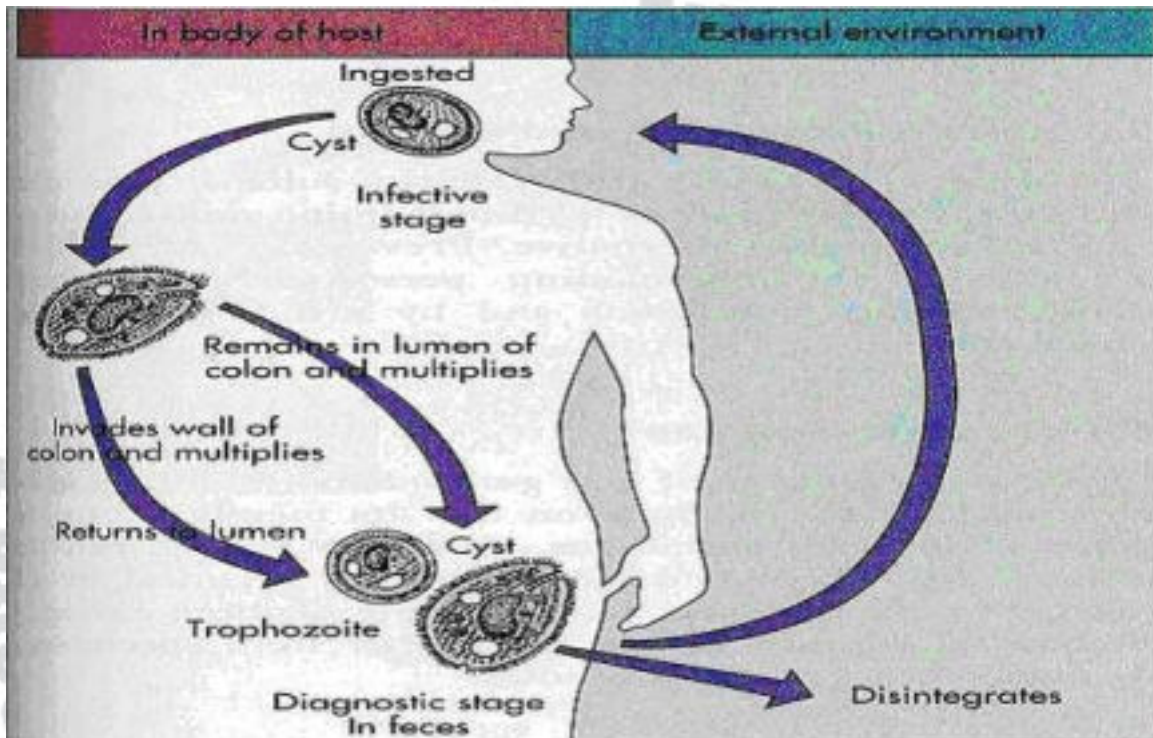


Figure 14; life cycle of *Balantidium coli*

### Laboratory Diagnosis

Microscopic examination of faeces for trophozoite and cysts is performed. The trophozoite is very large, varying in length from 50 to 200µm and in width from 40 to 70µm. The surface is covered with cilia.

### Treatment

The drug of choice is tetracycline; iodoquinol and metronidazole are alternative agents.

## UNIT SIX

### COCCIDIA (SPOROZOA)

#### INTRODUCTION

Coccidia are members of the class sporozoa, Phylum Apicomplexa. Apical complex is present at some stage and consists of elements visible with electron microscope. The life cycle is characterized by an alternation of generations, i.e. sexual (gametogony) and asexual (schizogony) reproduction and most members of the group also share alternative hosts.

The locomotion of a mature organism is by body flexion, gliding, or undulation of longitudinal ridges. The genus *Plasmodium* that are the causes of malaria is the prototype of this class.

#### 4.1. Malaria

There are four species normally infecting humans, namely, *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae*.

##### Life cycle

The life cycle of malaria is passed in two hosts (alternation of hosts) and has sexual and asexual stage (alternation of generations).

Vertebrate host - man (intermediate host), where the asexual cycle takes place.

The parasite multiplies by schizogony and there is formation of male and female gametocytes (gametogony).

Invertebrate host - mosquito (definitive host) where the sexual cycle takes place.

Union of male and female gametes ends in the formation of sporozoites (sporogony).

The life cycle passes in four stages:

Three in man:- Pre - erythrocytic schizogony

- Erythrocytic schizogony
- Exo- erythrocytic schizogony

One in mosquito - Sporogony

*Introduction into humans* - when an infective female Anopheles mosquito bites man, it inoculates saliva containing sporozoites (infective stage).

**Pre- Erythrocytic schizogony** - sporozoites reach the blood stream and within 30 minutes enter the parenchymal cells of the liver, initiating a cycle of schizogony. Multiplication occurs in tissue schizonts, to form thousands of tiny merozoites. Merozoites are then liberated on rupture of schizonts about 7th – 9th day of the bites and enter into the blood stream. These merozoites either invade the RBC's or other parenchymal liver cells. In case of *P. falciparum* and possibly *P. malariae*, all merozoites invade RBC's without re-invading liver cells. However, for *P. vivax* and *P. ovale*, some merozoites invade RBC's and some re-invade liver cells initiating further *Exo-erythrocytic* schizogony, which is responsible for relapses. Some of the merozoites remain dormant (hypnozoites) becoming active later on.

Erythrocytic schizogony (blood phase) is completed in 48 hrs in *P. vivax*, *P. ovale*, and *P. falciparum*, and 72 hrs in *P. malariae*. The merozoites reinvade fresh RBC's repeating the schizogonic cycles

Erythrocytic merozoites do not reinvade the liver cells. So malaria transmitted by blood transfusion reproduces only erythrocytic cycle

### **Gametogony**

Some merozoites that invade RBC's develop into sexual stages (male and female gametocytes). These undergo no further development until taken by the mosquito.

### ***Sporogony (extrinsic cycle in mosquito)***

When a female Anopheles mosquito vector bites an infected person, it sucks blood containing the different stages of malaria parasite. All stages other than gametocytes are digested in the stomach.

The microgametocyte undergoes ex-flagellation. The nucleus divides by reduction division into 6-8 pieces, which migrate to the periphery. At the same time 6-8 thin filaments of cytoplasm are thrust out, in each passes a piece of chromatin. These filaments, the microgametes, are actively motile and separate from the gametocyte.

The macrogametocyte by reduction division becomes a macrogamete.

Fertilization occurs by entry of a micro gamete into the macro gamete forming a zygote.

The zygote changes into a worm like form, the ookinete, which penetrates the wall of the stomach to develop into a spherical oocyst between the epithelium and basement membrane. The oocysts increase in size. Thousands of sporozoites develop inside the oocysts. Oocysts rupture and sporozoites are liberated in the body cavity and migrate everywhere particularly to the salivary glands. Now the mosquito is infective

The sporogonous cycle in the mosquito takes 8-12 days depending on temperature



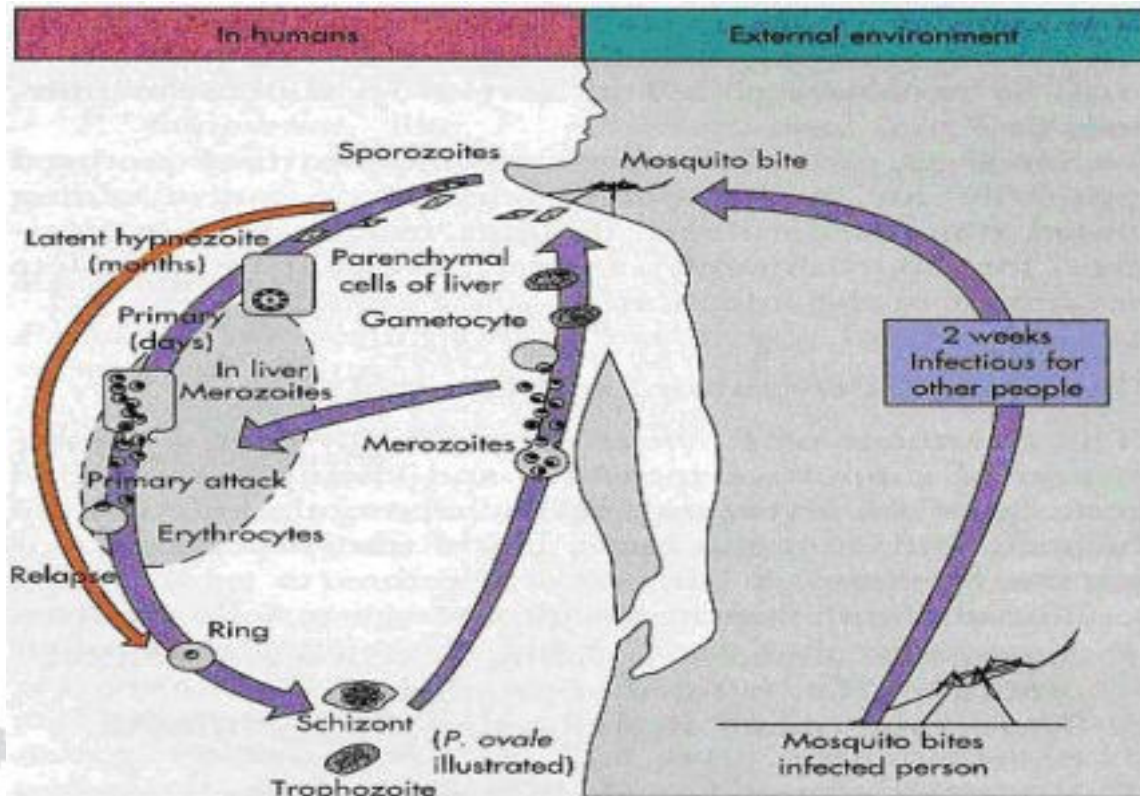


Figure 15; Life cycle of Plasmodium species

#### 4.1.1. Plasmodium falciparum

*Plasmodium falciparum* demonstrates no selectivity in host erythrocytes, i.e. it invades young and old RBCs cells. The infected red blood cells also do not enlarge and become distorted.

- Multiple sporozoites can infect a single erythrocyte, and show multiple infections of cells with small ring forms.
- The trophozoite is often seen in the host cells at the very edge or periphery of cell membrane at accolé position.
- Occasionally, reddish granules known as Maurer's dots are observed
- Mature (large) trophozoite stages and schizonts are rarely seen in blood films, because their forms are sequestered in deep capillaries, liver and spleen.

- Peripheral blood smears characteristically contain only young ring forms and occasionally crescent shaped gametocytes.

## **Epidemiology**

*P.falciparum* occurs almost exclusively in tropical and subtropical regions. Weather (rainfall, temperature & humidity) is the most obvious cause of seasonality in malaria transmission. To date, abnormal weather conditions are also important causes of significant and widespread epidemics. Moreover, drug-resistant infection of *P.falciparum* is the commonest challenge in many parts of the world. In Ethiopia, even though all the four species of plasmodium infecting man have been recorded, *P.falciparum* is the one that most causes the epidemic disease and followed by vivax and malariae. *P.ovale* is rare. Infection rates in Ethiopia are 60%, 40%, 1%, and <1% for *P. falciparum*, *P. vivax*, *P. malariae*, and *P. ovale*, respectively.

## **Clinical features**

Of all the four Plasmodia, *P. falciparum* has the shortest incubation period, which ranges from 7 to 10 days. After the early flu-like symptoms, *P.falciparum* rapidly produces daily (quotidian) chills and fever as well as severe nausea, vomiting and diarrhea. The periodicity of the attacks then becomes tertian (36 to 48 hours), and fulminating disease develops. Involvement of the brain (cerebral malaria) is most often seen in *P.falciparum* infection. Capillary plugging from an adhesion of infected red blood cells with each other and endothelial linings of capillaries causes hypoxic injury to the brain that can result in coma and death. Kidney damage is also associated with *P.falciparum* malaria, resulting in an illness called “black water” fever. Intravascular hemolysis with rapid destruction of red blood cells produces a marked hemoglobinuria and can result in acute renal failure, tubular necrosis, nephrotic syndrome, and death. Liver involvement is characterized by



abdominal pain, vomiting of bile, hepatosplenomegally, severe diarrhea, and rapid dehydration.

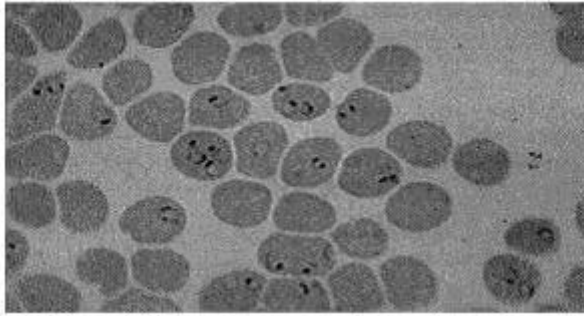


Figure 16; Ring form of *P.falciparum*, with multiple infection of an erythrocyte

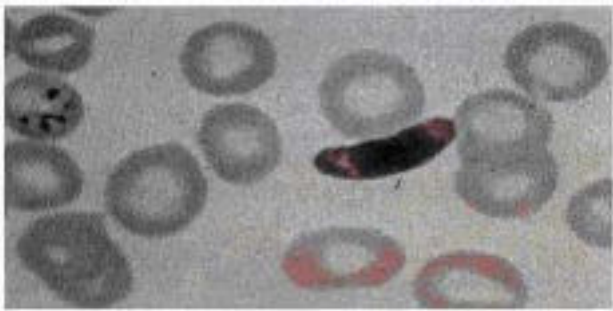


Figure 17; mature gametocyte of *P.falciparum*.

### Treatment

Because chloroquine – resistant strains of *P.falciparum* are present in many parts of the world, infection of *P.falciparum* may be treated with other agents including mefloquine, quinine, guanidine, pyrimethamine – sulfadoxine, and doxycycline. If the laboratory reports a mixed infection involving *P.falciparum* and *P.vivax*, the treatment must eradicate not only *P.falciparum* from the erythrocytes but also the liver stages of *P.vivax* to avoid relapses provided that the person no longer lives in a malaria endemic area.

#### **4.1.2. *Plasmodium vivax***

*P. vivax* is selective in that it invades only young immature erythrocytes.

Infections of *P. vivax* have the following characteristics:

- Infected red blood cells are usually enlarged and contain numerous pink granules or schuffner's dots.
- The trophozoite is ring-shaped but amoeboid in appearance.
- More mature trophozoites and erythrocytic schizonts containing up to 24 merozoites are present.
- The gametocytes are round

#### **Epidemiology**

*P. Vivax* is the most prevalent of the human plasmodia with the widest geographic distribution, including the tropics, subtropics, and temperate regions. However, it is the second most prevalent in Ethiopia following *P. falciparum*

#### **Clinical features**

After an incubation period (usually 10 to 17 days), the patient experiences vague flu-like symptoms, such as headache, muscle pains, photophobia, anorexia, nausea and vomiting. As the infection progresses, increased numbers of rupturing erythrocytes liberate merozoites as well as toxic cellular debris and hemoglobin in to circulation. In combination, these substances produce the typical pattern chills, fever and malarial rigors. These paroxysms usually reappear periodically (generally every 48 hours) as the cycle of infection, replication, and cell lyses progresses. The paroxysms may remain relatively mild or may progress to severe attacks, with hours of sweating, chills, shaking persistently, high temperatures (103<sup>0</sup>F to 106<sup>0</sup>F) and exhaustion. Since *P. vivax* infects only the reticulocytes, the parasitemia is usually limited to around 2 to 5% of the available RBCs.

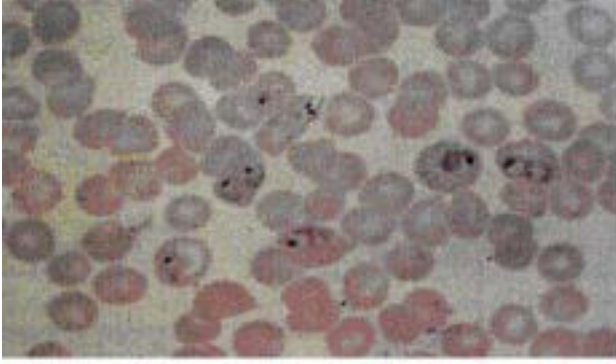


Figure 18; *Plasmodium vivax* ring form and trophozoites

## Treatment

Chloroquine is the drug of choice for the suppression and therapeutic treatment of *P.vivax*, followed by primaquine for radical cure and elimination of gamatocytes.

### 4.1.3. *Plasmodium malariae*

In contrast with *P.vivax* and *P.ovale*, *P.malariae* can infect only mature erythrocytes with relatively rigid cell membranes. As a result, the parasite's growth must conform to the size and shape of red blood cell.

This requirement produces no red cell enlargement or distortion, but it results in distinctive shapes of the parasite seen in the host cell, "band and bar forms" as well as very compact dark staining forms. The schizont of *P.malariae* is usually composed of eight merozoites appearing in a rosette.

## Epidemiology

*P. malariae* infection occurs primarily in the same sub-tropical and temperate regions as infections with the other plasmodia but is less prevalent.

## Clinical features

The incubation period for *P. malariae* is the longest of the plasmodia, usually 18 to 40 days, but possibly several months to years. The early symptoms are flu-like with fever patterns of 72 hours (quartan or malarial) in periodicity.

## Treatment

Treatment is similar to that for *P. vivax* and *P. ovale*.

### 4.1.4. *Plasmodium ovale*

*P. ovale* is similar to *P. vivax* in many respects, including its selectivity for young, pliable erythrocytes. As a consequence the classical characteristics include:

- The host cell becomes enlarged and distorted, usually in an oval form.
- Schiffner's dots appear as pale pink granules.
- The infected cell border is commonly fimbriated or ragged
- Mature schizonts contain about 10 merozoites.

## Epidemiology

*P. ovale* is distributed primarily in tropical Africa. It is also found in Asia and South America.

## Clinical features

The incubation period for *P. ovale* is 16-18 days but can be longer. Clinically, ovale malaria resembles vivax malaria with attacks recurring every 48-50 hours. There are however, fewer relapses with *P. ovale*. Less than 2% of RBCs usually become infected.

## **Treatment**

The treatment regimen, including the use of primaquine to prevent relapse from latent liver stages is similar to that used for *P.vivax* infection.

## **Laboratory diagnosis**

Microscopic examination of thick and thin films of blood is the method of choice for confirming the clinical diagnosis of malaria and identifying the specific species responsible for disease.

Malaria parasites in thick and thin blood films are best stained at pH 7.1 – 7.2 using a Romanowsky stain (contains azure dyes and eosin).

The thick film is a concentration method that may be used to detect the presence of organisms. The thin film is most useful for establishing species identification.

Serologic procedures are available but they are used primarily for epidemiological surveys or for screening blood donors.

## **Immunity**

There is evidence that antibodies can confer hormonal immunity against malaria infection.

## **Prevention**

- Chemoprophylaxis and prompt diagnosis and treatment.
- Control of mosquito breeding
- Protection of insect bite by screening, netting and protective clothing
- Use of insect repellents.



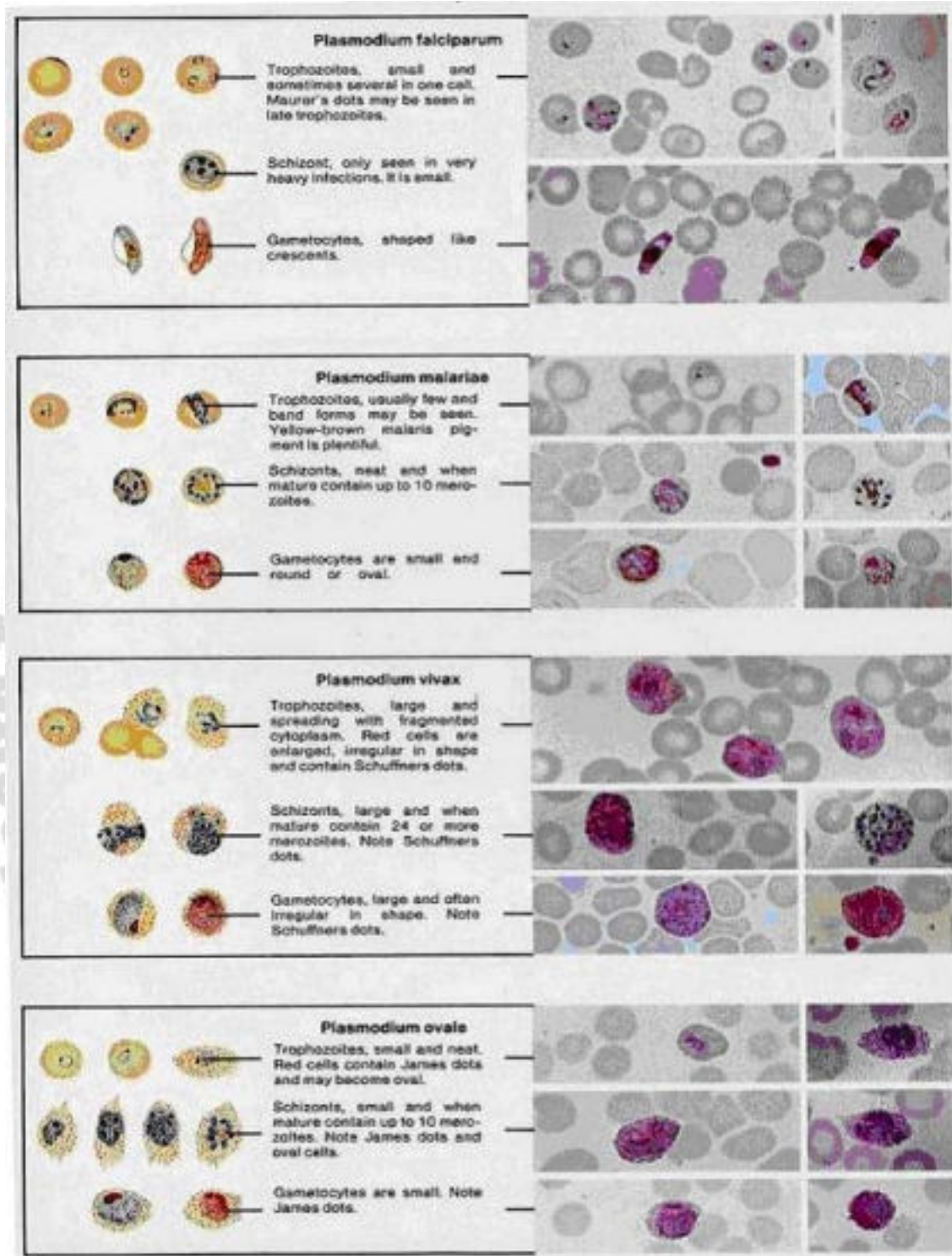


Figure 19; Romanowsky stained thin malaria films and their different stages.



## 4.2. Other Coccidian parasites

**4.2.1. *Toxoplasma gondii*** – causes *toxoplasmosis*. The definitive host is the domestic cat and other felines. Humans and other mammals are intermediate hosts. *T.gondii* is usually acquired by ingestion and transplacental transmission from an infected mother to the fetus can occur. Human-to-human transmission, other than transplacental transmission, does not occur. After infection of the intestinal epithelium, the organisms spread to other organs, especially the brain, lungs, liver, and eyes. Most primary infections in immunocompetent adults are asymptomatic. Congenital infection can result in abortion, stillbirth, or neonatal disease with encephalitis, chorioretinitis and hepatosplenomegaly. Fever, jaundice, and intracranial calcifications are also seen. For the diagnosis of acute and congenital infections, an immunofluorescence assay for detection of antibody is used. Microscopic examination of Giemsa-stained preparations shows crescent-shaped trophozoite. Cysts may be seen in the tissue. Treatment is with a combination of sulfadiazine and pyrimethamine.

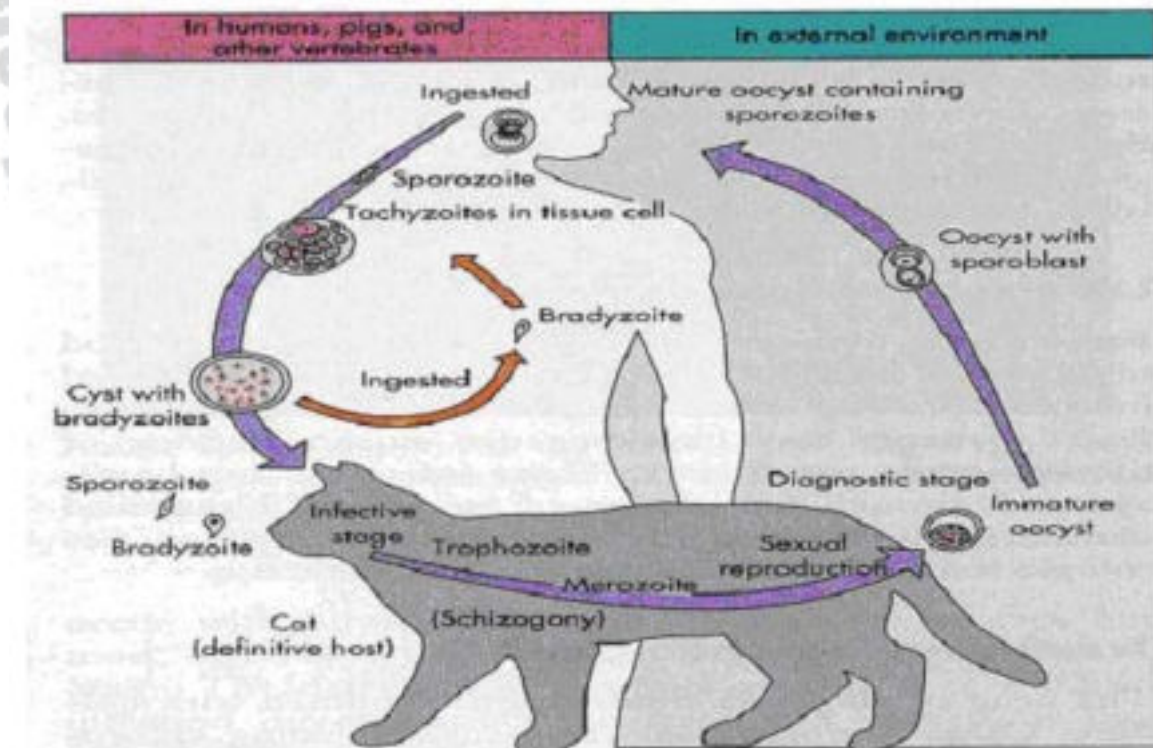


Figure 20; Life cycle of *Toxoplasma gondii*

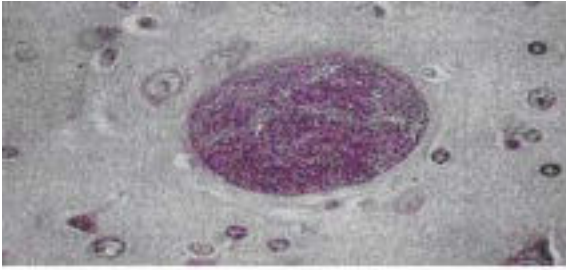


Figure 21; cyst of *Toxoplasma gondii* in mouse brain.

**4.2.2. Cyclospora cayetanensis** - is an intestinal protozoan that causes watery diarrhea in both immunocompetent and immunocompromised individuals. It is classified as a member of the Coccidian; the organism is acquired by fecal – oral transmission, especially via contaminated water supplies. There is no evidence for an animal reservoir. The diarrhea can be prolonged and relapsing, especially in immunocompromised patients. Infection occurs worldwide. The diagnosis is made microscopically by observing the spherical oocysts in a modified acid-fast stain of a stool sample. There are no serologic tests. The treatment of choice is trimethoprim-sulfamethoxazole.

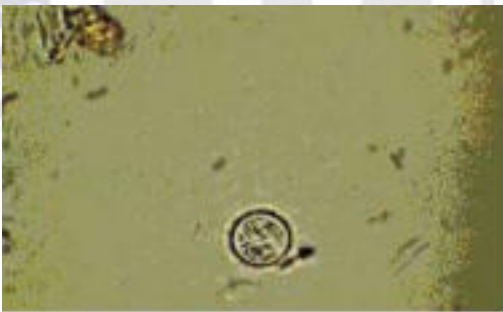


Figure 22; Oocyst of *cyclospora cayetanensis*

**4.2.3. Isospora belli** - is an intestinal protozoan that causes diarrhea, especially in immunocompromised patients, e.g., those with AIDS. Its life cycle parallels that of other members of the Coccidia. The organism is acquired by fecal-oral transmission of oocysts from either human or animal sources. The oocysts excyst

in the upper small intestine and invade the mucosa, causing destruction of the brush border.

The disease in immunocompromized patients presents as a chronic, profuse, watery diarrhea. The pathogenesis of the diarrhea is unknown. Diagnosis is made by finding the typical oocysts in fecal specimens. Serologic tests are not available. The treatment of choice is trimethoprim-sulfamethoxazole.

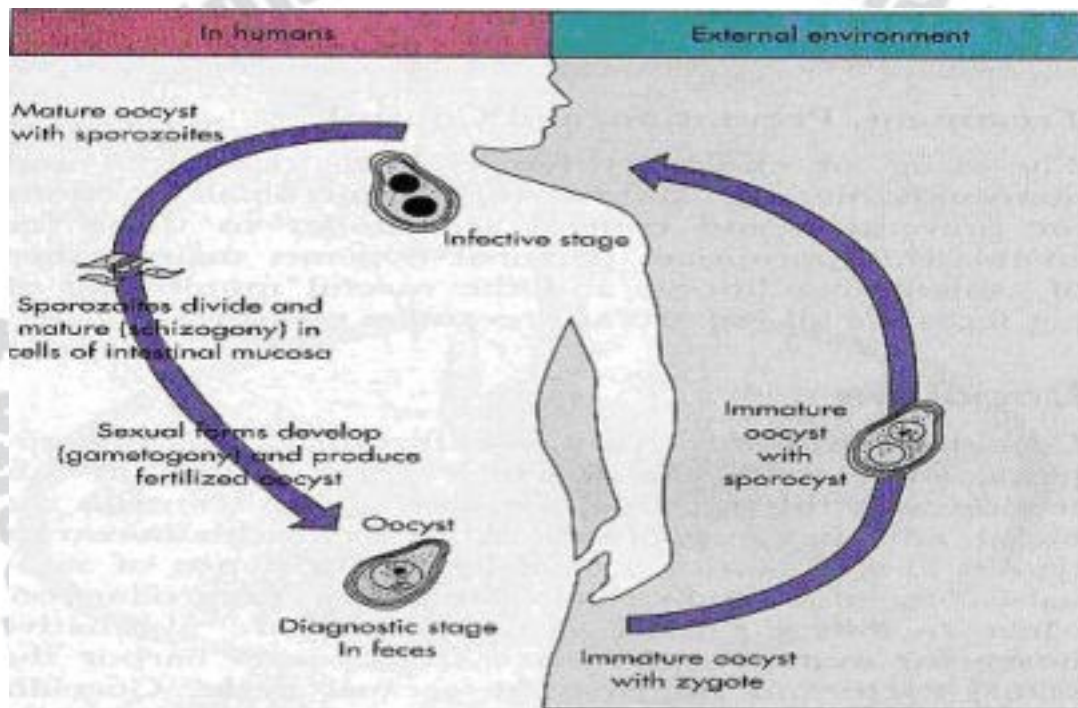


Figure 23; Life cycle of *Isospora belli*.

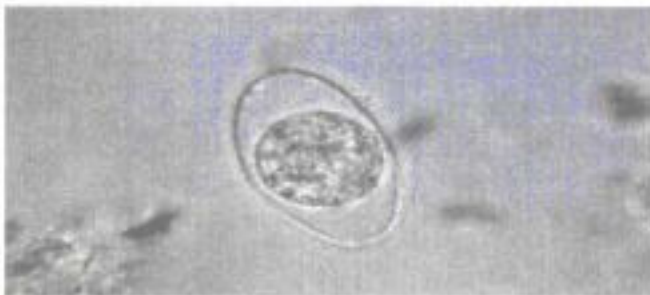


Figure 24; immature Oocyst of isospora species

**4.2.4. *Cryptosporidium parvum*** – causes cryptosporidiosis, the main symptom of which is diarrhea. It is most severe in immunocompromized patients, e.g., those with AIDS. The organism is acquired by faecal-oral transmission of Oocysts from either human or animal sources. The oocysts excyst in the small intestine, where the trophozoite (and other forms) attach to the gut wall. Invasion does not occur. The jejunum is the site most heavily infested. The pathogenesis of the diarrhea is unknown; no toxin has been identified.

*Cryptosporidium* causes diarrhea worldwide, for large outbreaks of diarrhea caused by *Cryptosporidium* are attributed to inadequate purification of drinking water. The disease in immunocompromized patients presents primarily as a watery, non-bloody diarrhea causing large fluid loss. Symptoms persist for long periods in immunocompromized patients, whereas self-limited in immunocompetent individuals. Although immunocompromized patients usually do not die of cryptosporidiosis, the fluid loss and malnutrition are severely debilitating. Diagnosis is made by finding oocysts in fecal smears when using a modified Kinyoun acid-fast stain. Serological tests are not available. There is no effective drug therapy.

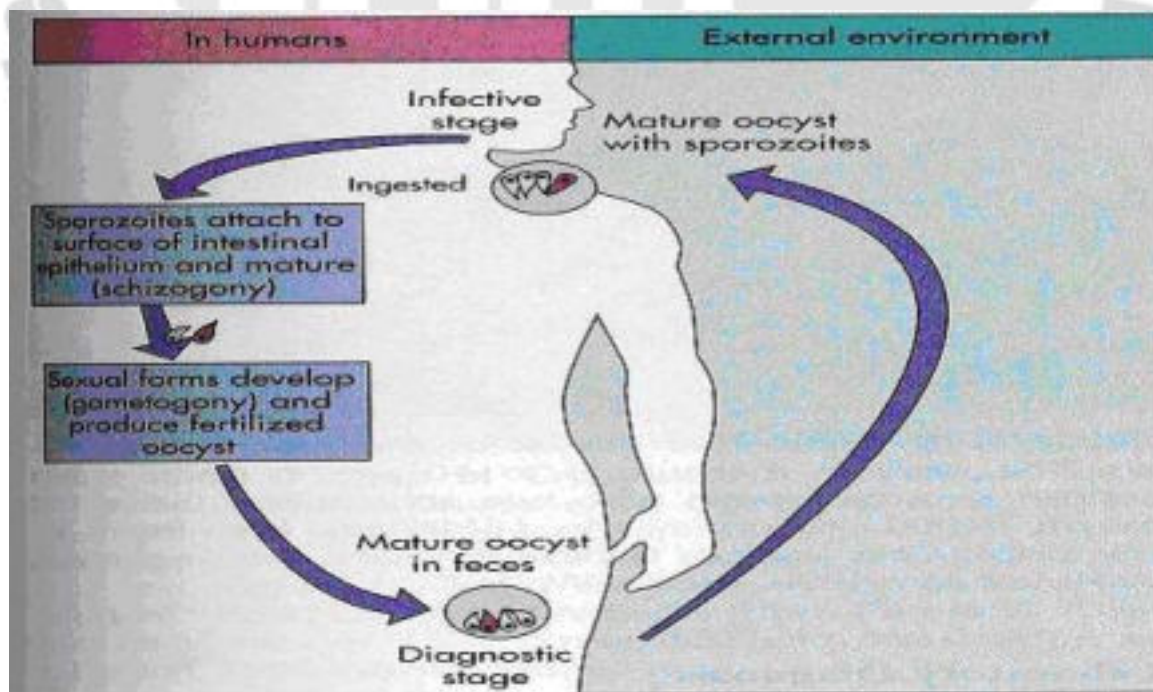


Figure 25; life cycle of *Cryptosporidium* species.





Figure 26; Acid-fast stained *Cryptosporidium* oocyst.

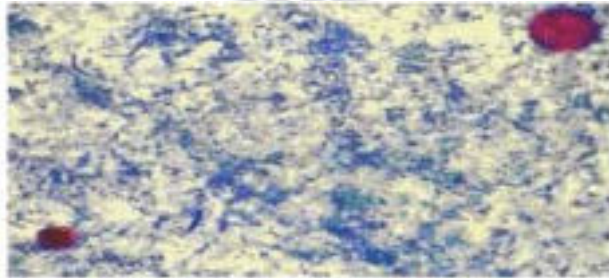


Figure 27; Oocysts of *Cryptosporidium parvum* (lower left) & *Cyclospora cayetanensis* (upper right). Both stained red with Ziehl-Neelsen stain.

**4.2.5. Microsporidia** - are a group of protozoa characterized by obligate intracellular replication and spore formation. *Enterocytozoon bienusi* and *Septata intestinalis* are two important microsporidial species that cause severe, persistent, watery diarrhea in AIDS patients. The organisms are transmitted from faecal-oral route. It is uncertain whether an animal reservoir exists. Diagnosis is made by visualization of spores in stool samples or intestinal biopsy samples; the drug of choice is albendazole.

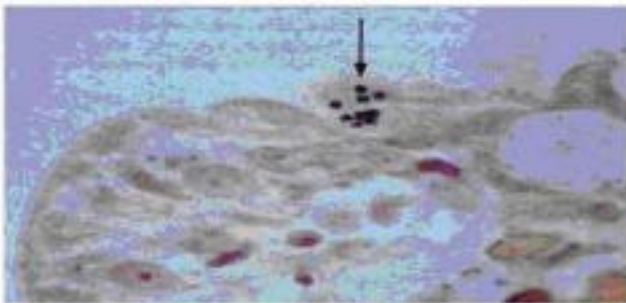


Figure 28; Gram-positive spores of Microsporidia in jejunal biopsy

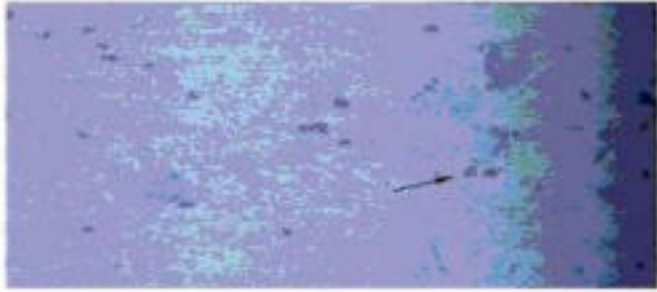


Figure 29; smear of formalin-fixed stool specimen showing pinkish red-stained Microsporidia spores (Chromotrope based stain)





## Review Questions

- 1) What are the two distinctive characteristics that differentiate protozoa from other Eukaryotic protists?
- 2) What are the ecological advantages of protozoa?
- 3) Explain the reproductive process, transmission route and pathogenesis of protozoan parasites.
- 4) How are medically important protozoa classified?
- 5) Describe the pathogenesis of *E.histolytica*.
- 6) Explain the clinical features of *Giardia lamblia*.
- 7) What are the drugs of choice for treatment of *Trichomoniasis vaginalis*?
- 8) What is the hemoflagellate responsible for causing kala-azar?
- 9) What are the protozoal species responsible for old world cutaneous leishmaniasis?
- 10) Explain the pertinent clinical syndrome of *Leishmania aethiopica*.
- 11) What are the causative protozoa for African trypanosomiasis?
- 12) Explain the immune systems involved and the immune phenomenon in the infection of African trypanosomiasis.
- 13) What are the common characteristics of the class sporozoa?
- 14) Which of the plasmodia parasite has the shortest incubation period?
- 15) List and describe the pathogenesis of parasitic protozoa frequently found in immunocompromized individuals.

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# **UNIT SEVEN**

## **MEDICAL HELMINTHOLOGY**

### **LEARNING OBJECTIVES**

At the end of this session, students should be able to:

- Understand medically important helminthes including their life cycles, modes of transmissions, clinical features, diagnosis, treatment and prevention.
- Describe blood, intestinal, liver and lung flukes.
- Understand common round worms.
- Understand different species of Cestodes.

### **INTRODUCTION**

Medical helminthology is concerned with the study of helminthes or parasitic worms. Helminthes are trophoblastic metazoa (multi-cellular organisms). Helminthes are among the common parasitic causes of human suffering. They are the cause of high morbidity and mortality of people worldwide. They cause different diseases in humans, but few helminthic infections cause life- threatening diseases. They cause anemia and malnutrition. In children they cause a reduction in academic performance. Helminthes also cause economic loss as a result of infections of domestic animals. There is age dependent distribution of infections from geo-helminthes and schistosomes. As a result of predisposing behavioral and immunological status, children disproportionately carry the burden of schistosomes and geo-helminthes.

The sources of the parasites are different. Exposure of humans to the parasites may occur in one of the following ways:

1. Contaminated soil (Geo-helminthes), water (cercariae of blood flukes) and food (Taenia in raw meat).
2. Blood sucking insects or arthropods (as in filarial worms).

3. Domestic or wild animals harboring the parasite (as in echinococcus in dogs).
4. Person to person (as in *Enterobius vermicularis*, *Hymenolopis nana*).
5. Oneself (auto-infection) as in *Enterobius vermicularis*.

They enter the body through different routes including: mouth, skin and the respiratory tract by means of inhalation of airborne eggs.

The helminthes are classified into three major groups. These are:

1. Trematodes (Flukes)
2. Nematodes (Round worms)
3. Cestodes (Tape worms)

The Trematodes and Cestodes are groups of flat worms.

The major objective of this lecture note is to provide good understanding of the most common helminthes prevalent in the tropics in general and in Ethiopia in particular.

## UNIT EIGHT

### MEDICALLY IMPORTANT TREMATODES (FLUKES)

#### INTRODUCTION

Trematodes belong to the phylum platyhelminthes. They are found in a wide range of habitats. The great majority inhabit the alimentary canal, liver, bile duct, ureter and bladder of vertebrate animals.

According to the sites they inhabit, there are four groups of flukes. These are: Blood flukes, Intestinal flukes, Liver flukes, and Lung flukes

#### 1.1. BLOOD FLUKES

These are flukes that reside mainly in the blood vessels of various organs and the schistosomes are the prototype and the commonest flukes in our country.

##### 1.1.1. SCHISTOSOMIASIS (BILHARZIASIS)

It is estimated that about 600 million people in 79 countries suffer from schistosomiasis (Bilharziasis). The schistosomes cause intestinal, hepato-splenic, pulmonary, urogenital, cerebral and other forms of schistosomiasis. Schistosome is the only fluke with separate sexes. The female worm lies in the gynecophoral canal of the male. This condition is important for transportation.

There are five medically important species:

1. *Schistosoma mansoni*: causes intestinal schistosomiasis.
2. *Schistosoma haematobium*: causes vesical (urinary) schistosomiasis.
3. *Schistosoma japonicum*: causes intestinal schistosomiasis.
4. *Schistosoma intercalatum*: causes intestinal schistosomiasis.
5. *Schistosoma mekongi*: causes intestinal schistosomiasis. This seems to cause milder disease in man. It causes disease in other vertebrate hosts.

The first two schistosomes (*S. mansoni* and *S. haematobium*) are prevalent in Ethiopia.

### **SCHISTOSOMA MANSONI**

**Habitat** - This species lives in the veins of the intestine.

**Geographical distribution:** It is found in Africa, South America, Middle East (some Arab countries) etc. Stream and lake-based transmission is common.

The snail hosts that harbor *S. mansoni* are the genera: *Biomphalaria* (*B. glabrata*) and *Trochicorbis*. These have oval shells.

### **Morphology**

**Male:** The male ranges in size from 1-1.4 cm in length and the body is covered by coarse tubercles. It has 6-9 testes

**Female:** The female is 1.5-2.0 cm in length. The ovary is present in the anterior third and Vitelline glands occupy the posterior two-thirds. It lays about 100-300 eggs daily. The uterus is short containing few ova.

### **URINARY SCISTOSOMIASIS**

**Etiology** - *Schistosoma haematobium*

**Habitat** - The worm lives in the veins of the bladder of humans.

The peak prevalence is the 10-14 year age group. The snail hosts that harbor *S. haematobium* are the genera *Bulinus* (*Bulinus africanus*, *B. truncatus*) and *Physopsis*.

**Male:** The male ranges in size from 1-1.5 cm in length. The body is covered by fine tubercles. It has 4-5 testes.

**Female:** The female ranges in size from 2-2.5 cm in length. The ovary is present in the posterior third. Vitelline glands occupy the posterior thirds. Uterus is long containing many ova. It lays about 20-200 eggs daily.



**Distribution:** In Ethiopia, *S. haematobium* is found in the Lower Awash Valley in the east and in Benshangul-Gumuz (Assossa) regional state in the west in low altitudes below 1000 meters above sea level.

### **SCHISTOSOMA JAPONICUM**

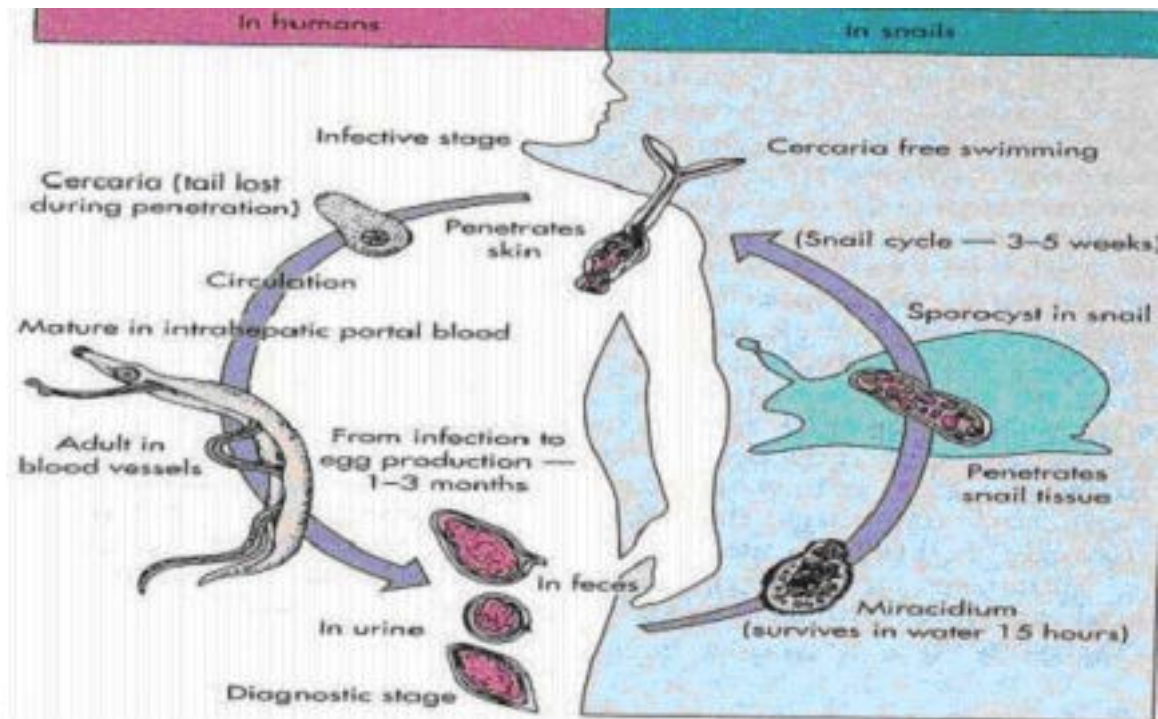
The female adult worm lays about 500-3500 eggs daily. The eggs are ovoid, bearing only a minute lateral spine or a small knob postero-laterally. It is found in Japan, China, and Philippines, etc.

### **SCHISTOSOMA INTERCALATUM**

This is the rarest and least pathogenic schistosome that matures in man. It is found in Western and Central Africa. The daily egg output is about 300. The eggs have a terminal spine.

### **LIFE CYCLE OF SCHISTOSOMES**

Adult worms reside in pairs: the female lying in the gynecophoral canal of the male. After fertilization, eggs are passed into the venules. A larval form – the miracidium - develops within the egg. Its lytic enzymes and the contraction of the venule rupture the wall of the venule liberating the egg into the perivascular tissues of the intestine (*S. mansoni*) or urinary bladder (*S. haematobium*). The eggs pass into the lumens and organs and are evacuated in the feces (*S. mansoni*) or the urine (*S. haematobium*). On contact with fresh water the miracidia hatch from the eggs and swim about until they find the appropriate snail, which they penetrate. After two generations of sporocyst development and multiplication within the snail, the fork-tailed cercariae emerge. Infection to man takes place during bathing or swimming. The cercariae penetrate the skin, are carried into the systemic circulation and pass through to the portal vessels. Within the intrahepatic portion of the portal system, the worms feed and grow to maturity.



**Figure 1.1.** Life cycle of schistosomes

### Symptoms and complications

Patients infected with *S. haematobium* suffer from terminal haematuria and painful micturition. There is inflammation of the urinary bladder (cystitis), and enlargement of spleen and liver.

Patients infected with *S. mansoni* suffer from cercarial dermatitis (swimmers itch) and dysentery (mucus and blood in stool with tenesmus) as well as enlargements of the spleen and liver.

*S. haematobium* causes squamous cell carcinoma in the bladder.

### Laboratory Diagnosis

#### *S. mansoni*

- ♦ Microscopic examination of the stool for eggs after concentration by sedimentation method. The egg has characteristic lateral spine.

- ◆ Rectal snip

### **S. haematobium:**

- ◆ Examination of the urine after allowing it to sediment in a conical urinalysis glass. A drop from the sediment is taken and examined for eggs. Egg has terminal spine.
- ◆ Biopsy from bladder



**Figure 1.2.** Eggs of *S. mansoni* and *S. haematobium*

### **Treatment:**

Praziquantel: single oral dose of 40 mg/kg divided into two doses.

### **Prevention:**

#### **1. Health education:**

- A. On use of clean latrines and safe water supply
- B. Avoid urination and defecation in canals, avoid contact with canal water

#### **2. Snail control:**

- A. Physical methods:
  - i. Periodic clearance of canals from vegetations.
  - ii. Manual removal of snails and their destruction.
- B. Biological methods: Use of natural enemies to the snails such as Marisa.

C. Chemical methods: Molluscides are applied in the canals to kill the snails. e.g. Endod

## 1.2. INTESTINAL FLUKES

- ◆ ***Fasciolopsis buski***: These giant intestinal flukes (2-7.5 cm in length) are found in some Asian countries.
- ◆ **Heterophyids**: Minute flukes acquired by ingestion of raw fresh water fish. They are found in Asian countries. Neither are found in Ethiopia.

## 1.3. LIVER FLUKES

- ◆ ***Clonorchis sinensis***: Chinese liver fluke - adult worms live in bile ducts.
- ◆ ***Faciola hepatica***: Sheep liver fluke - is a common parasite, cosmopolitan in distribution. It is large (3 cm in length). Adult worms reside in the large biliary passages and gall bladder.
- ◆ Other: ***Faciola gigantica***: lives in the liver of cattle. Human infections are very rare.

## 1.4. LUNG FLUKES

At least eight different species of lung flukes, all belonging to the genus *Paragonimus*, are known to infect man. *Paragonimus westermani*, best known species, affects man causing paragonimiasis (lung disease). It is found in Asia (China, India, Indonesia, Malaya etc) and some African countries. So far there is no report of it in Ethiopia.

## **UNIT NINE**

### **NEMATODES (ROUND WORMS)**

All the important human parasites of the Phylum Nematelminthes (Aschelminthes) belong to the Class Nematoda.

#### **GENERAL CHARACTERISTICS OF NEMATODES**

They are un-segmented, elongated and cylindrical. They have separate sexes with separate appearances. They have a tough protective covering or cuticle. They have a complete digestive tract with both oral and anal openings. The nematodes are free living (Majority) or parasites of humans, plants or animals.

#### **The parasitic nematodes:**

The nematodes are generally light cream-white colored. Their life cycle includes: egg, larvae and adult.

#### **The parasitic nematodes are divided into:**

##### **1. Intestinal nematodes**

##### **1.1. Intestinal nematodes with tissue stage**

- A. *Ascaris lumbricoides*
- B. Hookworms
- C. *Strongyloides stercoralis*

##### **1.2. Intestinal nematodes without tissue stage**

- A. *Enterobius vermicularis*
- B. *Trichuris trichiura*.

##### **2. Tissue and blood dwelling nematodes**

- 2.1. Filarial worms
- 2.2. *Dracunculus medinensis*



2.3. Trichinella

2.4. Larva migrans.

## **2.1. INTESTINAL NEMATODES WITH TISSUE STAGE**

### **2.1.1. ASCARIS LUMBRICOIDES**

These are common roundworms infecting more than 700 million people worldwide.

#### **Morphology:**

Male adult worm measures 15-20 cm in length. The posterior end is curved ventrally. The female worm measures 20-40 cm in length. Its posterior end is straight.

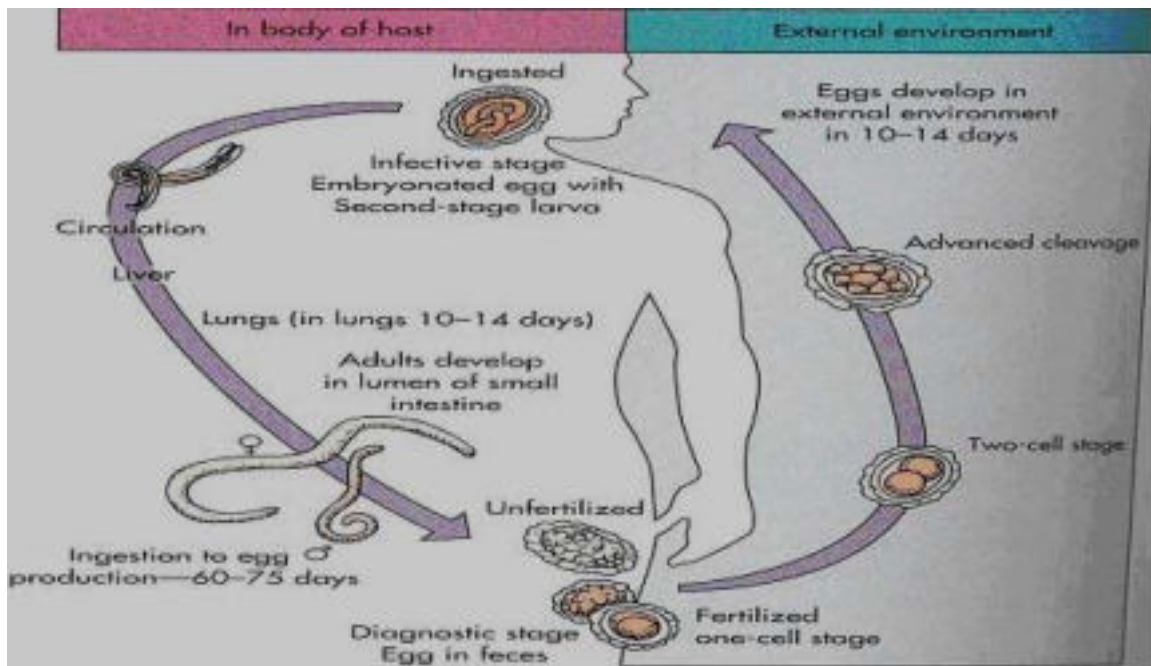
#### **Infective stage and modes of infection:**

The egg containing larva when ingested with contaminated raw vegetables causes ascariasis.

#### **Life cycle:**

Ingested eggs hatch in the duodenum. The larvae penetrate the intestinal wall and circulate in the blood. From the heart they migrate to the lungs, ascend to the trachea, descend to the esophagus and finally reach the small intestine to become adult. The female pass immature eggs which pass to the soil and mature in 2 weeks.





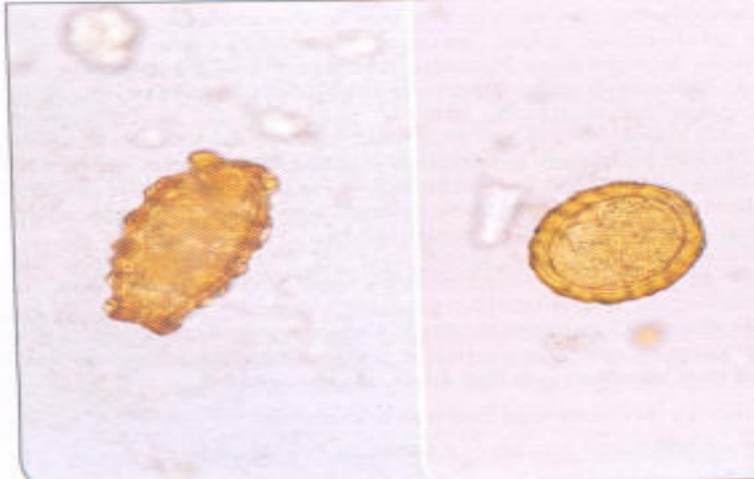
**Figure 1.3.** Life cycle of *Ascaris lumbricoides*

### Pathogenecity and clinical features

Adult worms in the intestine cause abdominal pain and may cause intestinal obstruction especially in children. Larvae in the lungs may cause inflammation of the lungs (Loeffler's syndrome) – pneumonia-like symptoms.

### Diagnosis

1. Examination of stool for eggs by direct saline smear method. The egg is ovoidal, 75x60 microns, covered by albuminous mamillatins.
2. Demonstration of adult worms



**Figure 1.4.** Egg of *Ascaris lumbricoides*

### **Treatment**

Mebendazole, Albendazole and Piperazine

### **2.1.2. HOOK WORMS**

There are two species of hookworm:

1. *Ancylostoma duodenale*
2. *Necator americanus*

The adults are found in the small intestines of man. Mixed infection is common.

Both of the species are found in Ethiopia, but *N. americanus* is more common.

#### **2.1.2.1. Ancylostoma duodenale:**

Grayish-white in color. The body is slightly ventrally curved. The anterior end follows the body curvature. The buccal cavity is provided ventrally with pairs of teeth and dorsally with a notched dental plate.

Distribution: This species is found in the northern part of the world including China, Japan, Europe, North Africa and Ethiopia.

### **Morphology**

**Male:** The male measures 10 cm in length. The posterior end is broadened into a membranous copulatory bursa that is provided with two long spicules.

**Female:** The female measures 12 cm in length. The posterior end is straight.

#### **2.1.2.2. Necator americanus**

This species, so called American hookworm, is found in predominantly the tropics. The anterior end is hooked against the body curvature. The mouth is provided ventrally and dorsally with cutting plate.

##### **Morphology**

**Male:** The male measures 8 cm in length. The posterior end is broadened into a membranous copulatory bursa, which is provided with two long spicules fused distally.

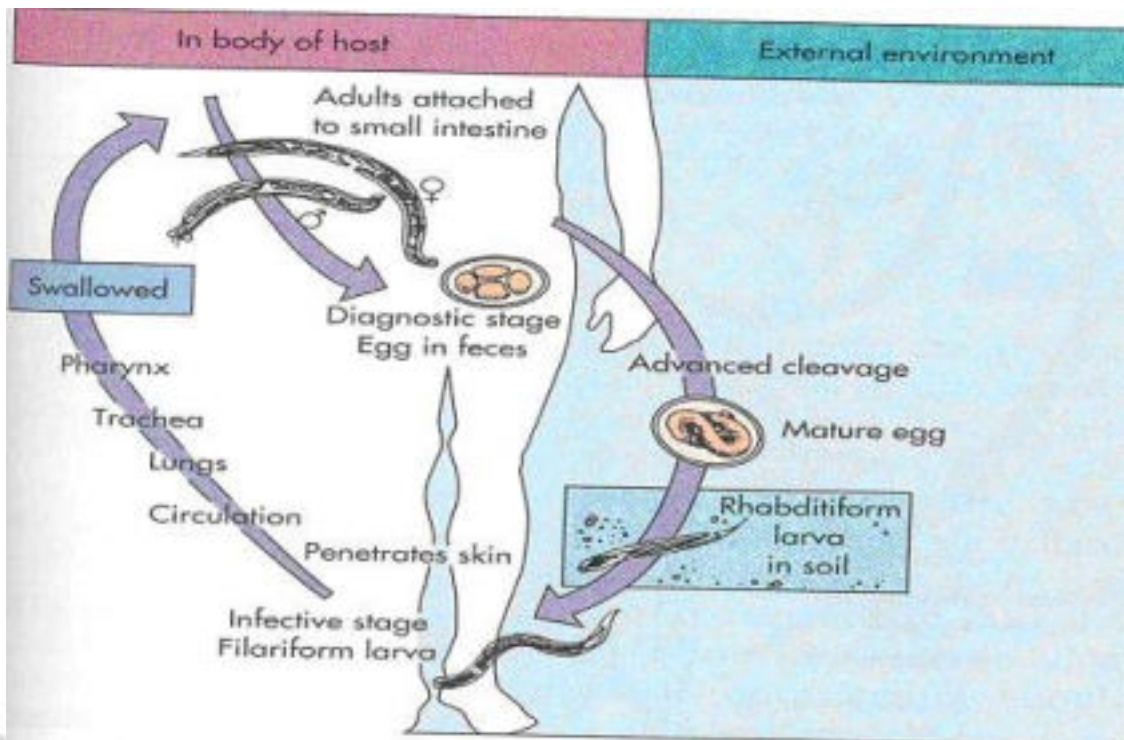
**Female:** The female measures 10 cm in length. The posterior end is straight

##### **Infective stage and methods of infection:**

The filariform larva infects by skin penetration.

##### **Life cycle**

Adult male and female worms live in the small intestine. The female lays eggs (oval, 60x40 microns), which contain immature embryo in the 4 cell stage. When the eggs pass in the stool to the soil and under favorable conditions of temperature, moisture and oxygen, they hatch into larvae, which molt twice and become infective. When the filariform larvae penetrate the skin, they circulate in the blood, reach the lungs, ascend to the trachea, descend to esophagus to reach the small intestine and become adults.

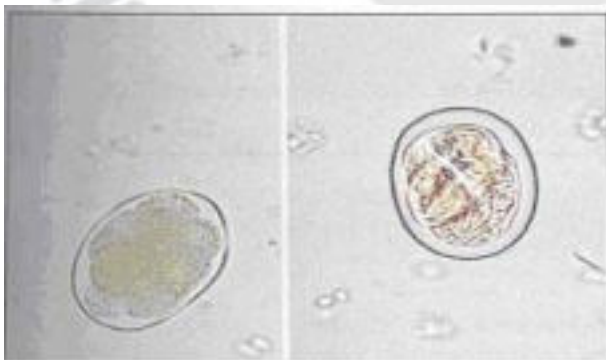


**Figure1.5.** Life cycle of hookworms

### Pathogenecity

Adult worms in the intestine feed on blood causing iron deficiency anemia. The larvae may cause inflammation of the lungs.

**Diagnosis:** Examination of stool by direct saline smear to detect the eggs.



**Figure1.6.** Egg of hookworm

## **Treatment**

Mebendazole: 1 tab 2x daily for 3 days.

### **2.1.3. LARVA MIGRANS**

There are three types of larva migrans:

#### **a. Cutaneous larva migrans (Creeping eruption)**

Various animals harbor hookworms. Two species of dogs and cats are important.

1. *Ancylostoma braziliens*: infects both dogs and cats.
2. *Ancylostoma caninum*: infects only dogs.

Both of these are common in the tropics and subtropical regions where human hookworms can best complete their life cycles. If man comes in contact with infective larvae, penetration of the skin may take place; but the larvae are then unable to complete their migratory cycle. Trapped larvae may survive for weeks or even months, migrating through the subcutaneous tissues. They may evoke a fairly severe reaction - pruritus and dermatitis. The dermatitis leads to scratching and then bacterial superinfection.

#### **Treatment**

Thiabendazole: Applied topically.

#### **b. Visceral larva migrans**

A syndrome caused by the migration of parasitic larvae in the viscera of a host for months or years. It may be caused by transient larval migration in the life cycles of several parasites such as hookworm, *Ascaris lumbricoides*, *T. spiralis*, *S. streccoralis* and other filarial worms.

#### **Toxocariasis**

This is a kind of visceral larva migrans caused by

- ♦ *Toxocara canis* (Dog ascarid) and



- ◆ *Toxocara cati* (Cat ascarid).

These cause persistent larval migration and thus the visceral larva migrans is called toxocariasis.

### **Morphology**

- ◆ The larvae of *Toxocara canis* and *Toxocara cati* measure about 400 µm in length.
- ◆ The life cycle of these parasites in their respective hosts is similar to that of *A. lumbricoides* in humans.

### **Epidemiology**

Visceral larva migrans is cosmopolitan in distribution.

### **Transmission:**

Ingestion of eggs of *Toxocara* species in contaminated food or soil or direct contact with infected patients. Children are more at risk.

### **Clinical features:**

- ◆ Majority are asymptomatic.
- ◆ Eosinophilia
- ◆ Cerebral, myocardial and pulmonary involvement may cause death.

**Diagnosis** - Identification of larvae in tissue.

**Treatment** - Thiabendazole: 25 mg/kg twice daily for 5 days.

### **C. Intestinal larva migrans**

This is an extremely rare kind of larva migrans



#### 2.1.4. STRONGYLOIDES STERCORALIS

The worms may be present as parasitic in the host or free living in the soil.

##### **Morphology:**

**Male:** The male measures 1 mm in length with curved posterior end and carries two spicules

**Female:** The female measures 2.5 mm in length with straight posterior end.

**Infection:** follows skin penetration by filariform larvae.

##### **Life cycle**

Adult male and female worms live in the small intestine. After fertilization, the female penetrates the mucosa of the small intestine and lay eggs in the submucosa. The eggs hatch and the larvae penetrate the mucosa back to the lumen. If the environmental conditions are favorable, the larvae will come out with the stool to the soil. They transform into adults, which lay eggs, and hatching larvae get transformed to adults and so on. If the environmental conditions are not favorable, the larvae in the stool will moult and transform into infective filariform larvae, which pierce the intestine (auto-infection). Larvae penetrating the skin from the soil or by autoinfection are carried by the blood to the lungs, ascend to the trachea, descend to the esophagus and mature in the small intestine.

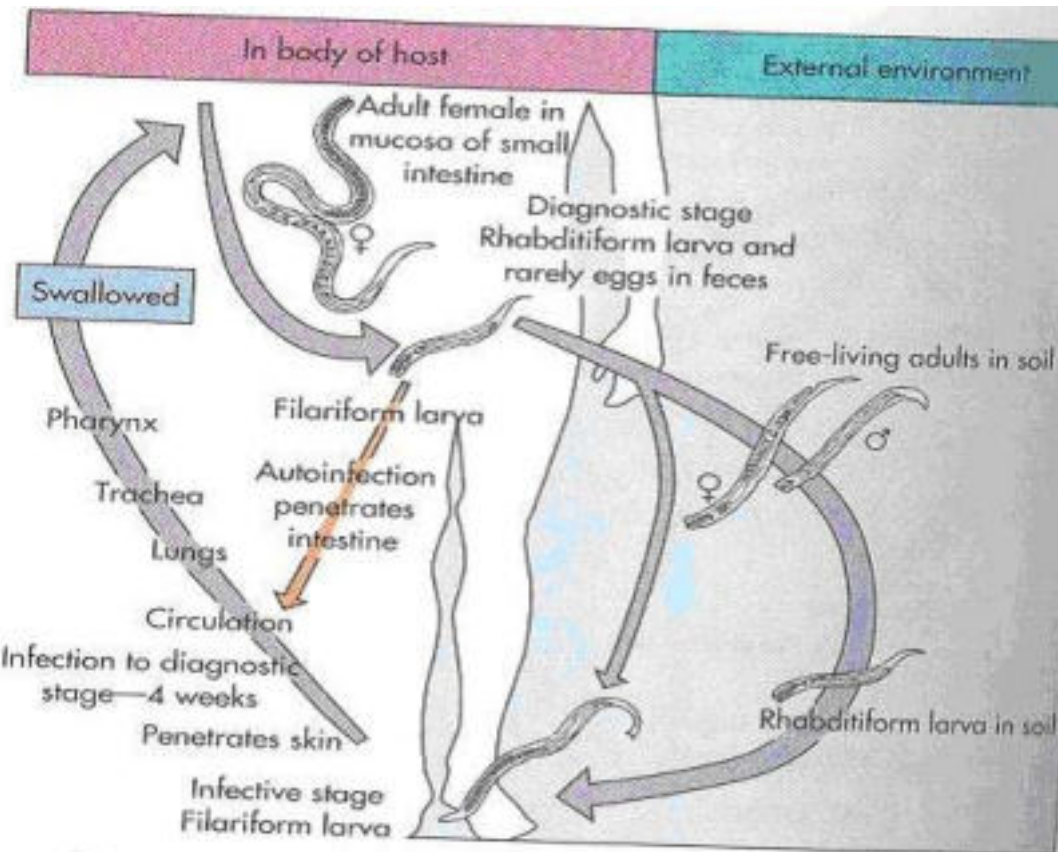


Figure 1.7. Life cycle of *Strongyloides stercoralis*

### Clinical presentation

The patient complains of mucoid diarrhea. Larvae in the lungs may cause pneumonia.

### Disseminated strongyloidiasis:

Multiplicity of symptoms are present due to the injury of other organs by the migrating larvae. Organs such as liver, heart adrenals, pancreas, kidneys, and CNS, etc. may be affected. This is usually seen in immunocompromized individuals.

**Diagnosis** - Detection of rhabditiform larvae of strongyloides in stool.



**Figure 1.8.** Larva of *S. stercoralis*

#### **Treatment:**

Thiabendazole: 25 mg/kg twice daily for 3 days.

## **2.2. INTESTINAL NEMATODES WITHOUT TISSUE STAGE**

### **2.2.1. ENTEROBIUS VERMICULARIS (PIN WORM OR THREAD WORM)**

*Enterobius vermicularis* is a small white worm with thread-like appearance. The worm causes enterobiasis. Infection is common in children.

#### **Morphology**

**Male:** The male measures 5 cm in length. The posterior end is curved and carries a single copulatory spicule.

**Female:** The female measures 13 cm in length. The posterior end is straight.

#### **Infective stage**

Infection is by ingestion of eggs containing larvae with contaminated raw vegetables.

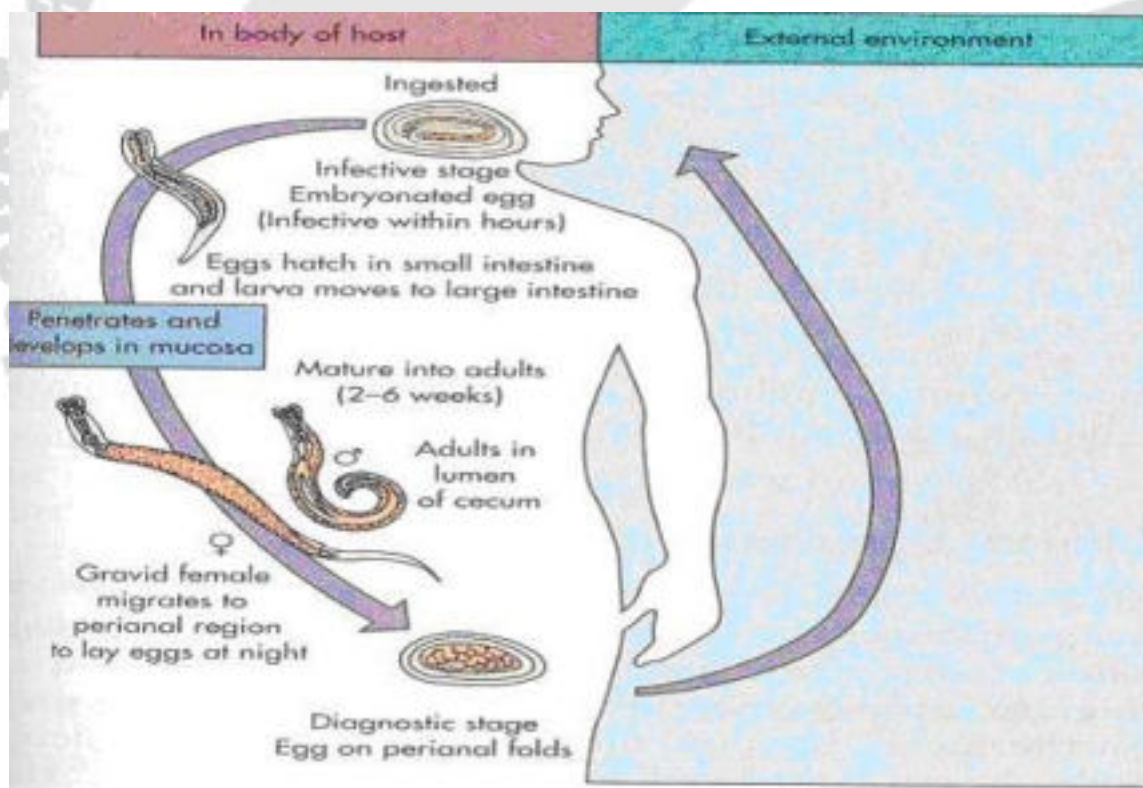
#### **Mode of infection**

- By direct infection from a patient (Fecal-oral route).

- Autoinfection: the eggs are infective as soon as they are passed by the female worm. If the hands of the patient get contaminated with these eggs, he/she will infect him/herself again and again.
- Aerosol inhalation from contaminated sheets and dust.

### Life cycle

Adult worm lives in the large intestine. After fertilization, the male dies and the female moves out through the anus to glue its eggs on the peri-anal skin. This takes place by night. The egg is 50x25 microns, plano-convex and contains larva. When the eggs are swallowed, they hatch in the small intestine and the larvae migrate to the large intestine to become adult.



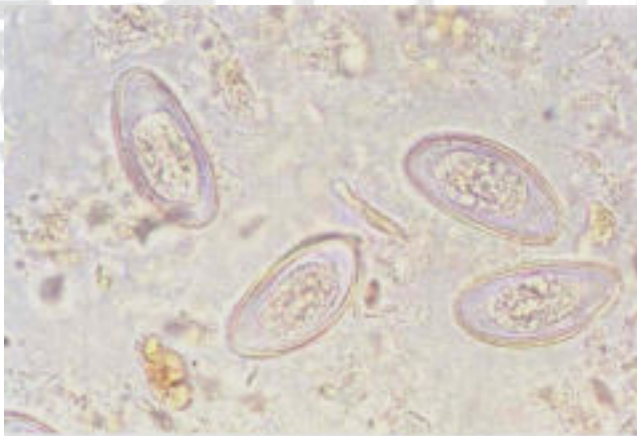
**Figure 1.9.** Life cycle of *E. vermicularis*

## Clinical presentation

The migration of the worms causes allergic reactions around the anus and during night it causes nocturnal itching (pruritus ani) and enuresis. The worms may obstruct the appendix causing appendicitis.

## Diagnosis

- ◆ Eggs in stool: Examination of the stool by direct saline smear to detect the egg: this is positive in about 5% of cases because the eggs are glued to the peri-anal skin.
- ◆ Peri-anal swab: The peri-anal region is swabbed with a piece of adhesive tape (cellotape) held over a tongue depressor. The adhesive tape is placed on a glass slide and examined for eggs. The swab should be done in the early morning before bathing and defecation.



**Figure 1.10.** Egg of *E. vermicularis*

## Treatment

Mebendazole; Piperazine.



### 2.2.2. TRICHURIS TRICHIURA (WHIP WORM)

The worm is divided into a thin whip-like anterior part measuring  $\frac{3}{5}$  of the worm and a thick fleshy posterior part of  $\frac{2}{5}$  the length.

**Male:** The male measures 3-4.5 cm in length. Its posterior end is coiled and possesses a single cubicle.

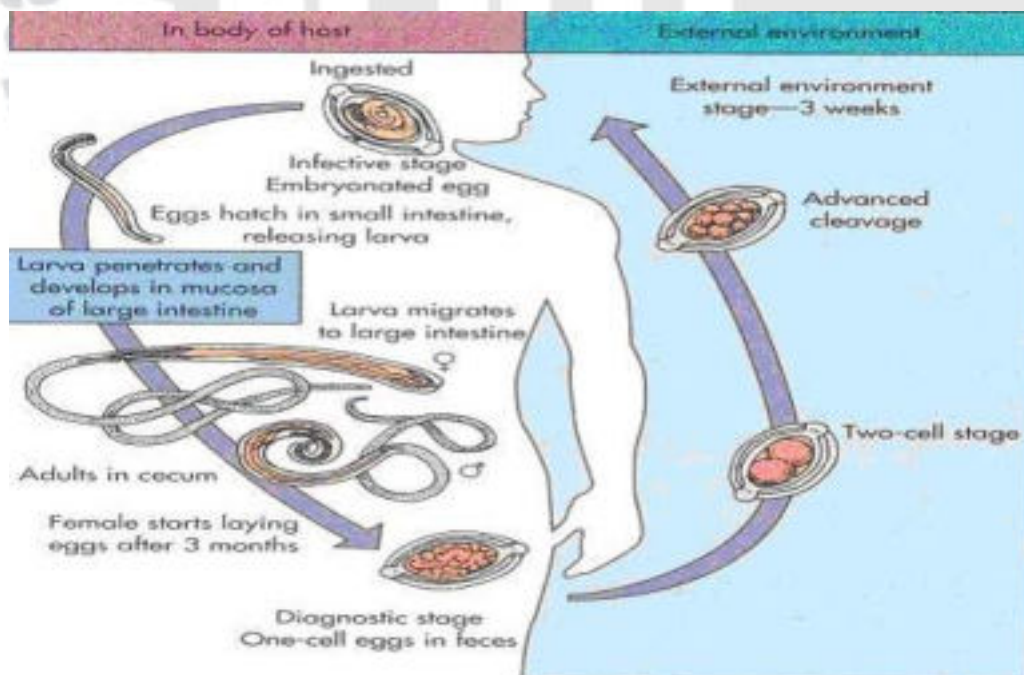
**Female:** The female measures 4-5 cm in length. Its posterior end is straight

#### Infective stage and mode of infection

Infection is by ingestion of eggs containing larvae with contaminated raw vegetables.

#### Life cycle:

Ingested eggs hatch in the small intestine and the larvae migrate to the large intestine to become adult. After mating, the female lays immature eggs, which pass with the stool to the soil and mature in 2 weeks.



**Figure 1.11.** Life cycle of *Trichuris trichiura*



## Symptoms

The patient complains of dysentery (blood and mucus in stool together with tenesmus). Rectal prolapse is also possible.

## Diagnosis

Finding of characteristic eggs. The egg of trichuris is barrel-shaped, 50x25 microns. The shell is thick with a one mucoid plug at each pole.



**Figure 1.12.** Egg of *Trichuris trichiura*

## Treatment

Mebendazole: 1 tablet twice daily for 2 days.

## 2.3. TISSUE NEMATODES

This group includes the filarial worms, the guinea worm (*Dracuncululus medinensis*) and *Trichinella spiralis*.

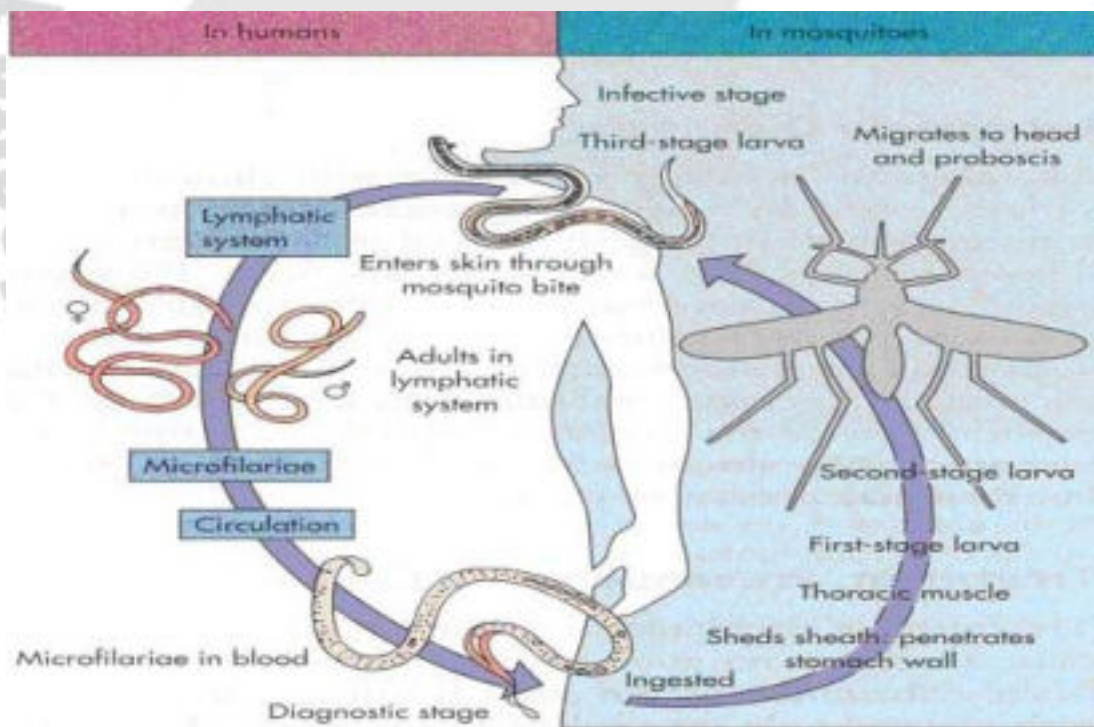
### 2.3.1. FILARIAL WORMS

The filarial worms have complex life cycles involving a developmental stage in an insect vector. They require an arthropod vector for their transmission. The worms inhabit either the lymphatic system or the subcutaneous tissues of man. The

female worm gives rise to a young worm called microfilaria. The microfilariae, when taken by the arthropod intermediate host during biting, develop into filariform larvae, which are the infective stages. Humans get infected when bitten by the infected arthropod intermediate host.

#### 2.3.1.1. *Wuchereria bancrofti*

This is a parasite of lymph nodes and lymphatic vessels- causing lymphatic filariasis. This filarial worm is transmitted by the bite of various species of mosquitoes. It is believed that over 100 million people are infected. The microfilariae are nocturnal – seen in greatest numbers in peripheral blood in the night between 10 PM -2 AM. The physiological basis of this nocturnal periodicity is not understood.



**Figure1.13.** Life cycle of *W. bancrofti*

## Mode of transmission and pathogenesis

The filariform larvae are introduced through the skin by the bite of the arthropod intermediate host. The larvae invade the lymphatics, usually the lower limb, where they develop into adult worms. The microfilariae are librated into the blood stream. They remain in the pulmonary circulation during day, emerging into the peripheral circulation only during night, to coincide with the biting habit of the vector. Presence of the adult worms causes lymphatic blockage and gross lymphedema, which sometimes lead to elephantiasis.

**Epidemiology:** *W. bancrofti* infection is not reported in higher altitudes of Ethiopia, but limited to lowlands of Gambella. The epidemic area covers a long distance along the Baro River.

### Pathogenecity and clinical features:

- ◆ The adult worm obstructs the flow of lymph in the lymph nodes and the lymphatic vessels draining the lower limbs and the external genitalia.
- ◆ The lower limbs and external genitalia become swollen. The skin becomes thick and fissured. The disease is called bancroftian elephantiasis.
- ◆ The major symptoms and findings include: lymphangitis, lymphedema, fever, headache, myalgia, hydrocele and chyluria.

### Diagnosis

- ◆ Blood film examination after staining by Giemsa or Leishman stain to detect microfilaria. The film should be taken by night.



**Figure 1.14.** Microfilaria of *W. bancrofti* in blood smear

**Treatment** - Diethyl carbamazine (DEC): 2 mg/kg 3x daily for 2 weeks.

### **Endemic non-filarial elephantiasis (Podoconiosis)**

Non-filarial elephantiasis of the lower limbs is common in Ethiopia. Silicon, aluminium and iron particles in the red clay soil are absorbed through skin abrasions in bare footed persons. The mineral particles cause obstruction of the lymphatics.

#### **2.3.1.2. *Onchocerca volvulus***

Infection by this filarial worm is common in Ethiopia. Endemic foci are found in Bebek, Gojeb valley, Dedessa valley, Agaro, Metekel, and in Northwestern Ethiopia around Gondar.

#### **Morphology:**

**Male:** Similar to that of *Wuchereria bancrofti*.

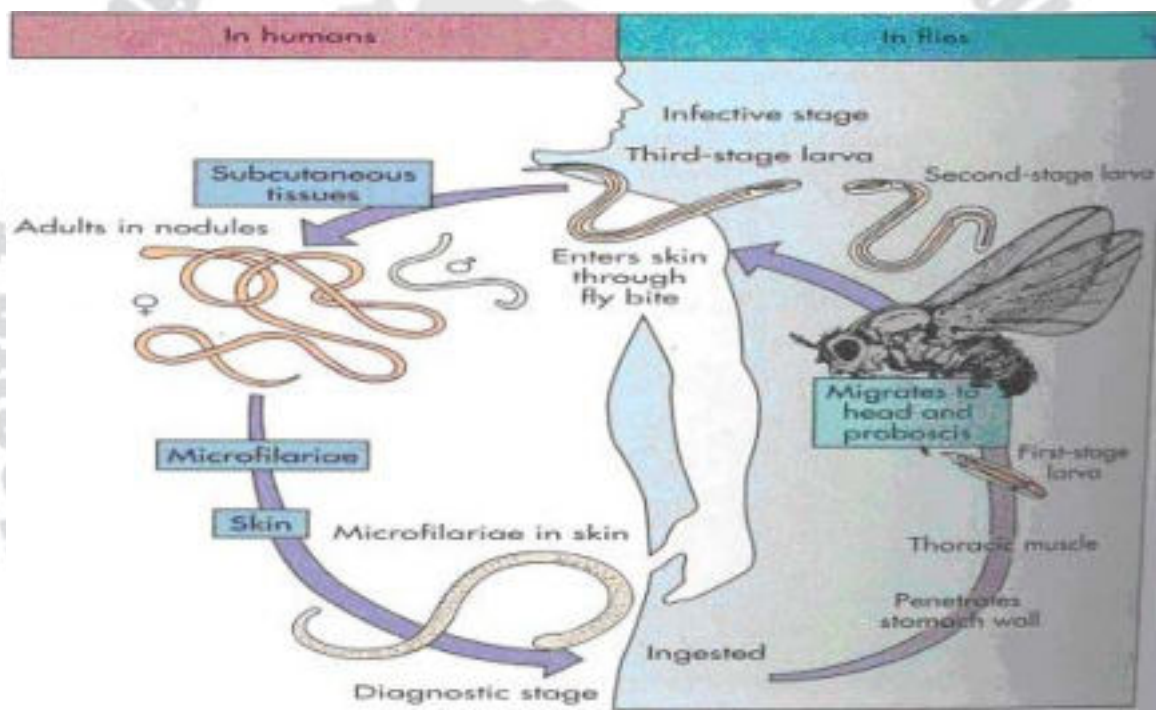
**Female:** The female measures 30-50 cm in length. It is present inside of a fibrous nodule (onchocercomata or onchocerca tumor).

## Intermediate Host and vector

Female Simulium, (*Simulium damnosum*), Black fly, found around plantations following rivers or river basins.

## Microfilaria

Measures 300 microns in length. It is non-sheathed microfilaria. It is present in the subcutaneous tissue fluids and not in blood.



**Figure 1.15.** Life cycle of *O. volvulus*

**Infective stage and mode of infection** is similar to that of *Wuchereria bancrofti*.

## Pathogenecity and clinical manifestations:

The disease, onchocerciasis or river blindness includes:

- Skin fibrous nodules (onchocercomata) enclosing female worms. The nodules are common in neck, iliac crest and the coccyx.



- Skin hypo- or hyper- pigmentation. Dermatitis is present. In advanced cases, the skin becomes thickened and wrinkled, showing lizard or leopard skin appearance.
- Elephantiasis of the external genitalia and corneal opacity and optic atrophy may finally cause blindness.

### Diagnosis

Superficial biopsy (skin snip) is taken from the skin using sharp razor blade. The specimen is allowed to stand for 30 minutes in saline before it is examined microscopically for microfilariae.



**Figure 1.16.** Microfilaria of *O. volvulus*

### Treatment

Ivermectin: 50 mg/kg bodyweight, given every 6 or 12 months. Because it kills microfilariae but not adult worms, retreatment is necessary over a period of years.

### Prevention

- Vector control
- Mass treatment



- Establishment of villages away from Simulium breeding places.
- Use of repellents
- Protective clothing

#### **2.3.1.3. Loa loa**

The eye worm, *Loa loa*, causes Loiasis. The insect vectors include mango flies of Chrysops - *Chrysops silacea*, *Chrysops dimidiata*. Loiasis is endemic in Central and West Equatorial Africa. The abundant rubber plantations provide a favorable environment for the vector to transmit the disease.

#### **Morphology**

Adult male worms: 30-34 mm in length

Adult female worms: 40-70 mm in length

#### **Pathogenesis**

The microfilaria have a sheath. Their diurnal periodicity corresponds to the feeding pattern of the insect vector, which bites humans from 10:00 AM to 4:00 PM.

#### **Clinical Features**

Incubation period is about one year. It causes calabar swelling beneath the skin due to parasites. There is fever, pain, pruritus, urticaria, allergic reactions, retinopathy, glomerulonephritis, meningo-encephalitis etc.

#### **Laboratory diagnosis**

- Detection of microfilaria in peripheral blood, urine, sputum, CSF - stained with Giemsa or unstained
- Eosinophilia

## Treatment

DEC, 6 to 10 mg per kilogram per day for 2 to 3 weeks: but has side effects - allergic reactions

### 2.3.2. DRACUNCULUS MEDINENSIS (Guinea worm or Medina worm)

*Dracunculus medinensis* causes dracunculiasis. The infection is endemic to Asia and Africa: India, Nile Valley, central, western and equatorial Africa, lowlands of Ethiopia and Eritrea.

#### Morphology

Gravid female worms measure 70-120 cm in length. Their body cavity is almost fully occupied by a uterus greatly distended with rhabditiform larvae (250-750  $\mu\text{m}$  in length). A digestive tube and cuticular annulations distinguish the larvae from microfilariae.

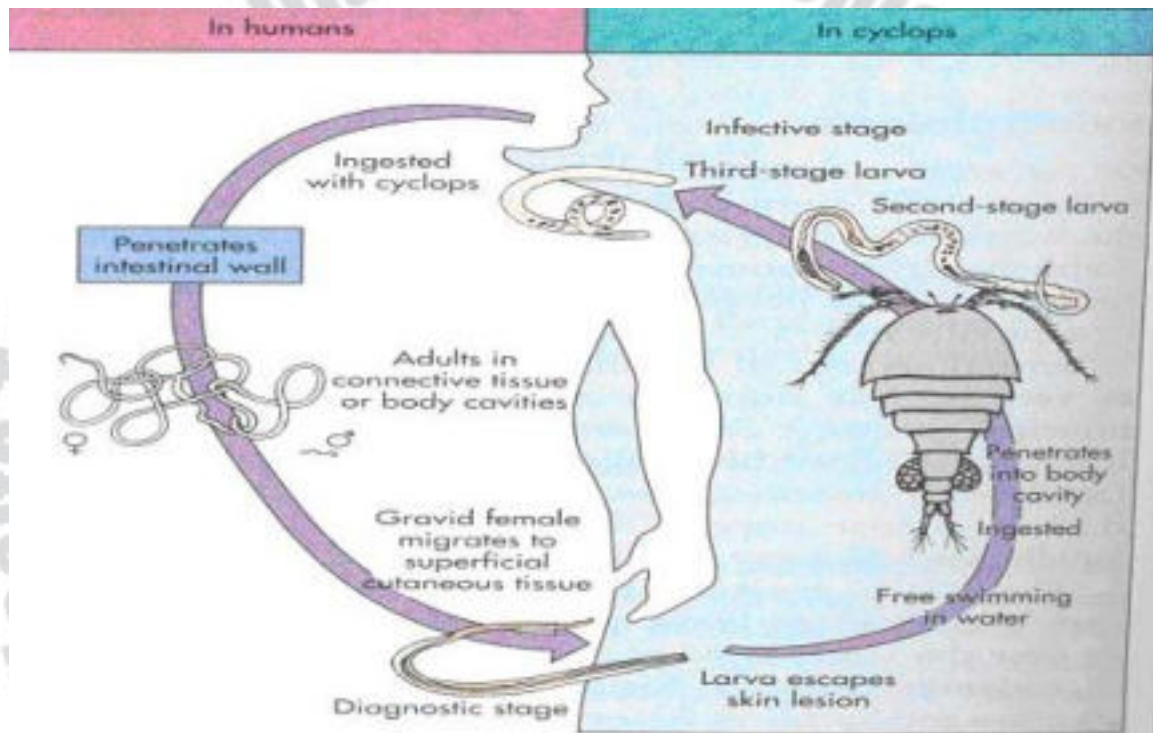


**Figure 1.17** Larvae of *D. medinensis*

#### Pathogenecity and life cycle

Infection is acquired by drinking unfiltered or not boiled water that contains Cyclops species. The larvae are released in the stomach, penetrate the intestinal wall and find their way to the subcutaneous tissue. Mating takes place in the axillary or inguinal regions 3 months after infection. The male worms then die in the tissue and the female worms move down to the limbs within 10 months. In

about 1 year, female worms in the subcutaneous tissue provoke the formation of a burning blister in the skin of the legs. When in water, the blister bursts, and about 5 cm of the worm is extruded from the resulting ulcer - thus releasing many thousands of first stage larvae. The larvae swim in water and are ingested by the intermediate host - Cyclops species- within about 4 days. Inside the Cyclops, the larvae molt twice and become infective in 2 weeks



**Figure 1.18.** Life cycle of *Dracunculus medinensis*

### Clinical feature

The female parasites in the subcutaneous tissue release toxic byproducts of histamine-like nature, which cause systemic allergic reactions, like erythema, urticaria, pruritus, fainting, asthma, dyspnea, etc. This is followed by the appearance of a blister on the legs, which ruptures on contact with water releasing larvae into the water by the female worm. The wound may ulcerate. The worms migrate into other tissues and may cause arthritis, pericarditis, abscesses etc. It occasionally penetrates the eyeball and causes loss of the eye.

## Diagnosis

1. Clinical: Observation of blister, worm or larvae
2. Histologic features of subcutaneous sinus tract
3. Eosinophilia and radiographic evidence

## Treatment

Surgical excision when the worm is in the leg  
Niridazole (Ambilhar) or DEC

## Prevention

Health education on:

- ◆ Boiling or filtering of drinking water
- ◆ Treating of patients and educating them not to enter water bodies
- ◆ Using insect larvicides to kill Cyclops in water.

### 2.3.3. TRICHINOSIS

***Etiologic agent - Trichinella spiralis***

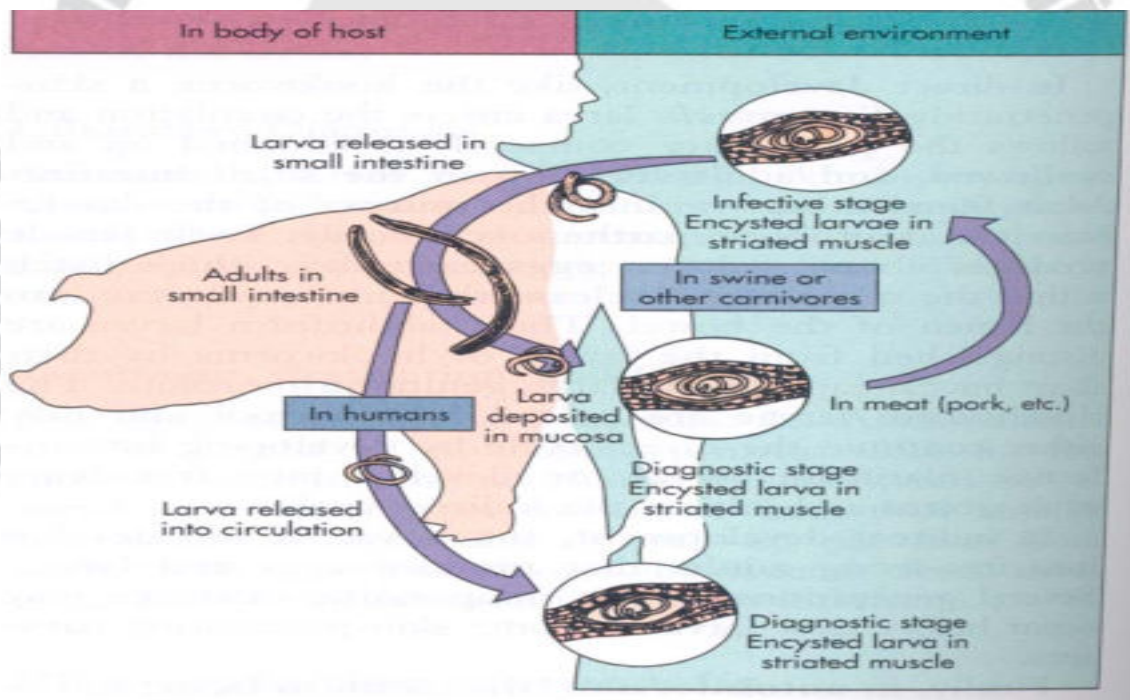
This is the only important species in this group. It causes trichinosis - a cosmopolitan infection. More than 100 different animal species can be infected with *Trichinella* species, but the major reservoir host for human infections is swine.

### Morphology

Adult female worm measures 3-4 mm in length and the adult male worm measures 1.4-2.6 mm in length. The encysted larvae measure 800-1300  $\mu\text{m}$  in length.

## Pathogenecity and life cycle

After ingesting infected meat, the capsule of the encysted larvae is digested by gastric juice, and the larvae are released in the duodenum or jejunum where they molt four times to become adult worm. After mating, the male worm dies and the female worm begins to deliver the embryos 4-7 days after the infection. The larvae penetrate the intestinal wall and migrate through the lymphatic vessels to the blood stream, which carries them to various organs. Skeletal muscles and diaphragm are most frequently parasitized. Others include the tongue, masseter and ocular muscles.



**Figure 1.19** Life cycle of *Trichinella spiralis*

## Clinical features

There are two clinical phases.

1. The intestinal phase: lasting 1-7 days - asymptomatic; sometimes cause nausea, vomiting, diarrhea, constipation, pain, etc, and

2. The muscle phase: which causes myalgia, palpabral edema, eosinophilia, fever, myocarditis, meningitis, bronchopneumonia etc.

**Diagnosis:**

- ◆ Muscle Biopsy
- ◆ Detection of larvae in blood or CSF
- ◆ Detection of larvae and adult worms in stool (rare).
- ◆ ELISA

**Treatment** - Thiabendazol

**Prevention**

- ◆ Cooking of all meat before consumption
- ◆ Inspection of pigs
- ◆ Pork must be stored at  $-15^{\circ}\text{C}$  for 20 days.



## **UNIT TEN**

### **CESTODES (TAPEWORMS)**

#### **INTRODUCTION**

The tapeworms are hermaphroditic and require an intermediate host. The adult tapeworms found in humans have flat body, white or grayish in color. They consist of an anterior attachment organ or scolex and a chain of segments (proglottids) also called strobilla. The strobilla is the entire body except the scolex. The scolex has suckers or grooves. It has rosetellum, which has 1 or 2 rows of hooks situated on the center of the scolex.

Adult tapeworms inhabit the small intestine, where they live attached to the mucosa. Tapeworms do not have a digestive system. Their food is absorbed from the host's intestine.

#### **3.1. HYMENOLEPIS NANA (DWARF TAPEWORM)**

##### **Morphology**

Adult worm measures 1-3 cm in length. It is made up of head (scolex), neck and segmented body. The head carries four suckers and a rostellum armed with one row of hooks. The segments of the body are divided into mature and gravid segments. In the mature segment, there are three testes in the middle.

##### **Infective stage and mode of infection**

The egg, which is immediately infective when passed by the patient, is rounded, about 40 microns in diameter. It contains a six-hooked oncosphere within a rigid membrane (the embryosphere). This embryosphere has two polar thickening or knobs from which project 4-8 long, thin filaments called polar filaments.

Infection takes place by:

1. Ingestion of egg with contaminated raw vegetables.
2. Direct infection from a patient
3. Auto infection: the eggs of *H. nana* are infective as soon as they are passed with feces by the patient. If the hands of the patient are contaminated by these eggs, she/he infects herself/himself again and again.

### **Pathogenecity**

Light infections produce no symptoms. In fairly heavy infections, children may show lack of appetite, abdominal pain and diarrhea.

**Treatment** - Niclosamide: 4 tablets chewed in a single dose daily for 5 days.

### **3.2. HYMENOLEPIS DIMINUTA (RAT TAPEWORM)**

*Hymenolepis diminuta* differs from *Hymenolepis nana* in that:

- ♦ The adult worm measures about 10-60 cm
- ♦ The rosetellum on the head has no hooks
- ♦ In the mature segment, there are two testes at one side and another testis on the other side.

### **Life cycle**

The adult worms are present in the small intestine of man and rats. Eggs passed in stool are similar to the eggs of *H. nana* but are brown in color with no polar filaments arising from the polar thickening. The eggs are ingested by the rat flea where they develop to cysticercoid stage. Infection to man takes place accidentally by food or contaminated hands by cysticercoid stage.

## **Pathogenecity**

Most infections are asymptomatic, but occasionally, patients may present with nausea, anorexia and diarrhea.

## **Treatment**

same as *Hymenolepis nana*.

### **3.3. ECHINOCOCCUS**

There are two different species. These are: *Echinococcus granulosus* and *Echinococcus multilocularis*

#### **3.3.1. Echinococcus granulosus (dog tape worm)**

Responsible for most cases of echinococcosis. Echinococcosis is caused by larval tapeworms. The disease is common in East Africa (the highest prevalence is seen in Kenya: 10-15%).

## **Morphology**

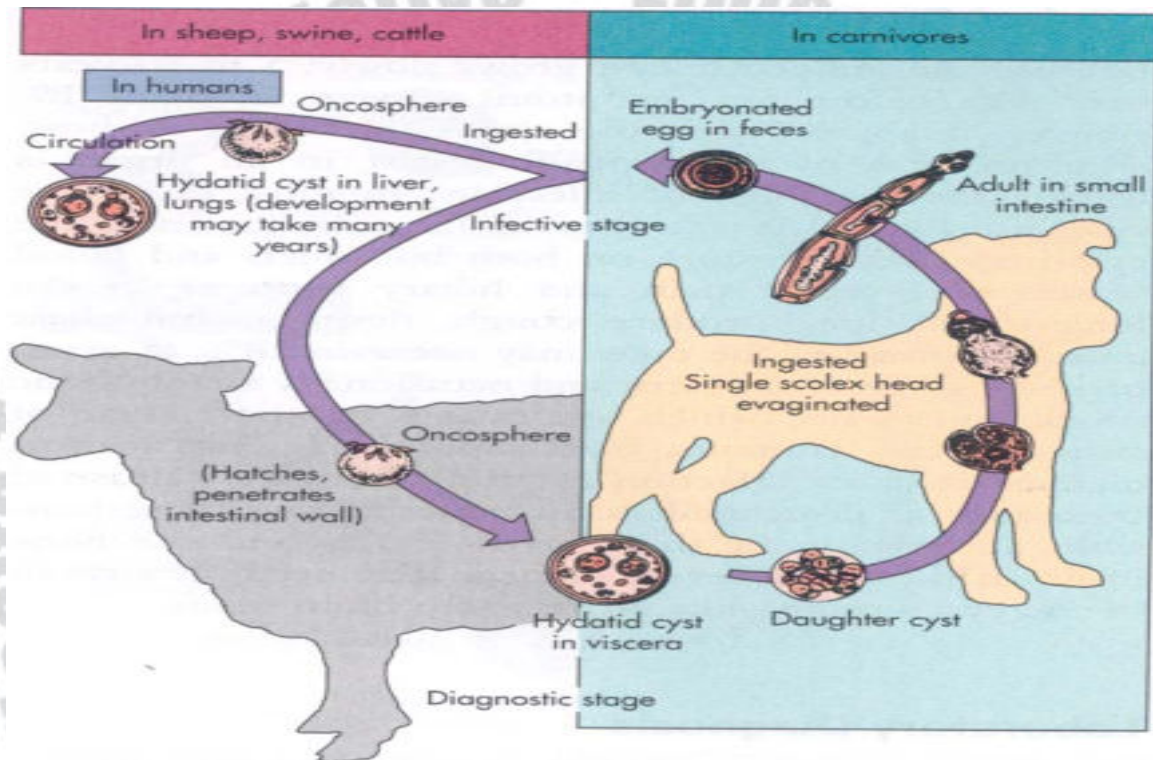
The adult worm measures 3-6 mm in length (up to 1 cm). It has scolex, neck and strobilla. Adult worms live in small intestine of definitive host (dog). Man is an intermediate host - carrying the hydatid cyst (larva). Man contracts infection by swallowing eggs in excreta of definitive host.

## **Life cycle and Pathogenecity**

Oncosphere hatch in duodenum or small intestine into embryos (oncosphere) which:

- ◆ Penetrate wall
- ◆ Enter portal veins

- ♦ Migrate via portal blood supply to organs: eg: lungs, liver, brain etc., thus, causing extra intestinal infections. In these organs, larvae develop into hydatid cysts. The cysts may be large, filled with clear fluid and contain characteristic protoscolices (immature forms of the head of the parasite). These mature into developed scolices, which are infective for dogs.



**Figure 1.20.** Life cycle of *Echinococcus granulosus*

### Mode of human infection

Ingestion of eggs by the following ways:

- i) Ingestion of water or vegetables polluted by infected dog feces.
- ii) Handling or caressing infected dogs where the hairs are usually contaminated with eggs.

### Clinical features

Asymptomatic infection is common, but in symptomatic patients

- ◆ It may cause cough - with hemoptysis in lung hydatid disease.
- ◆ Hepatomegaly - with abdominal pain and discomfort
- ◆ Pressure -from expanding cyst
- ◆ Rupture of cyst - severe allergic reaction - anaphylaxis.

#### **Diagnosis:**

- ◆ X-ray or other body scans
- ◆ Demonstration of protoscolices in cyst after operation
- ◆ Serology

#### **Treatment**

- ◆ Surgery
- ◆ Albendazole 400 mg twice a day for one to eight periods of 28 days each, separated by drug-free rest intervals of 14 to 28 days.

#### **3.3.2. *Echinococcus multilocularis***

Foxes are the definitive hosts, while various rodents such as mice serve as intermediate hosts.

#### **3.4. TAENIA SAGINATA (BEEF TAPEWORM)**

In adult stage, *T. saginata* inhabits the upper jejunum where it may survive for as long as 25 years. It causes intestinal infection, Taeniasis. It has worldwide distribution.

These are one of the true and segmented tapeworms. Their body is divided into three regions;

1. Scolex: the hold fast organ
2. Neck: posterior to the scolex
3. Stobilla: the main bulk, made up of proglottids.

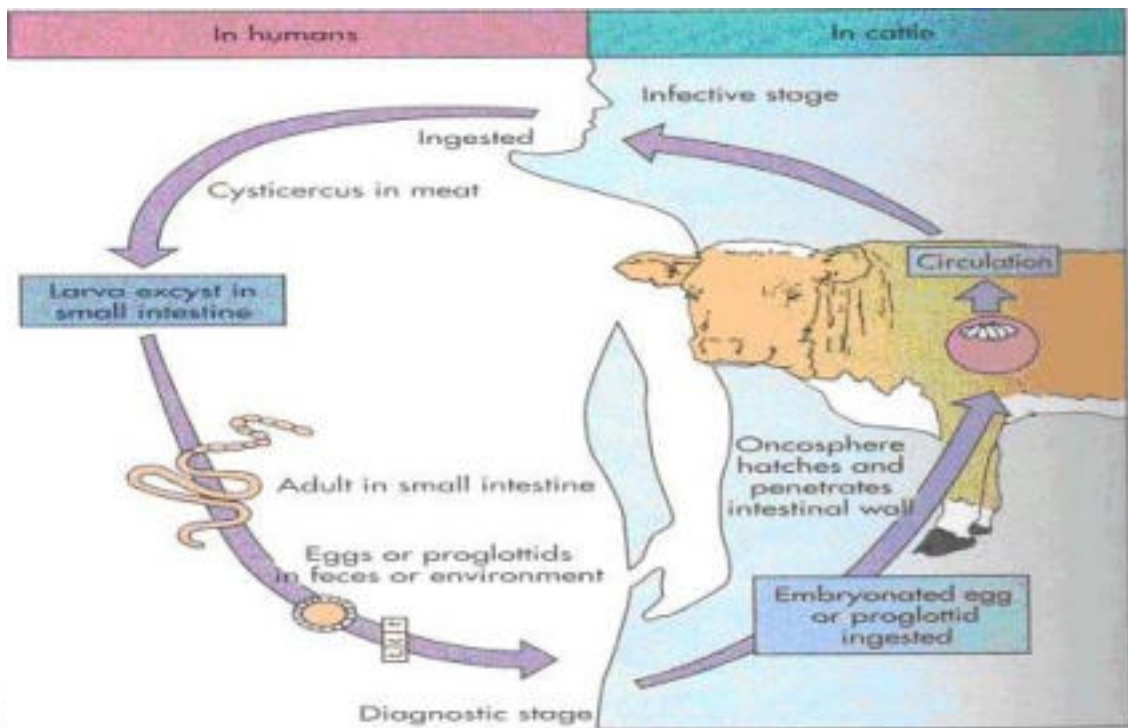
### **Morphology:**

Adult worm measures 5-10 meters in length. The pyriform scolex has 4 suckers but no rostellum. The mature segments have irregularly alternate lateral genital pores. Each of the terminal segments contains only a uterus made up of a median stem with 15-30 lateral branches.

### **Life cycle**

The adult worm lives in the small intestine of man. Gravid segments pass out in the stool and become disintegrated and eggs come out to the soil. The gravid proglottid uterus contains about 100,000 eggs. The egg of *T. saginata* is round, about 40 microns in diameter. The 6-hooked embryo is enclosed in a radially striated embryophore. Eggs are ingested by an intermediate host, cattle. The 6-hooked embryo escapes from its shell, penetrates through the intestinal wall into the blood vessels and is carried to the muscles where it develops into a larval stage, **cysticercus bovis** (made up of an invaginated /inverted head and spherical body). Infection to man takes place by the ingestion of raw or insufficiently cooked beef. In the small intestine of man, the head of the cysticercus gets invaginated and the body becomes segmented.





**Fig 1.21.** Life cycle of *Taenia saginata*

### Pathogenecity

Infected persons may complain of epigastric pain, abdominal discomfort, diarrhea, weight loss, hunger sensation, vomiting, etc.

### Diagnosis

Recovery of the gravid segments or the eggs from the stool



**Figure1.22.** Egg of *Taenia* Spp

**Treatment:**

Niclosamide: Four tablets chewed in a single dose.

Mebendazole 100mg twice daily for three days

**Prevention:**

- ◆ Thorough cooking of meat (above 57°C).
- ◆ Proper disposal of human excret

**3.5. TAENIA SOLIUM (PORK TAPEWORM)**

The adult worms of *T. solium* reside or inhabit the upper jejunum. Infection has worldwide distribution.

**Morphology:**

Adult worm measures about 3 meters in length. The globular scolex has rostellum with 2 rows of hooklets. There are <1000 proglottids.

Gravid proglottid liberates about 30,000-50,000 eggs.

**Life cycle**

Embryonated eggs passed with stool are ingested by pig and the embryo is released. It penetrates the intestinal wall and is carried by vascular channels to all parts of the body. After a period of 2-3 months of development the encysted larval stage called cysticerci or bladder worm occurs in the striated muscles of the tongue, neck, trunk brain, eye, and the nervous system. The cysticercus survives for 5 years. Humans become infected by eating pork containing larvae, *cysticercus cellulosae*. When improperly cooked cysticercus infected meat is eaten by man, the scolex remains undigested and attaches itself to the intestinal wall and chain of proglottids begin to grow to adult worm.

### Clinical manifestations

Resembles that of *T. saginata* infection

### Diagnosis

Demonstration of eggs in stool specimen

### Treatment

Niclosamide: 2 gm PO stat

### Prevention:

- ◆ Treatment of infected persons.
- ◆ Thorough cooking of pork and proper processing
- ◆ Proper disposal of human excreta (good hygiene/sanitation).

**Table 1:** Comparison between *Taenia saginata* and *Taenia solium* species

|                   | <i>Taenia saginata</i> | <i>Taenia solium</i> |
|-------------------|------------------------|----------------------|
| Length (m)        | 5-10                   | 2-3                  |
| Proglottid number | 1000-2000              | 800-900              |
| Hooklets          | Absent                 | Present              |
| Suckers           | Pigmented              | Non- Pigmented       |
| Uterus branch     | 15-30                  | 5-10                 |

### 3.6. DIPHYLOBOTRIUM LATUM (FISH TAPEWORM OR BROAD TAPEWORM)

The broad tapeworm infecting man has worldwide distribution, occurring in areas where improperly cooked or raw fresh water fish is prominent in diet.

### Morphology

*Diphylobotrium latum* is the broadest and longest tapeworm. The adult worm measures up to 30 feet with 3000-4000 proglottids, which are wider than they are long. The tapeworm has no rostellum hooks or suckers.

## **Life cycle**

Unlike Taenia, the gravid segments are retained by the worm. Operculated eggs passed in feces hatch into small ciliated coracidium larvae which swim about freely. These are eaten by crustaceans -Cyclops or Diaptomus - in which the larvae develop into second stage larvae- the proceroid. When the crustaceans are swallowed by fresh water fish, the larvae migrate into the flesh of the muscle fish and develop to pleuroceroid or sparganum larvae. Humans are infected by ingesting raw or improperly cooked fish. The tapeworm matures in the intestine and after 3 weeks, the adult worm discharges eggs. The life cycle requires two intermediate hosts.

So far there is no report of the parasite in Ethiopia.

## **Clinical manifestation**

Most infections are asymptomatic. Rarely, it causes severe cramping, abdominal pain, vomiting, weakness and weight loss. Pernicious anemia can also result, due to interference of vitamin B<sub>12</sub> absorption in jejunum.

## **Diagnosis**

Eggs in stool: Single shell with operculum at one end and a knob on the other.

## **Treatment**

Niclosamide: 2 gm PO stat after light breakfast.

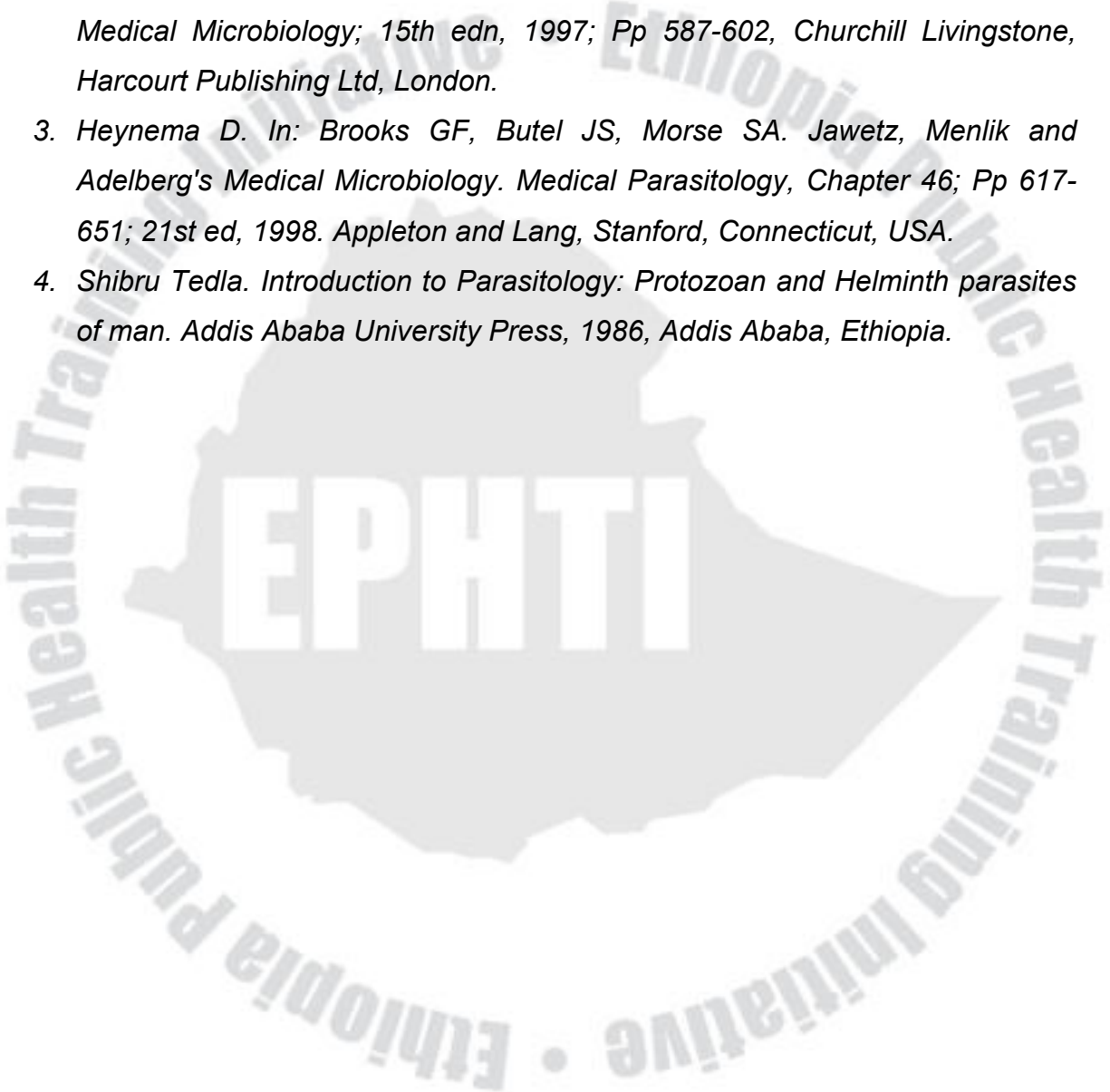
## **Prevention:**

Prohibiting the disposal of untreated sewage into fresh water /lakes.

Personal protection: cooking of all fresh water fish.

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# **UNIT ELEVEN**

## **MEDICAL ENTOMOLOGY**

### **LEARNING OBJECTIVES**

At the end of this chapter the student is expected to:

- Describe the general features of all arthropods.
- Explain the medical importance of arthropods.
- Identify the difference between mechanical and biological carriers.
- List the different types of biological carriage.
- Discuss the classification of arthropods.
- List some of the most important vector control measures.

### **INTRODUCTION**

Medical entomology is a science, which deals with the study of arthropods. Members of the phylum arthropoda are the most numerous and widely distributed of all animal groups. Their medical importance lies in their ability to cause morbidity and mortality, and their extensive distribution over the face of the earth. They may be found in every part of the world and in every type of environment. Many, particularly those within the class insecta and arachnida, live in close association with humans; others while primarily parasites of animals, will readily attack or feed upon humans and some may specifically adapt as human parasites.

### **ARTHROPODS**

The arthropods include animals varying considerably in size and shape but have fundamental features in common.

Generally all arthropods have the following characteristics in common:

- They are bilaterally symmetrical.



- Their bodies are divided into a number of rings or segments.
- They have jointed appendages, which may take the form of legs, antennae, or mouthparts.
- They have a hard chitinous exoskeleton (cuticle), which helps for the protection and insertion of muscles.
- The exoskeleton is partitioned by chitinous plates (sclerites) in order to allow movement. The dorsal and ventral sections, the tergum, and sternum respectively are heavily chitinized. The lateral section, joining the tergum and sternum (pleuron) is less heavily chitinized and thus more flexible.
- They have a body cavity called haemocoel, which contains haemolymph (blood and lymph) that bathes internal organs.
- Ecdysis or moulting is a phenomenon characteristic of all arthropods whereby the cuticle is shed at regular intervals in order to accommodate the growing tissues.

## **BIOLOGY OF ARTHROPODS**

Arthropods use the following systems for survival and perpetuation.

### **(a) Digestive system**

The alimentary canal comprises three distinct regions: the foregut or stomodaeum, the midgut or mesenteron, and the hindgut or proctodaeum:

Foregut - extends from the mouth to the proventriculus (muscular sac provided with stony cuticular plates or teeth serving for grinding and mixing of food.)

Mid gut - this is the stomach; physiologically the most active part of the alimentary canal, being concerned with digestive function.

Hind gut -consists of the ileum, colon, and rectum and finally opens to the anus.

### **(b) Circulatory system**

The circulatory system of all arthropods is of the “open” type, i.e. the fluid that circulates is not restricted to a network of conducting vessels as for example in vertebrates, but flows freely among the body organs. A consequence of the open system is that insects have only one extra cellular fluid, hemolymph, in contrast to vertebrates which have two such fluids, blood and lymph. Through this system hemolymph is pumped from the heart to the aorta then to the whole body. The circulatory system has no respiratory function.

### **(c) Respiratory system**

In the vast majority of insects, respiration is by means of internal air tubes known as trachea. These ramify through the organs of the body and its appendages, the finest branches being termed tracheolea. The air generally enters the trachea through paired, usually lateral openings termed spiracles, which are segmentally arranged along the thorax and abdomen. Respiratory spiracles also serve as exit of air conducting branches from the tracheal tube. Respiratory spiracles serve as exit of air conducting braches from the tracheal tube.

### **(d) Nervous system**

The many diverse activities of the various systems of an insect are coordinated by the nervous system. This system is composed of elongated cells, or neurons, which carry information in the form of electrical impulses from internal and external sensory cells to appropriate effectors. These consist of Nerve ganglia in the head, ventral part of the body, which later extends to body parts.

### **(e) Excretory System**

The function of the excretory system is to maintain hemostasis. i.e. maintaining the uniformity of the hemolymph. It accomplishes this by the elimination of metabolic wastes and excesses, particularly nitrogenous ones, and the regulation of salt and water. The malpighian tubules are the major organs involved in filtration of the hemolymph. These tubules lie freely in the body cavity (haemocoel) and open to the junction between the mid gut and the hindgut. After joining the digestive tract, waste fluids are excreted through the anus. The hindgut (specially the rectum) is involved in reabsorption of important ions and water.

### **(f) Reproductive System**

Arthropods have separate sexes. Male contains testes, vas deference, seminal vesicle and ejaculatory duct, which open by aedeagus (penis). The female contains two ovaries, oviduct, and uterus that opens to the vagina.

### **DEVELOPMENT OF ARTHROPODS**

The development of arthropods, which is called metamorphosis, is from egg to adult. This development could be:

Incomplete → development from the egg to nymph, which looks like the adult

OR

Complete → development, which extends from the egg to larva, pupa that later differentiate to the adult arthropod.

### **IMPORTANCE OF ARTHROPODS IN PARASITOLOGY**

Arthropods affect the health of man by being:

#### **(a) Direct agents for disease /discomfort.**

The following effects may be seen by the direct effect of arthropods.

- **Annoyance** – comes from disruptive activities of insects, such as flying around or landing on the head, and from feeding, possibly causing blood loss, though they don't remove sufficient blood to cause a medical problem in humans.
- **Entomophobia** – is an irrational fear of insects. One extreme form of entomophobia is delusory parasitosis, in which individuals become convinced that they are infested with insects when no actual infestation exists. This may cause undue alarm and anxiety, leading to unwarranted use of insecticides, and in severe cases, requiring professional treatment.
- **Envenomization** – is the introduction of a poison into the body of humans and animals. Arthropods may also inoculate poison to the host. E.g. Scorpion
- **Allergic reactions** – a hypersensitive response to insect proteins. All of the mechanisms associated with envenomization can also cause exposure to allergens. In fact, human deaths from bee and wasp stings usually are associated with a hypersensitive reaction rather than direct effect of a toxin.
- **Dermatosis and dermatitis** – dermatosis is a disease of the skin and dermatitis is an inflammation of the skin. Both dermatosis and dermatitis can be caused by arthropod activities. Many mite species, such as scabies mites produce acute skin irritations.

#### (b) Agents for disease transmission

Arthropods can carry disease causative agents in the following two ways.

- **Mechanical carrier**

Here they lodge the disease causative agent without altering its development or multiplication

e.g. house fly

- **Biological carrier**

When arthropods become biological carriers for transmission of disease, it means that certain stages in the life cycle of parasite takes place in the body of the insect.

e.g. Anopheles mosquitoes.

Biological carrier is any of the following types:

- **Propagative**- where there is multiplication of the parasite with no developmental change  
e.g. Yellow fever virus in Aedes mosquito.
- **Cyclopropagative** – in this type both multiplication and developmental change are going on.  
e.g. Plasmodium species in Anopheles mosquito
- **Cyclodevelopmental** – here there is developmental change of the parasite but no multiplication  
E.g. Wucherera bancrofti in Culex mosquito
- **Transovarian**- when the parasite passes to progeny arthropods through the ova  
E.g. Rickettsia typhi in ticks

If we are clear about the importance of arthropods as a source of human infection, it is important to accurately identify and classify them for crucial treatment, prevention, and control of infection

## **CLASSIFICATION OF ARTHROPODS**

There are three medically important classes of Arthropods:

1. **Class Insecta**- consists of mosquitoes, fleas, bugs, lice and flies, etc.
2. **Class Arachnida**- consists of ticks, mites and scorpion.
3. **Class Crustacea**- consists of cyclops.

A brief description of the general features and classification of each of the above classes of arthropods are presented below.

## **(1) Class Insecta**

The general feature of this class includes:

- Division of body into head, thorax and abdomen.
- Possess one pair of antenna on the head.
- 3 pairs of legs, carried by thorax.
- Wings may be present and could be one /two pairs.

This class is divided into four orders

**(a) Order Diptera:** this order consists of mosquitoes and flies. They have one pair of wing and development is by complete metamorphosis.

**(b) Order Siphonaptera:** consists of fleas. Arthropods in this order are wingless but have strong leg to help them jump. Their development is by complete metamorphosis.

**(c) Order Anoplura:** Is order consists of lice, which are wingless and with short legs. Their development is by incomplete metamorphosis

**(d) Order Hemiptera-** This order consists of bugs. Bugs have rudimentary wings and develop by incomplete metamorphosis.

## **2. Class Arachnida**

- Body divided into cephalothorax (head and thorax fused) and abdomen.
- Possess 4 pairs of legs.
- They are wingless
- No antennae
- undergo incomplete metamorphosis.

There are 3 orders in this class

### **(a) Order Acarina**

This consists of Ticks and mites. The adult tick or mite has 4 pairs of legs and the Nymph 3 pairs of legs.

### **(b) Order Araneida**

This consists of spiders.

### **(c) Order Scorpionida**

This order consists of scorpions.



### 3. Class Crustacia

The general feature of this class includes

- Body divided into cephalothorax and abdomen
- 4 pairs of legs
- 2 pairs of antenna
- Wingless
- Most are aquatic

This class includes the Cyclopes.

## MEDICAL CONDITIONS RELATED TO ARTHROPODS

### A. FLY RELATED CONDITIONS

**Myiasis** is invasion of tissue of humans and other vertebrate animals with dipterous fly larva, which for at least a period feed upon the living, necrotic or dead tissues of animals.

Houseflies can transmit a number of diseases to humans owing to their habits of visiting almost indiscriminately faeces and other unhygienic matter and people's food. Pathogens can be transmitted by three possible ways:

- By contaminated feet, body hairs and mouthparts of flies.
- Flies frequently vomit on food during feeding this can lead to infection.
- Probably the most important method of transmission is defecation, which often occurs on food.

Through the above mechanisms houseflies transmit a number of bacterial, viral, and protozoal diseases, e.g. sand flies transmit leishmaniasis, tsetse flies transmit trypanosomes.

## B. MOSQUITO RELATED CONDITIONS

Mosquitoes cause a number of diseases in humans; the different types of mosquitoes and the parasite they transmit are listed in the following table.

| Mosquitoes           | Parasite                                 |
|----------------------|------------------------------------------|
| Anopheles mosquitoes | Plasmodium sp.                           |
| Culex mosquito       | Wuchereria bancrofti                     |
| Aedes mosquito       | Wuchereria bancrofti, yellow fever virus |
| Mansonia             | Brugia malayi                            |

## C. FLEA RELATED CONDITIONS

Fleas can be ectoparasites, which may sometimes cause allergic dermatitis and are intermediate hosts for certain bacteria like yersinia pestis and Rickettsia typhi. In tropical America and Africa the most troublesome flea is Tunga penetrance, which is about 1 mm in length but after burrowing into the skin, it may swell to 1 cm and cause extreme irritation. Surgical removal is required. Sometimes the condition may also be complicated by secondary bacterial infection, which is usually the case in our country.

## D. LICE RELATED CONDITIONS

Lice are usually ectoparasites, and they can live in different part of the body.

For example

- Pediculus humanus capitis – head lice
- Pediculus humanus corporis – body lice
- Phitrius pubis – pubic /crab lice

Lice are also responsible for transmission of diseases such as relapsing fever and epidemic typhus, most commonly in the highlands of Ethiopia.

## **E. BUG RELATED CONDITIONS**

Other than being ectoparasites and a nuisance to humans, bugs like *Triatoma* (Kissing bug) are disease vector of *Trypanasoma cruzi*, which is seen in some countries of Latin America.

## **F. TICK RELATED CONDITIONS**

Ticks can cause mechanical injury to the skin. They may sometimes produce toxins, which affect release of acetylcholine at the neuromuscular junctions. This in turn produces a progressive ascending paralysis also called 'tick paralysis'.

Ticks also transmit diseases like francella and Rickettsial illnesses.

## **G. MITE RELATED CONDITIONS**

A mite called *Sarcoptes scabies* causes itchy, popular eruptions in the skin usually termed as scabies.

House dust mites either produce or concentrate potent allergens commonly found in non-ventilated houses.

## **VECTOR CONTROL MEASURES**

Many tools for arthropod control are found in today's arsenal with their own advantages and drawbacks. In this section some of the major approaches that have been used to control vectors and some that show promise for the future are presented:

### **(1) Mechanical methods**

E.g. Use of bed nets, wire mesh, etc.

### **(2) Ecological control**

Ecological control procedures involve the removal, destruction, modification, or isolation of materials that might favor the survival of an

insect pest by affording food or making a site suitable for breeding and/ or dormancy. e.g. draining marshy areas.

**(3) Chemical methods**

Deals with the use of natural or synthetic chemicals that directly cause the death, repulsion, or attraction of insects. E.g. use of DDT

**(4) Biological methods**

Refers to the regulation of vector population using predators, like certain species of fish, which feeds on larval stages of some arthropods, and microbial agents. There are several advantages in using biological control agents. Unlike pesticides, biological control agents are safe to use and do not pose any threat to the environment.

**(5) Genetic control**

Involves manipulation of the mechanisms of heredity. In some research centers sterilized male mosquitoes are used in order for them to compete with natural ones and thereby decreasing the new generation of mosquitoes.

## SUMMARY

Medical entomology is a science that deals with the study of arthropods, which play a significant role in the transmission of a number of diseases to humans. Arthropods affect the health of man by being either direct agents for disease or discomfort or agents for disease transmission. In mechanical carriers the pathogen does not multiply in the arthropod whereas the arthropod is an integral part of the life cycle of the pathogen in biological carriers.

The three medically important Classes of Arthropods are the Class Insecta, Crustacea, and Arachnida, which have their own distinguishing features. A clear understanding of the classification and characteristics of each of the classes is of paramount importance in devising ways of control the vectors.

## LEARNING ACTIVITY

Answer the following questions.

- 1) List the general features of all arthropods.
- 2) Discuss the circulatory system of arthropods.
- 3) List the direct effects of arthropods on humans.
- 4) Discuss the different types of biological carriers giving examples.
- 5) Explain the medical conditions related to arthropods.
- 6) List the different approaches in the control of vectors.

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## GLOSSARY

**Autoinfection:** self infection

**Chyluria:** lymphatic fluid

**Definitive host:** An animal that harbors a parasite where it reaches sexual maturity in or on it.

**Gynecophoral canal:** This is a canal in the male schistosome where the adult female worm is carried.

**Haematuria:** Presence of blood in the urine

**Hermaphrodite:** having both sexes in one

**Intermediate host:** Hosts normally infected with certain parasites, which are also capable of infecting humans.

**Molluscicide:** Chemical used to kill snails

**Proglottid:** a unit of tapeworm body