ملزمة 1
الطفيليات الطبية النظري
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Specific Learning Objectives

At the end successful student will be able to

1. Identify organisms which parasitizes man
2. Define common terms used in Medical Parasitology
3. List the various environmental, cultural and socioeconomic factors that affect the distribution of parasites
4. Explain effect caused by parasites
5. Describe the classification and characteristics of parasite groups
6. Explain mode of transmission, source of infection, and portal of entry of parasites

1.1 Definition of Terms Used in Parasitology

Parasitology:- is a science that deals with parasites.

Medical Parasitology:- Is the study of parasites that causes disease in man.

Parasite:- is an organism living temporarily or permanently in or on another organism (host) from which is physically or physiologically dependant upon other.

Nature of Parasites- A parasite could be unicellular, worm or an arthropode.

Features of Parasites

1. Smaller than their host,
2. Outnumber the host,

3. Short life span than their host, and
4. Have greater reproductive potential than their host.

Association of Organisms

When there is an association between two organisms their relation will be one of the following type:

1 Mutualism;- Mutual benefit is derived from the association.

2. Symbiosis:- Permanent association between two different organisms, so dependant on each other, that their life part is impossible.

3 Commensalism:- When the parasite benefited from the host while the host neither benefited nor harmed.

4. Parasitism:-One organism live at the expense of the other, The later usually suffers from the association.

Parasites can be Classified:-

I. According to their habitat:

1. *Ectoparasites:* parasites living on or affecting the skin surface of the host. E.g. lice, tick, etc.
2. *Endoparasites*: Parasites living within the body of the host. E.g. *Leishmania* species, *Ascaris lumbricoides, etc.*

II. According to their dependence on the host:

1. *Permanent (obligate) parasites:* The parasite depends completely upon its host for metabolites, shelter, and transportation. This parasite can not live outside its host. E.g. *Plasmodium* species, *Trichmonas vaginalis, etc.*

2. *Temporary (facultative) parasite*: The parasite is capable of independent existence in addition to parasitic life. E.g. *Strongyloids stercolaris, Naegleria fowleri, etc.*

III. According to their Pathogenicity:

1. *Pathogenic parasites:-* It causes disease in the host.
   E.g., *E. histolytica*
2. *Non-Pathogenic (commensal) parasite:-*The parasite derives food and protection from the host without causing harm to the host. E.g. *Entamoeba coli*
3. *Opportunistic parasites:-* Parasites which cause mild disease in immunologically healthy individuals, but they cause severe disease in immuno-deficient hosts.
   E.g. *Pneumocystis carnii, Toxcoplasma gondii, Isospora belli*

Host :- Hosts are organism which harbors the parasite.

Types of Hosts:-

1. ***Definitive host:-*** Depending on the parasitic species, *it is either* a host which harbors the adult stage of a parasite or most highly developed form of the parasite occurs; or sexually mature stages of a parasite and fertilization takes place in it, e.g., man is the definitive host of *Taenia saginata*. When the mature or most highly developed form is not obvious the definitive host is the mammalian host, e.g., human is the definitive host for trypanosomes that cause African trypanosomiasis.
2. ***Intermediate host***:- Is a host harboring sexually immature or larval stage of a parasite and in which no fertilization takes place in it.
   E.g. Cow is the intermediate host for *Taenia saginata*

Amplifier host- Intermediate hosts in which parasites under go multiplication.

3. ***Reservoir host***:- A wild or domestic animal which harbors a parasite and acts as sources of infection to humans.
4. ***Carrier host:-*** A host harboring and disseminating a parasite but exhibiting no clinical sign.
5. ***Accidental (Incidental) host:-*** Infection of a host other than the normal host species. A parasite may or may not continue full development in this host.

Vector:- Any arthropod or other living carrier which transports a pathogenicmicroorganisms from an infected to non-infected host.

A. ***Biological vectors***:-Those vectors that complete the life cycle a parasite
E.g. *Anopheles* (Vector of *Plasmodium*), *Phlebotomus* (Vector of *Leishmania*), *Glossina* (vector of *Trypanosoma*), *Simulium* (Vector of *Onchocerca*), etc.

B. ***Mechanical (Parathenic or transport) Vectors***: They are passive carriers of parasites, not essential in the life cycle. E.g. House fly and Chocroach as a mechanical vector for Amoebae, *Giardia*, etc.

Diagnostic Stage:- A developmental stage of a pathogenic organism that can be detected in stool, blood, urine, sputum, CSF or other human body secretions.

Infective Stage:- The stage of parasite at which it is capable of entering the host and continue development within the host.

Infection:- Invasion of the body by any pathogenic organism (except)arthropods and the reaction of the hosts tissue to the presence of the parasite or related toxins.

Infestation:- The establishment of arthropods upon or within a host.

Zoonosis:- Diseases of animals. Today this term is applied for those diseases that are transmittable to man.

Biological Incubation (Prepatent) Period:- It is time elapsing between initial infection with the parasite and demonstration of the parasites or their stages in excreta, blood, aspirate and other diagnostic material.

Clinical Incubation Period:- It is the interval between exposure and the earliest manifestation or infestation.

Autoinfection:- An infected individual acts as a source for hyperinfection to himself.

Superinfection (Hyperinfection):- When an individual harboring the parasite is reinfected by the same parasite.

Retroinfection:- A retrograde infection caused by the newly hatched larva of *E. vermicularis* from the perianal region to reach the colon, where the adolescent form of the parasite develop.

1.2 Sources of Exposure to Parasitic Infections

A. ***Contaminated soil***:- Soils polluted with human excreta is commonly responsible for exposure to infection with *Ascaris lumbricoides, S.stercolaris, Trichuris trichuria* and hookworms.

B. ***Contaminated water***:- Water maycontain

(a) Viable cysts of Amoeba, flagellates and *T. solium* eggs,

(b) Cercarial stages of human blood fluke,

(c) Cyclops containing larva of *Dracunculus medinesis*,

(d) Fresh water fishes which are sources for fish tape worm, and intestinal flukes infection

(e) Crab or cray fishes that are sources for lung fluke and

(f) Water plants which are sources for *Fasciolopsis buski*.

C. Insufficiently cooked meat of pork and beef which contains infective stage of the parasite.

E.g., Trichenilla spiralis, *Taenia* species.

D. Blood sucking arthropods:-These are responsible for transmission of: e.g.,

1. Malaria parasites by female anopheles mosquito
2. *Leishmania* by phlebotomus
3. *Trypanosoma* by tsetse fly
4. *Wuchreria* by *Culicine* mosquito

E. Animals (a domestic or wild animals harboring the parasite), e.g.

1. Dogs are direct sources for human infection with the hydatid cyst caused by *E. granulosus* and cutaneous larva migrans caused by *Toxocara canis,*
2. Herbivores animals commonly constitute the source for human infection with *Trychostrongylus* species.

F. Human beings:-Another person his clothing**,** bedding or the immediate environment that he contaminated are directly responsible for all or a considerable amount of infection with a pathogenic amoeba *E. histolytica, E. vermicularis, H. nana* **.**

G. S**exual intercourse** :- e.g., Trichomonas vaginalis

H. Autoinfection **:-** e.g., *S. stercoralis, E. vermicularis,* and *T. solium*

1.3 Mode of Transmission

1.3.1 Direct mode of Transmission:-

The parasite dose not require biological vectors and/or intermediate hosts and require only a single host to complete its life cycle. It may use mechanical vectors for transmission.

Direct Mode of Transmission can be classified as:

I. **Horizontal Direct Mode of Transmission:** Transmission is mainly effected through:- Feco-oral route:

Most intestinal parasites transmitted in this way.

- Sexual intercourse
- Blood transfusion
- Direct skin penetration (soil transmitted helminthes)

II ***Vertical Direct Mode of Transmission:***

Transmission of the parasite is from the mother to child through:

- Congenital / transplacental
- Transmammary (breast milk)

1.3.2 Indirect Mode of Transmission

The parasite has complex life cycle and requires biological vectors and/or one or more intermediate hosts for transmission.

1.4 Route of Transmission

The infective stage of the parasite may be transmitted in the following ways:

I. By ingesting infective stage of parasites:

1. In food, water or from hands that have been contaminated with faeces,
   E.g. *E. histolytica, E. vermicularis*
2. In raw or undercooked meat, e.g. *T. saginata, T. solium, T. spiralis*
3. In raw or undercooked fish, crab, or water vegetation e.g. intestinal flukes
4. Water containing Cyclopes e.g., *D. medinensis*

II. Penetration of Skin When in Contact with

1. Faecally polluted soil, e.g., *S.stercoralis*,Hook worms
2. Water containing infective stages of the parasite
   E.g., Cercaria of Schistosome species.

III. Through Insect Bite

e.g, filarial worms, *Trypanosoma* species, *Plasmodium* species , *Leishmania* species

IV. **Sexual Contact,** e.g., *Trichomonas vaginalis*

V. **Transmammary**, e.g., *S. stercoralis*

VI. **Inhalation of contaminated** air, e.g., E. vermicularis, *P. carnii*

VII. **Transplacental**, e.g., *T. gondii*

VIII. **Kissing**, e.g., *Trichomonas gingivalis, Trichomonas tenax*

1.5 Host Parasite Relationship

1.5.1 Effects of Parasites on their Hosts

A Parasite can affect the host in a number of ways such as:-

1. Consumption of the nutritive elements of the host
   E.g. Hookworm –sucks blood, *D. latum* selectively remove V B_{12}.
2. Obstruction of passages
   E.g., heavy infection with adult *Ascaris* may cause intestinal obstruction
3. Bleeding e.g. Schistosomes eggs
4. Destruction of tissues: e.g. Trophozoites of *E. histolytica* causes necrosis of liver, *Leishmania donovani* results marked destruction of marrow elements.
5. Compression of organs, e.g. Hydatid cysts in liver, brain cause pressure

6. Release of toxic substances, e.g., Rupture of *E. granulosus* cyst result anaphylactic shock
7. Opening path way to secondary infections e.g. Ulcer formed as a result of *D. medinensis* infection exposes to Bacterial, Viral infection
8. Allergy development, e.g., Bite of arthropode
9. Transmission of pathogens to man, e.g., lice transmitting *Rickettsia*
10. Predisposition to malignancy-e.g., Infection with bilharziasis predisposes to maliganacy
11. Chronic immune stimulation leading to unresponsiveness to infections.

1.5.2. Host Susceptibility Factors

Not all parasitic infection causes disease of clinical significance. Both host and parasitic factors are involved.

1.5.2.1 Host Factors

1. Genetic constitution
2. Age
3. Sex
4. Level of immunity: natural and acquired immunity.
5. Nutrition (malnutrition or under nutrition)
6. Intensity and frequency of infections
7. Presence of co-existing disease or conditions which reduces immune response. e.g. Pregnancy, HIV
8. Life style and occupation

1.5.2.2 Parasite factors

1. Strain of the parasite and adaptation to human host
2. Parasite load (number of parasite)
3. Site (s) occupied in the body

4. Metabolic process of the parasite, particularly the nature of any waste products or toxins produced by the parasite during its growth and reproduction.

1.5.3. Escape mechanism of parasite from the immune system

That parasitism is wide spread in almost all species of animals would imply that parasites have developed the capacity to escape or render ineffective the host internal defense mechanisms. Parasites can evade the host immune responses by variety mechanisms:

1. Site

Intracellular parasites as *T. cruzi*, *Leishmania* and the intracellular stage of *Plasmodia* are to some extent protected from the action of antibodies as are those forming cysts as *T. gondii* and larva of *T. solium*, *Echinococcus* and *Trichinella spiralis*.

Parasite living in macrophages as *Toxoplasma, T. cruzi* and *Leishmania* are able to avoid or inactivate the lysosomal enzymes, which are the cells weapons of offences against microbial organisms.

2. Avoidance of recognition :

This can be accomplished by:

2.1. Production of successive waves of progeny with different surface antigens
(i.e., variation of antigens) as in African trypanosomes.

2.2. Molecular mimicry: Certain parasites are recognized as self and consequently do not stimulate immunologic reactions in their host. Thus *Schistosome* worms are capable of masking their foreigners by acquiring a surface layer of host antigens which possibly protect them from antibody damage. These are called “eclipsed” antigens, since these antigens by resembling those of the host are not recognized as foreign and therefore are hidden

from the immune recognition. This phenomenon of antigen sharing between a parasite and a host is called Molecular mimicry.

3. *Suppression of immune response*:

Several parasitic species e.g, *Plasmodium*, *Toxoplasm*, *Trypanosoma* and *Trichinella* are able to suppress the ability of the host to respond immunologically. This sometimes, results in an increase in the severity of any viral or bacterial infection also present. Immuno-supression is due to production by the parasite of large quantities of soluble antigens which:

3.1. Combine with the antibody and preventing it from attaching to the parasite

3.2. Induce B or T-cell tolerance either by blocking antibody forming cells or by depleting the stock of mature antigen-specific lymphocites (clonal exhaustion).

3.3. Activating specific suppressor cells (T-cells or macrophages).

1.6 General Life Cycles of Parasites

1.6.1. Direct Life Cycle

A parasite that can complete its life cycle in a single host.
E.g., *S. stercoralis, Hook worms, G. lamblia, E,. histolytica*, etc.

1.6.2. Indirect Life Cycle:

When a parasite requires an intermediate host or vector to complete its development.
E.g., *Plasmodium* species*, Leishmania* species *, Taenia* species. etc.

1.7 Types of Specimen Used For Parasitological Examination

Stool :-.e. g., intestinal nematodes, cestodes, trematodes and protozoa.
Blood :- e.g., Haemoparasites
Urine :- e.g., *S. hematobium, T. vaginalis,*
Sputum :- e.g., *P. westermani.*
Skin :- *e.g., L. aethopica, O. volvulus, D. medinensis* and *E. vermiculari*
Cerebro-Spinal fluid:- e.g., *Trypanosoma rhodisense* and *Naegleria fowleri.*
Bone marrow:- e.g., *L. donovani* and *T.gondii*
Lymphgland aspirates:- e.g *Trypanosoma rhodisense, L..donovani and T. gondii*
Liver aspirate :e.g.,*E.histolytica*, *L..donovani and T.gondii*
Spleen aspirate:- e.g *L..donovani and T.gondii*
Muscle biopsy:- e.g., *T. spiralis*
Rectal scraping:- e.g., Schistosoma species
Duodenal aspirate:- e.g., *G. lamblia, F. hepatica* and *S. stercoralis*
Bronchial biopsy :- e.g., *P.carnii*
Perianal swab:- e.g**.,***E.vermicularis*

2.1 Class Rhizopoda (Amoebae) (Rhiza = root, pod= foot) Amoebae

Protozoan parasite belongs to the class Rizopodea characteristically move by pseudopodia which present the organ of locomotion. Seven ameba, are belonging to the order amoebida, are found in man. One of them is found in the oral cavity and the remaining six species are found in the large intestine, these include: *Entamoeba histolytica, E.dispar, E. Coli, Endolimax nana, Iodamoeba butschlii and Entamoeba polecki;* of these only one, i.e. *E.histolytica* is pathogenic to man, *E. nana* and other amoebae may coexist in the large gut as commensals. *E. gingivalis* is commonly found carious teeth disease gum and tonsils.

All human intestinal amoebae have: 1) a trophozoite from which is motile organism, feed, and reproduce, and, 2) a cystic form which is the non-feeding, non motile, dormant stage of protozoa. Among amoeba, *E gingivalis* has only a trophozoite from. The trophozoite stage consists of a shapeless mass of moving cytoplasm which is divided into granular endoplasm and clear ectoplasm. Digested food substances are stored as glycogen and chromatoid bodies. Amoeba reproduce asexually by simply dividing into two (binnary fission).

Before going into structural details here for each of them, their nuclear character for identification is considered:

E. histolytica, E. coli and the mouth ameba *E gingivalis*., have conspicuous peripheral chromatin, arranged on the inner surface of nuclear membrane; where as such chromatin material is lacking in the remaining other three amoebae. The nuclear membrane in *E. histolytica* is delicate and the chromatin substance on the inner side of the nuclear membrane appears as fine beads, which are uniform in size and evenly

arranged. The central chromatin substance is called the Karyosome. It is small and has a halo around it.

In *E. coli*, the nuclear membrane is thicker and the chromatin on the inner surface of the membrane is distributed irregularly in the form of coarse plaques. The karyosome in this case is coarser and eccentrically placed. This is called '*'Coli type"* of nucleus.

The special feature of E. nana is the large karyosome located in the center or slightly eccentric. In *I. butschlii*, the karyosome is large but it is surrounded by a ring of achromatic granules giving a halo effect around

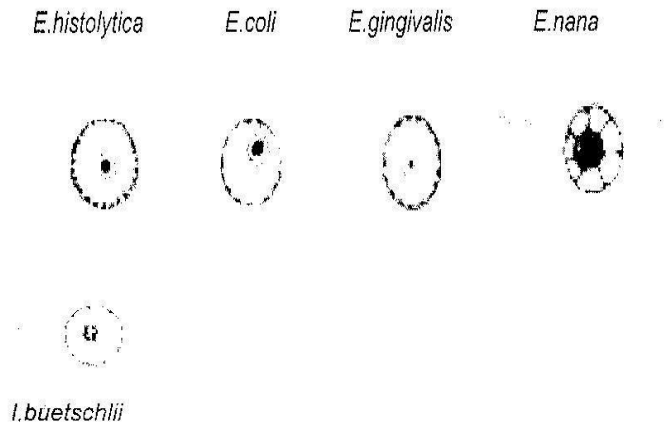


Figure 2.1.

Nuclei of the different species of amoebae (From Dey TK and DeyNC. Medical Parasitology, 9th ed. India, 1984.)

Entamoeba histolytica

Geographical Distribution:- Cosmopolitan distribution, mainly in the tropics and subtropics, and is mainly related to inadequate personal hygiene environmental sanitation, lack of safe water supply and poor socioeconomic situation.

Habitat:-***Trophozoite***:- Large intestine, liver abscesses and other extra-intestinal organs

Cyst:- found in the stools of chronic dysenteric patients and carriers.

Morphology

Trophozoite:

Size:- 12 to 35μm, Usually as long as 3 or 4 red blood cells.

Shape:- elongated form when actively motile and rounded form when at rest.

Motility:- Active, Progressive , directional amoeboid motility in fresh warm stool specimen.

Pseudopodia:- Finger like, broadly rounded end.

Cytoplasm:- Well differentiated into ectoplasm and endoplasm.

- May contain ingested host's red blood cells in dysenteric specimens

Nucleus:- Single nucleus, not visible in the motile form but in iodine stained smear clearly seen.

Cyst:- Size: 12-15μm (1½-2 red blood cells)

Shape: spherical

Nuclei: 1-4 nuclei

Nuclear membrane: thin, regular and circular lined with fine chromatin granules internally , and small, compact central karyosome.

Cytoplasm: Yellowish-gray and granular in iodine stained smear.

Stored food: Sausage shaped chromatoidal bars with blunt ends and glycogen mass in immature cysts with one or two nuclei.

Life cycle:

Entamoeba histolytica requires a single host to complete its life cycle. When mature tetra-nucleated cyst from contaminated food or drink or form hands contaminated with feces is ingested it excysts in the small intestine to produce metacystic trophozoite by a process of binary fission. The immature trophozoites migrate to the colon and grow to become mature trophozoite stage, multiply by binnary fission to invade the mucus membrane of the large intestine. Some times it can perforate the intestinal wall causing extra-intestinal amoebiasis. The trophozoite stage may pass with diarrhea or dysentery.

After a period of growth and multiplication, encystment occurs in the large intestine. In the process of cyst formation, the trophozoite discharge undigested food appears spherical in shape and condense to become pre-cyst. The pre-cyst secrets cyst wall to form a mono-nucleated cyst which is followed by a nuclear division to produce a bi-nucleated and then a tetra-nucleated mature cyst. Cyst and precyst will also pass in semi- formed or formed stool, where cyst is infective if it is ingested by any means of transmission.

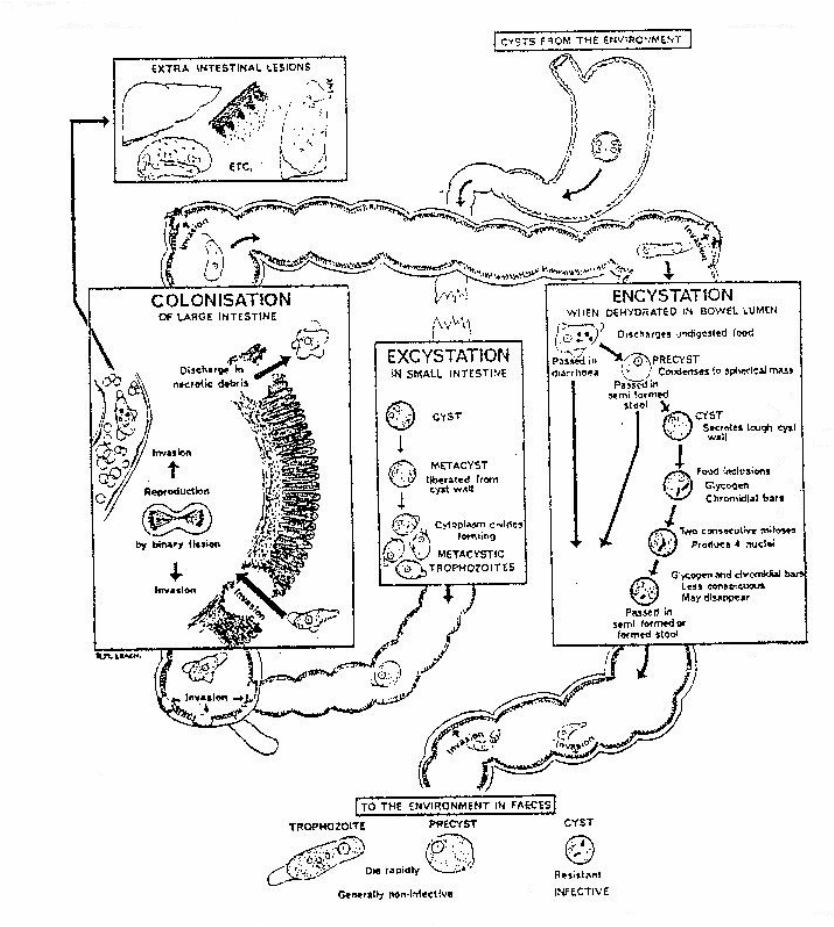


Figure 2.2.

Life cycle of *E.histolytica.* (From Jeffrey HC and Leach RM. Atlas of Medical Helminthology and Protozoology, 1975.)

Mode of Transmission

Infective stage: Tetra-nucleated mature cyst.

Man acquires infection of *E.histolytica* from:

- Ingestion of food or drink contaminated by infective cyst.

Clinical Features and Pathology

May be asymptomatic or exhibit amoebic dysentery or extra-intestinal amoebiasis in the liver, brain, spleen, lung, etc. Amoebic dysentery occurs when *E histolytica* trophozoites invade the wall of the large intestine and multiply in the submucosa, forming large flask shaped ulcers. The amoeaba ingest red cells from, damaged capillaries.

→

Amoebic Liver Abscess:

Occasionally amoeba is carried to the liver in the portal circulation and form abscesses, usually in the right lobe. There is pain & tenderness over the liver, wasting and fever with chills & night sweats. Patients with large or multiple abscess may become jaundiced and anemic.

Formerly a pathogenic invasive strain & a non pathogenic strain, of *E. histolytica* were thought to exist. The two 'strains' have now been recognized as separate species. *E. histolyrtica*is the invasive pathogenic and *E. dispar* has been designated the non- invasive non-pathogenic species. The two species are morphologically identical.

Prevention and Control

1. Cooking of food and vegetables
2. Hand washing after defecation and before eating
3. Safe water supply (treatment, boiling, filtration, etc.)
4. Control of mechanical vectors

5. Avoid use of night soil as a fertilizer proper sanitary disposal of faeces.
6. Treatment of infected individuals and health education.

Laboratory Diagnosis

Laboratory diagnosis of intestinal amoebiasis is based on:

1) Examination of a fresh diarrheic or dysenteric faecal specimen or rectal scraping for motile amoebae using saline, or
2) Examination of formed or semi-formed faeces for cyst stages. Such stool can be examined by direct saline and/or iodine smear, and Zinc sulphate floatation or centrifugal floatation method.
   - Charcot-Leyden crystals, representing the crystallized contents of granules from eosinophil leukocytes may also be found in a fecal smear.
   - Specimens must be examined without delay otherwiseidentification of the trophozoites becomes impossible because the amoeba lose their motility, extrude vacoules containing red cells, round up
   - With the recognition that *E.histolytica* is morphologically identical but genetically distinct form *E.dispar*, cysts, formerly reported as *E.histolytica* should be now reported as *E.historlytica* / E.dispar.

Differential Diagnosis of Amoebic Dysentery and Bacillary Dysentery

	Amoebic dysentery	**Bacillary dysentery**
Odor	Offensive	Odorless
Color of blood	Dark red	Bright red
Exudate	Few pus cells	Many pus cells
E.*histolytica*-	Present	Absent
Consistency	Mucus adherent to the	Not adherent

	container	
	Has charcoat -Leyden crystals	No crystal
Appearance -	Fecal matter with stratum of blood and mucus seen over the surface	Hardly any fecal matter consists of blood and mucus
Bacteria	Numerous, motile	Scanty, non motile

Entamoeba Hertmanni

Geographical Distribution: Cosmopolitan

Habitat: both trophozoite and cyst live in the small intestine

Morphology

Known as “small race” of *E.histolytica* because of similarity in their morphology. It feeds on bacteria not red blood cells.

Trophozoite

4-12μm, smaller than *E.histolytica*

Active but less vigorous directional motility by finger-like pseudopodia

Single nucleus with fine bead-like coarse chromatine granules on the thick nuclear membrane and has large centeral karyosome.

Cyst:- 5-10μm in size and spherical in shape.

1-4 nuclei; Minute rice grain shaped chromatoidal bars and glycogen mass in the immature cyst stage.

Life cycle

Similar to the life cycle of *E.histolytica.* It requires a single host and has five main developmental stages.

Tetranucleated cyst→Metacyst→Metacystic trophozoite→Trophozoite→Precyst→Uninucleated cyst

Mode of Transmission

Through contaminated food or drink, or from hands contaminated with faeces.

Pathology:- Harmless commensal

Laboratory Diagnosis:

- Finding the characteristic trophozoite and cyst stages in stool specimen. Differential *characters*:
- Cyst of *E.hertmanni* is similar to that of *E.histolytica* / *E. dispar* but the former is smaller in size.
- Cyst of *E.hertmanni* is also similar to that of *E.nana* but the later has 4-hole like nucleus and don't have chromatoid body

Entamoeba coli

***Geographical Distribution*:** Cosmopolitan.

Habitat

Both trophozoite and cysts in the large intestine of man

Morphology

Trophozoite:-

Size: 15-50μm(average 25μm), usually bigger than *E.histolytica*

Shape : Oval or elongated

Motility:- Sluggish, non -progressive and non-directional

- Short blunt pseudopodia

Cytoplasm: Ectoplasm and endoplasm not well differentiated.

Nucleus: Single nucleus, visible in the fresh state without staining.

Thick nuclear membrane lined with coarse chromatin granules and eccentric karyosome .

Inclusions: Bacteria, Yeasts, but never red blood cells.

Cyst:-

Size: 12-20μm a little larger than the cyst of *E.histolytica.*

Shape: rounded or slightly oval.

Nucleus: 1-8 nuclei; thick irregular nuclear membrane large, diffuse,often eccentric karyosome

Cytoplasm: bright pale yellow in iodine stained smear.

Life cycle

Similar to the life cycle of *E.histolytica.*

Octanucleated cyst Metacyst Metacystic Trophozoite Trophozoite precyst unincleated cyst binucleated cyst Tetranucleated cyst

Mode of transmission

Ingestion of contaminated food or drink by infective cyst.

Pathology:- Harmless commensal, may cause diarrhea in children.

Laboratory diagnosis:-

Finding the characteristic trophozoite and cyst stages in stool specimen. Can be differentiated from *E.histolytica by its larger size. The cyst of E.coli shows a greater variation in shape and size than those of E.histolytica.*

Entamoeba gingivalis

Geographical distribution: world wide distribution

Habitat: Oral cavity

Morphology:- Has trophozoite stage only, no cyst stage

Trophozoite:-

Size:-10-20 nm

Motility: sluggish

Cytoplasm: well differentiate into ectoplasm and endoplasm

Pseudopodia:- multiple

Nucleus:- single, delicate nuclear membrane lined with fine chromatin granules and Small central karyosome.

Life cycle:-It is reproduced by binary fission and transmitted from one person to another through kissing , droplets spray from the mouth,contaminated spoons or cups.

Pathology: non pathogenic commensal amoebae

***Laboratory Diagnosis*:-**Finding the characteristic trophozoite stage from swab of the oral cavity. It is the only species to ingest host's leukocytes and has numerous food vacuoles.It should be differentiated from *Trichomonas tenax which belong to flagellates and found in oral cavity.*

Endolimax nana

Geographical distribution: cosmopolitan. ***Habitat:***
Trophozoite and cyst in the large intestine.

Morphology:-

Trophozoite

Size: 6-15μm (average 10μm)
Motility: multiple small rounded blunt pseudopodia moving slowly in all direction.
Cytoplasm: very granular with small vacuoles
Inclusion: Bacteria.
Nucleus: single large irregularly shaped eccentric karyosome and thick nuclear membrane with out lining of chromatin granules.

Cyst:-

Size: 8-10μm
Shape: oval
Nucleus: 1-4 nuclei large, irregular karyosome

Cytoplasm*:* Deep yellowish color in iodine smear.

***Life Cycle*:-**Trphozoite stage reproduces by binary fission and man acquires infection from contaminated food or drink with

mature cyst stage.

Laboratory Diagnosis:-Finding of the cyst and trophozoite stages in fecal smear.

Iodamoeba butschili

Geographical distribution: Cosmopolitan.

Habitat: both trophozoite and cyst in the large intestine.

Morphology:-

Trophozoite:-

Size: 10-25μm

Shape: compact, leaf -like

Motility: sluggish by clear, rounded, finger like pseudopodia.

Nucleus: single, no chromatin granules on the nuclear membrane and has large karyosome surrounded by achromatic granules.

Inclusion: bacteria; large food vacuole packed with glycogen mass that stains deep brown with iodine solution.

Cyst:-

Size: 8-10 μm

Shape: rounded, oval or irregular

Nucleus: single, very large oval karyosome surrounded with cluster of granules.

Vacuole: very large glycogen vacuole often taking up half part of the cyst and stains brownish red by iodine solution hence the name was given as Iodamoeba.

Life Cycle:-The trophozoite reproduces in the large intestine by binary fission. The infective stage is the mature cyst stage and man acquires infection from contaminated food or drink.

Pathology: It is non-pathogenic.

Laboratory diagnosis:-Finding the characteristic trophozoite and cyst stages in the direct fecal smear examination or using concentration technique.

Entamoeba polecki

Most commonly infects pigs and monkeys; but may cause mild diarrhea in human hhumaman

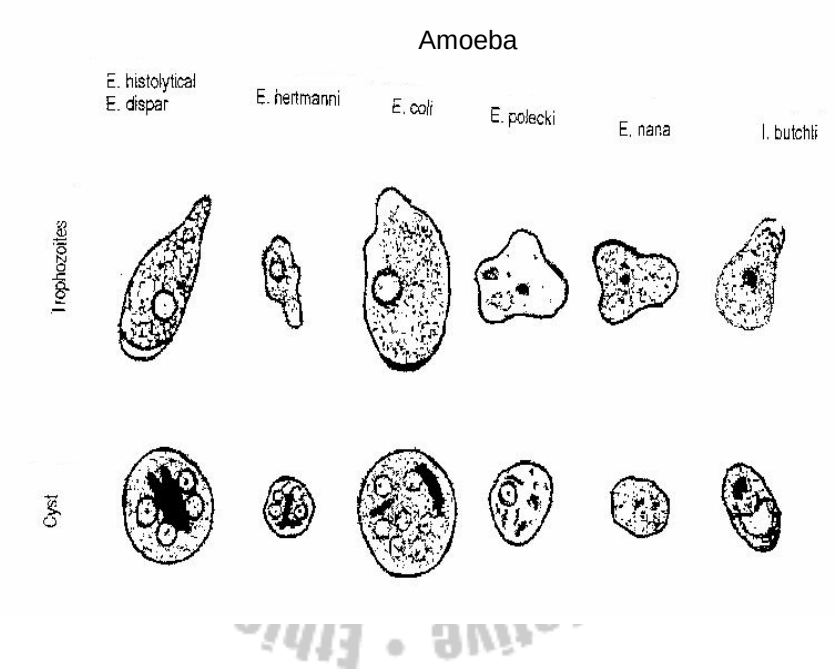


Figure 2.3.

Ameba found in stool specimens of humans. (From SmithJW,et al. Diagnostic Medical Parasitology : intestinal protozoa. American Society of Clinical Pathologist, 1976.)

2.1.2 Free living Pathogenic Amoebae

The free living amoeba constitute a large group, inhabiting fresh, brackish and salt water, moist soil and decaying vegetation. Some are coprozoic. For convenience this large and diverse group is separated into two groups on the bases of their ability to undergo transformation from an amoeba to a flagellate stage. *Naegleria* belongs to the family Vahkampfiidea, members of which are characterized as amoebaflagellates able to assume a temporary flagellate form while completely devoid of flagella in their amoeboid stage. *Acanthamoeba*, belonging to the family Acanthamoebidea, never produce flagella. Most cases of primary amebic meningoencephalitis can be ascribed to infection with *Naegleria*; less commonly Acanthamoeba is involved.

Naegleria fowleri

Geographical Distribution:- It is free living pathogenic amoebae with worldwide distribution.

This genus of amoeboflagellates has an amoeboid phase, which alternates with one possessing two flagella. The forms found in the tissues are ameboid, and in the tissue they are distinguished with difficulty from acanthamoeba. Most case have occurred during the hot summer months in young persons who within the preceding week swam or dived in fresh or brackhish water, lakes , hot springs and swimming pools have been apparent sources of the infection.

That the source of infection is not always aquatic is illustrated by a case report from Nigeria where the organisms were apparently inhaled during a dust storm by an 8-month old infant, from whose nasal mucosa and spinal fluid the organism were recovered prior to death. Another interesting report from Nigeria concerns a Muslim farmer thought to have

become infected during the ritual washing before prayer, which involved sniffing water up his nose.

Habitat: Nasal cavity, Central nervous system,

Morphology

Trophozoites

Size: 10-15μm

Cytoplasm - Well differentiated into ectoplasm and endoplasm

Motility - actively motile with broad pseudopodia elongated

Nucleus - Single with central karyosome

Inclusion -does not ingest host RBCs

Cyst - One nucleus and thin cystcyst wall which has no pore.

Flagellate

Shape - Oval

Nuclues - similar as above

Flagella - two flagella equal in length and longer than broad

Pathology

Causes primary or acute meningoencephalitis. Major symptoms include fever, stiff neck (meningitis), frontal headache, altered test (food or drink), vomiting, white blood cell count about 24,000 with many neutrophils.

Prevention and Control*:-*Infection can be prevented by avoiding contact, swimming and snuffing in waters of ponds, lakes, treatment of water and proper sewage disposal, treatment of infected individuals and health education.

Laboratory Diagnosis*:-*Only amoeboid trophozoite stage is found in man. Finding the amoeboid trophozoite stage in cerebrospinal fluid (CSF). It can be found in unstained or Giemsa stained CSF smear. The

CSF appears to be claudy, purulent and may contain eosinophils and red blood cells.

Acanthamoeba Species

Similar with that of N.fowleri except the following differences:

1. *Trophozoiutes are larger 15-25*μm
2. *Pseudopodea arefilamentous (spiky pseudopodia or acathopodia)*
3. *It does notforma flagellate stage inwater*
4. *Cysts are thickwalled with many pores*
5. *It may encysting the tissue*
6. *Pathology:-* Causes secondary or chronic meningoencephalitis.. Symptoms include fever, headache, rhinitis, meningoencephalitis, conjunctivitis, corneal ulceration, keratitis and loss of vision
7. Notkilledbydessication

Review Questions

1. What are the differences among cysts and trophozote of *E.hisolytica, E.hertmani* and E.coli?
2. Which amoeba do not have cyst stage in its life cycle?
3. Explain the general prevention and control methods of amoeba.
4. List pathogenic amoeba.
5. Discuss the general mode of transmission and laboratory diagnosis of amoeba.
6. How do you differentiate amoebic dysentery from bacilliary?
7. What is the clinical significance of studying *Naegleria* and *Acanthamoeba* species.

2.2 Class: Zoomastigophorea

Mastigophora: Flagellates

Flagellates infecting man are divided into two groups.

1. The oro-intestinal and urogenital flagellates and
2. The Hemo-somatic flagellates.

The oro-intestinal and urogenital flagellates are found in the intestine, oral cavity and genital tract. Many of them are not pathogenic. They are classified in to 2 orders, namely; Protomonadida and Diplonadida, The former is characterized by one nucleus and flagella at the anterior end; where as the latter has a pair of nuclei and flagella, which are symmetrically distributed at the interior end.

The hemoflagellates are present in the blood and invade various tissues of the body; remain either in the intercellular fluid, bathing the cells; as in trypanosoma, or are engulfed by the Red cells and leucocytes as in *Leshmania*. Of the six genera the parasite athogenic to man belong to two genera under the family Trypansomatidae, these are *Trypanosoma* and *Leishmania*.

2.2.1 The Oro-intestinal and Urogenital Flagellates

General Characteristics

1. Uses flagellum as locomotory organell
2. Reproduce by simple binary fission
3. Complete their life cycles in a single host and a second host whom they infect is necessary for the continuation of the species.
4. Most are commensal forms except *G.lamblia, T.vaginalis* and *D.fragilis*
5. The infective stage may be either the trophozoite or the cyst stage

6. Except the species of *Trichomonas and Dienatamoeba fragilis*, all have both cyst and trophozoite stages.

Dientamoeba fragilis

Dientamoeba fragilis originally classified as an amoeba, is now considered an amoeba like flagellate more closely related to the genus *Trichomonas*. Electron microscopic studies have revealed that the internal structures are typical offlagellate.

Geographical Distribution: World wide

Habitat: In the large intestine.

Morphology: Has trophozoite stage only, No cyst stage.

Trophozoite:-

Size: 6-15μm

Motility: Either non-motile (most often), or very actively motile in very fresh fluid stools with fan-like multiple pseudopodia. It becomes non-motile under the cover slip or disintegrates immediately.

Cytoplasm: clear ectoplasm.

Nucleus: Usually one or two nuclei but 3or 4 nuclei may be found rarely. Karyosomes split into 4-6 granules.

Inclusion bodies: Bacteria

Life cycle:- The mode of transmission is uncertain but most likely is feco-oral nature. It is postulated that the delicate trophozoite is transported from person to person inside the protective shell of helminth ova such as *Entrobius vermicularis*. It reproduces asexually by binary fission. It is considered to be harmless commensal

Laboratory Diagnosis:-The trophozoite stage is highly fragile and disintegrates explosively in water immediately. Hence it needs

immediate examination of fresh stool specimen to find the trophozoite stage.

Chilomastix mesnili

Geographical Distribution: cosmopolitan but mostly prevalent in warm climates.

Habitat*:* Trophozoite and cyst live in the colon and caecum of the large intestine.

Morphology

Trophozoite:- Size: 6-20 by 3-10μm

Shape: Triangular and tapered at one end

Motility: spiral in one definite direction.

Cytoplasm:

- Spiral groove that makes asymmetrical flagellate
- cytostome (mouth-like cleft) at the rounded end.

Nucleus: one nucleus, easily visible in unstained preparation

Flagella: Six flagella. Three anterior free flagella, one delicate flagellum lying in the cytostome and two flagella on the lateral margin of the cytostome

Cyst*:-* Size: 6-8 by 4-6μm

Shape: pear or lemon shaped

Cystostome and remains of locomotory organelles can be seen.

Nucleus: single; Thick nuclear membrane with small central karyosome.

Life Cycle

Cyst→Excystation→Trophozoite→Binary fission→encystation→Cyst in the faeces

Trophozoite stage reproduces by binary fission. The infective stage is the cyst from contaminated food or drink. Excystation occurs in the large intestine and trophozoite multiplies by binary fission.

Pathology*:* It is commensal

Laboratory Diagnosis:- Finding the trophozoite and cyst stages in stool specimen. The trophozoite stage is very similar to *Giardia lamblia ano Trichomonas hominis*; and needs careful identification.

Giardia lamblia

Also called *Giardia intestinalis* and *G.duodenale*

Geographical Distribution:- Cosmopolitan distribution in warm climate and is more prevalent in children than in adults. It is the most commonly diagnosed flagellate of the human intestinal tract. High prevalence occurs in young, malnourished children in large families, orphan asylums, and elementary schools.

Habitat: Upper parts of the small intestine mainly in the duodenum and jejunum.

Morphology:

Trophozoite*:-*Size: 10-21 by 5-15μm

Shape: pyriform (pear-shaped), i.e. rounded anteriorly and pointed posteriorly.

Motility: Progressive, rapid, tumbling and spinning often linked to a “falling leaf” type of motility in fresh liquid stools.

Bilaterally symmetrical

Covex dorsal surface and a flattened ventral side

Contents:

- Anteriorly there are two sucking discs each contains a nucleus, 4 pairs (8) flagella, Parabasal body and axonemes

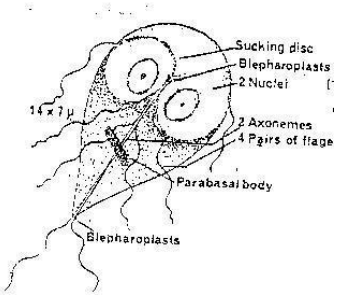


Figure 2.5. Trophozoite of G.lamblia. (From Jeffrey HC and Leach RM. Atlas of Medical Helminthology and Protozoology, 1975.)

Cyst :- Size: 8-12µm, oval shape with thick cyst wall.
Finely granular cytoplasm clearly separated from cyst wall.
2-4 oval nuclei at one pole, each with small, central karyosome.
Cytoplasm: clear when unstained; yellowish green or bluish in iodine solution.
Fibril: thread-like remains of flagella; axonemes and parabasal bodies folded as S-shaped placed length wise in the center of the cyst.

Life Cycle

Requires a single host to complete its cycle and reproduces by a simple longitudinal binary fission

Cyst ingested→excystation→Trophozoite→binary fission→Encystation→cyst in faeces

Infection occurs by ingestion of mature tetranucleated cyst with contaminated food, drink, finger, etc. Following ingestion, the cyst excyst in the upper part of the small intestin to form flagellates. They become attached to the intestinal wall by a sucking disc and absorb nourishment through their body surface. They multiply by longitudinal binar fission and some of them are carried down the intestinal tract to undergo encystation. The trophozoites and infective cysts are excreted in the faeces.

Clinical Feature and Pathology:-Major symptoms includes duodenitis, excess secretion of mucus or malabsorption of fat (steatorrhoea), sugar and vitamins, dehydration, diarrhoea, weight loss, poor appetite, vomiting, lethargy bile passage obstruction

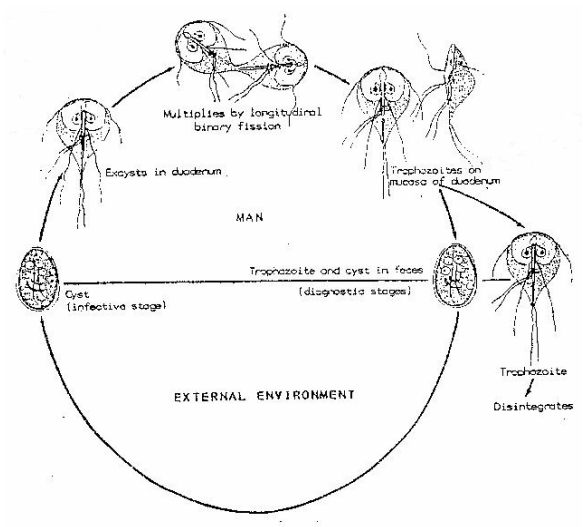


Figure 2.6. Life cycle of Giardia lamblia. **(**From Nasir NT. Review of Human Parasitology,2nd ed. The Scientific Book Center, Cairo)

Prevention and Control:

1. Improving personal, family and group sanitation and hygiene.
2. Avoid contamination of food, drink and hands with the faeces.
3. Safe water supply and latrine construction.
5. Treatment of infected individuals and health education.

***Laboratory Diagnosis*:-**Finding the trophozoite and cyst stages in stool specimen. The stool is usually offensive, bulky, pale, mucoid (fatty), diarrheic (watery) but there is no blood in the stool. Several specimens collected at different time need to be examined because trophozoites and cysts are excreted irregularly.

Intestinal and non-pathogenic flagellate that require differentiation from *G.lamblia* include: *C.mesnili* and *Pentatrichomonas hominis (*formerly *T.hominis).*

Trophozoites of the above mentioned flagellates can be easily differentiated from *G.lamblia* by their shape and movement (in fresh sample) and bcause they have only one nucleus (and fewer flagella). The only other trophozote that has two nuclei is *D.fragilis* but this organism has no flagella or median bodies and look likes a small amoeba.

Cyst of intestinal flagellates can be easily differentiated from those of G.lamblia because they are smaller and do not have the same characterstic appearance of G. lamblia (do not contain remains of flagella). *C.mensili* cysts are lemon shape and *D. fagilis* does not has cyst stage.

Relevance to Ethiopia

Infection by *Giardia lamblia* has a cosmopolitan distribution both in developed and developing nations. Infection rates ranging from 1% to 50% or so have been reported from various parts of the world. In African,

Asian and Latin American countries, about 200 million cases of *Giardia lamblia* infections have been estimated to occur annually. The infection may be endemic as in the tropics where it is a familial infection passed around by faecal-oral route, sporadic as in travelers, or epidemic as waterborne or institutional outbreaks (Helmut K and Zein AZ, 1993)

Giardiasis is wide spread in Ethiopia. A countrywide survey of giardiasis, using formal-ether concentration method, among school children and residents showed overall prevalence rates of 8.9% and 3.1% respectively. The corresponding rate for non-school children (5-19 years of age), however, was 4.4% showing that the School children are more significantly infected than their school children counter parts. *Giardia lamblia* infection was generally found to be more prevalent in children than in adult. Among children of school population those in their first decade of life were more affected (Hailu and Berihanu, 1995).

Although these and other prevalence data are not strictly comparable due to differences in sample selection and diagnostic methods used in different institutions, they indicate that while urbanization resulted in the reduction of the prevalence of giardiasis, it remains common infections in urban population. Infection rates reported here can not represent the actual prevalence rate giardiasis in Ethiopia as stool examination alone is not reliable to rule out infection of *Giardia* the cysts of which are excreted episodically.

Trichomonas hominis

Geographical Distribution*:* Next to *Giardia lamblia*, it is probably the most common and most cosmopolitan of the intestinal flagellates of man.

Habitat: Large intestine.

Morphology: has trophozoite stage only.

Trophozoite

Size: 10-15μm, pyriform (oval with two pointed poles) in shape

Motility: whirls and turns (jerky) in all directions, seeming to vibrate. Most resistant flagellate that remains motile even in old stool specimens.

Undulating membrane and costa reach 2/3 or full length of the body Nucleus: Single nucleus with central karyosome.

Flagellum: 3-5, usually 4 anterior free flagella and another flagellum on the margin of undulating membrane with a free trailing posterior end. Conspicuous cytostome opposite to the undulating membrane, has semi-rigid axostyle and parabasal body

Life Cycle:-The trophozoite stage reproduces by binary fission and requires direct host to host transmission through contaminated food and/or drink. It has high prevalence in children and more common in warm climates.

Pathology: It is non-pathogenic but may cause diarrhoea and infection can be prevented by personal hygiene and sanitation.

Laboratory Diagnosis: Finding the trophozoite stage in fresh stool specimen.

Trichomonas vaginalis

Geographical Distribution:-World wide distribution and mainly common in the temperate region.

Habitat:-In the genital tract of male and commonly in female, especially the vagina, cervix, urinary bladder, prostate and seminal vesicles.

***Morphology*:** Has trophozoite stage only.

Trophozoite

Size 15-25 by 5-12μm, is the largest *Trichomonas*.

Shape: pyriform

Moility: Jerky (on-spot), non-directional motility in fresh specimen.

Short undulating membrane: extending along one third of the body.

Nucleus: Single with uniformly distributed chromatin granules

Flagella: 4 anterior free flagella and one on the margin of the undulating membrane Axostyle may be split into several fibrils anteriorly.

Less conspicuous cytostome and has marked parabasal body.

Life Cycle:-The trophozoite stage reproduces by longitudinal binary fission and mode of transmission is usually via sexual intercourse but also by communal bathing, sharing of washclothes, toilet equipment seats and mother to daughter during birth.

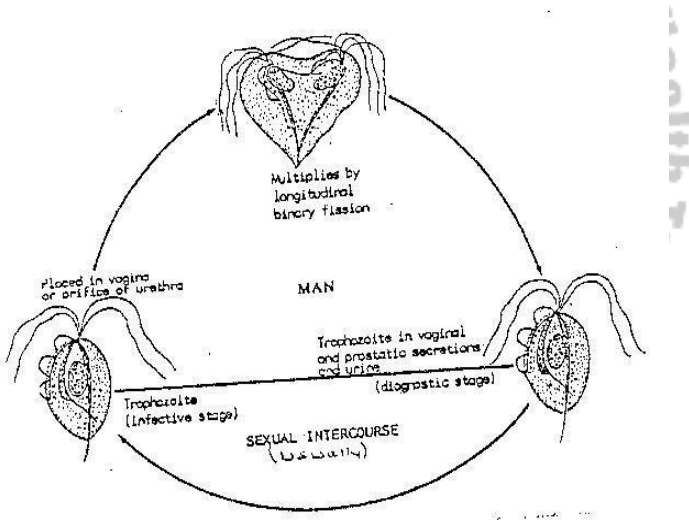


Figure 2.7. Life cycle of T.vaginalis **(**From Nasir NT. Review of Human Parasitology,2nd ed. Cairo: The Scientific Book Center.)

Pathology: Causes trichomoniasis. Major symptoms are Vaginitis, urethritis, prostatitis, chaffing of vulva, cervical erosion, burning sensation, yellowish prulent discharge, reversiable sterility in male.

Prevention and Control

1. Personal hygiene and sanitation
2. Simultaneous treatment of both partners.

Laboratory Diagnosis:-Finding the trophozoites in unstained or stained preparation of vaginal or urethral discharges(pap), urine sediment, vaginal swab, prostate secretions.

Relevance to Ethiopia

Trichomonas vaginalis is fairly common in Ethiopia as the level of hygiene is very low. Forty of 216(15%) prostitute in A.A tested were positive in 1995. (Kloos &Zein Ahmed, 1995). Another study done in A.A revealed 20% of women attending antenatal clinic harbour this parasite (Duncan ME, et al. Ethiop J Health Dev, 1995). Recent Unpublished study conducted inJimmatown detect a prevalence of 12.3% inwoman attending gynecology OPD (Tariku L, 2002).

Trichomonas tenax

Geographical Distribution

World wide.distribution with high incidence in warm climates.

Habitat: oral cavity.

Morphology: Has trophozoite stage only.

Trophozoite:-Size: 5-12μm

Shape**:** pyriform

Motility: active jerky motility

Undulating membrane: reaches two third of the body length.

Nucleus: Single

***Flagella*:** four anterior free flagella and one flagellum on the undulating membrane.

Thick axostyle extending a considerable distance behind the body. Has parabasal body

Life Cycle:- The trophozoite stage reproduces by binary fission and transmission is direct from mouth to mouth through kissing or communal use of contaminated food and drinking utensils.

Pathology:- It is non-pathogenic

Laboratory Diagnosis:- Finding the trophozoites in unstained or stained smear in swab taken from the oral cavity.

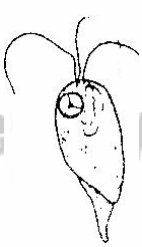
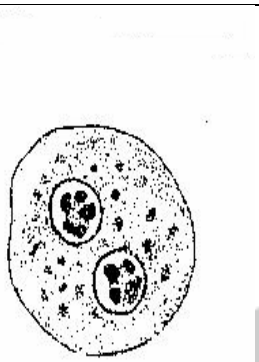
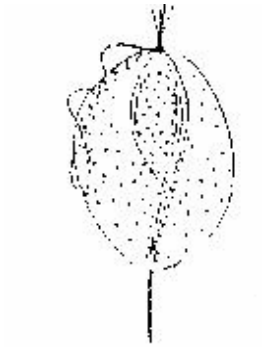
	T. hominis	*C.mesnili*	*G. lamblia*
t r o p h o z o l t e s			
Cyst	***No cyst***		

Figure 2.8. Flagellates.(From Smith JW, et al.Diagnostic Medical Parasitology:Intestinal Protozoa, Chicago.American society of Clinical Pathologist, 1976.

	D.fragilis	*T.vaginalis*
t r o p h o z o l t e s		
c y s t	***No cyst***	***No cyst***

Continuation of

Figure 2.8. Flagellates.(From Smith JW, et al.Diagnostic Medical Parasitology:Intestinal Protozoa, Chicago.American society of Clinical Pathologist, 1976.

Prevention and Control

1. Avoid eating raw or undercooked fish
2. Proper waste disposal of faeces in latrine
3. Avoid use of human faces as a fertilizer
4. Destroy snails and their habitat
5. Inspection of fish for metacercariae
6. Treating infected individuals and giving health education

Laboratory Diagnosis

Finding the characteristics eggs in the faece