Chapter 11

Medical Parasitology

Dr. Mohammed Hussien Taleb

Parasitology

- **Classification: The human parasites are classified within:**

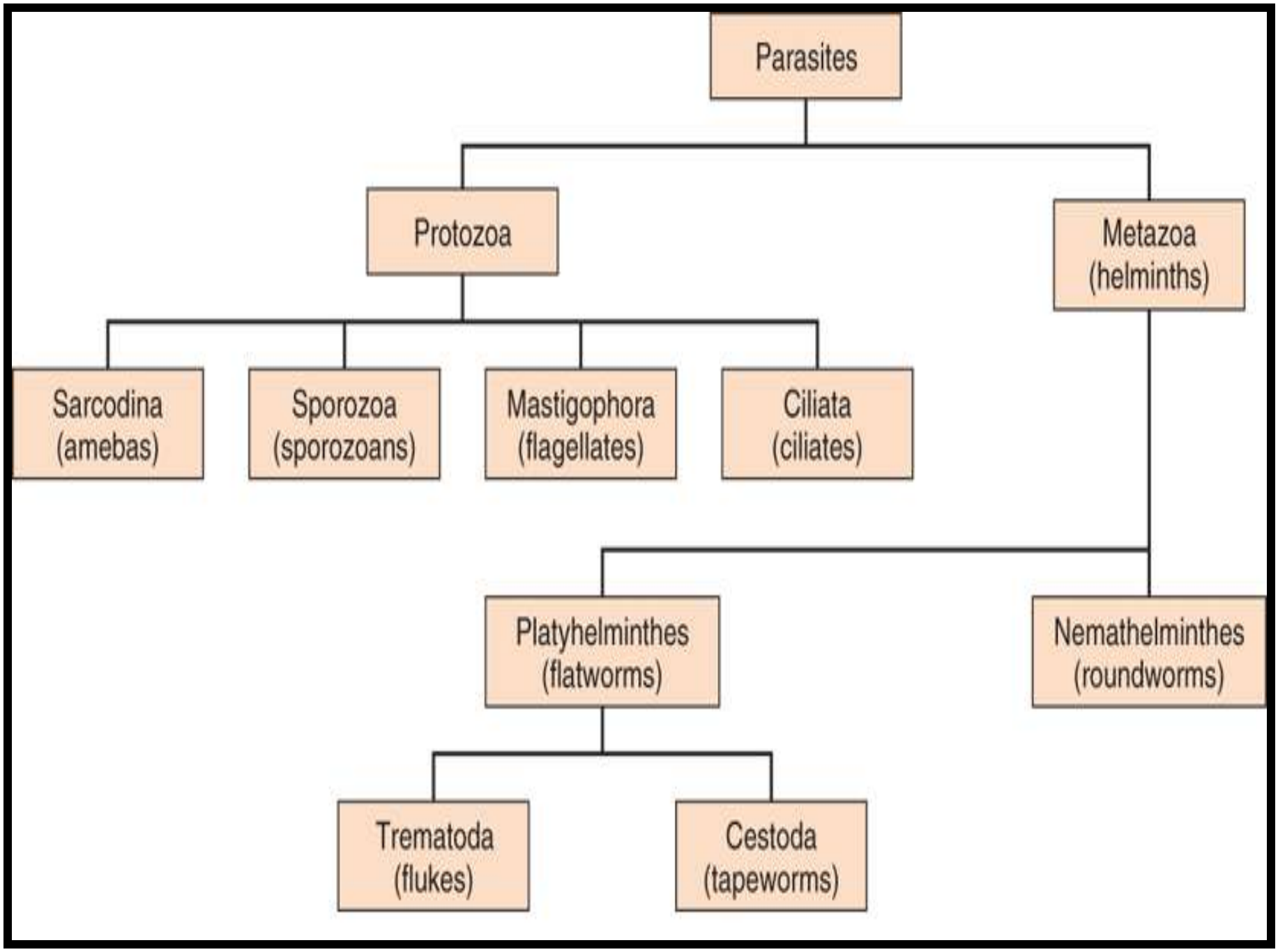
1. ***Protozoology*** = study of **protozoa** (primitive single cells).

2. ***Helminthology*** = study of **helminthes** (worms) including multicellular parasites with organs (also known as metazoa).

3. ***Entomology*** = study of **arthropods** (including insects).

Classification of Parasites

Protozoa	helminths
Unicellular Single cell for all function	Mulicellular Specialized cells
Amoebae: move by psudobodia. Flagellates: move by flagella. Ciliates : move by cilia Apicomplexa (sporozoa) Tissue parasites	Round worms (Nematodes) cylindrical, unsegmented Flat worms 1-Trematodes: leaf-like, unsegmented. 2-Cestodes: tape-like, segmented


Parasites
Protozoa
Metazoa (helminths)
Sarcodina (amebas)
Sporozoa (sporozoans)
Mastigophora (flagellates)
Ciliata (ciliates)
Platyhelminthes (flatworms)
Nemathelminthes (roundworms)
Trematoda (flukes)
Cestoda (tapeworms)

Protozoa

A- Flagellata

1-Giardia intestinalis

- Causative agent of giardiosis, lambliosis
- Giardia intestinalis ,a parasite of worldwide distribution.
- It is a parasite of the small intestine of humans that can cause enteritis.
- Infection occurs by peroral ingestion of Giardia cysts.
- Various species of mammalian animals are reservoir hosts.

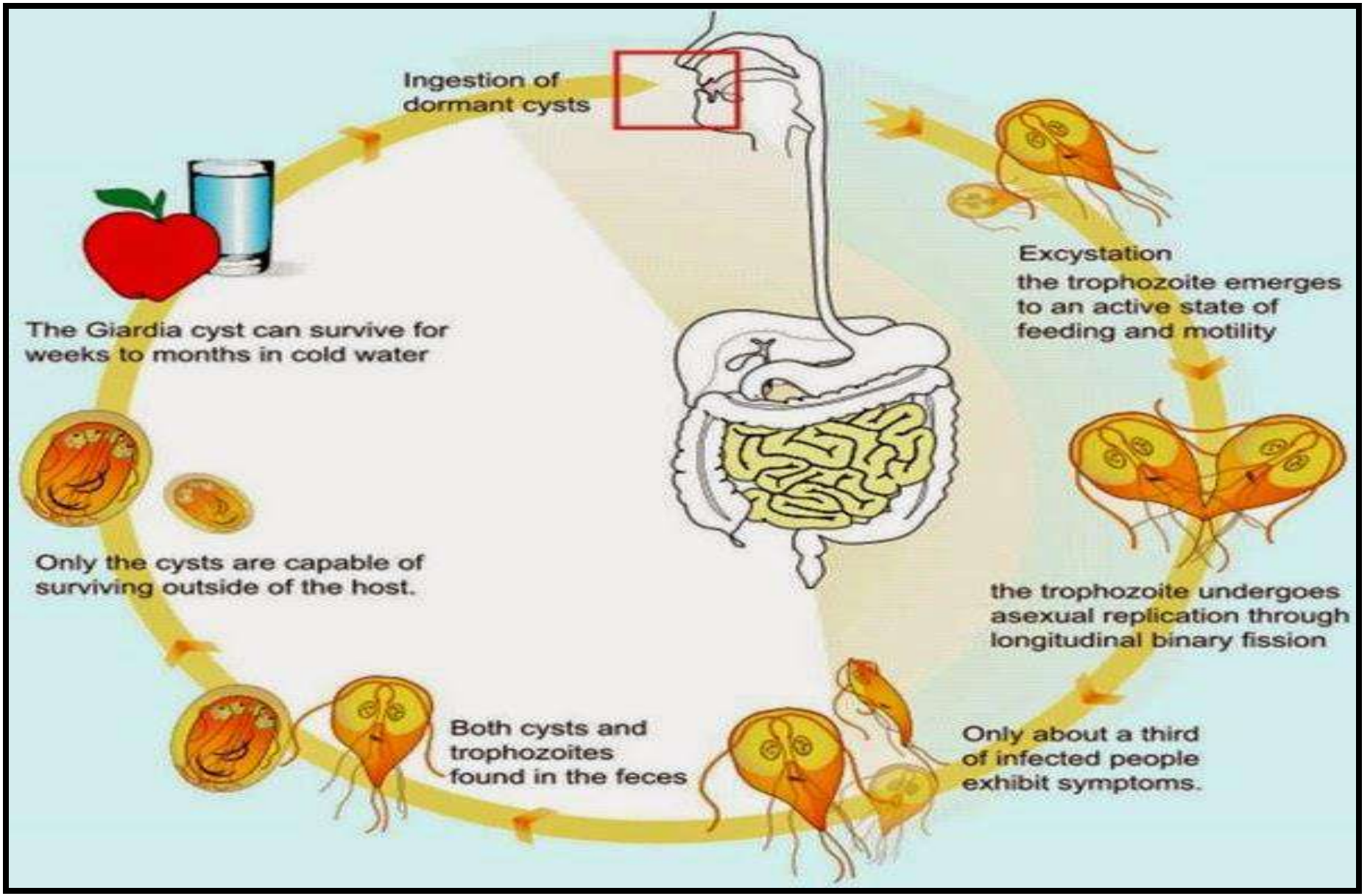
Parasite and life cycle

- Giardia exists in two morphological forms:
- A motile vegetative stage (the trophozoite)and a cyst stage.
- The trophozoites live on the small intestine mucosa (less frequently on the gallbladder mucosa as well).
- Reproduction is by means of longitudinal binary fission of the trophozoites, which are able to produce variant specific surface proteins.

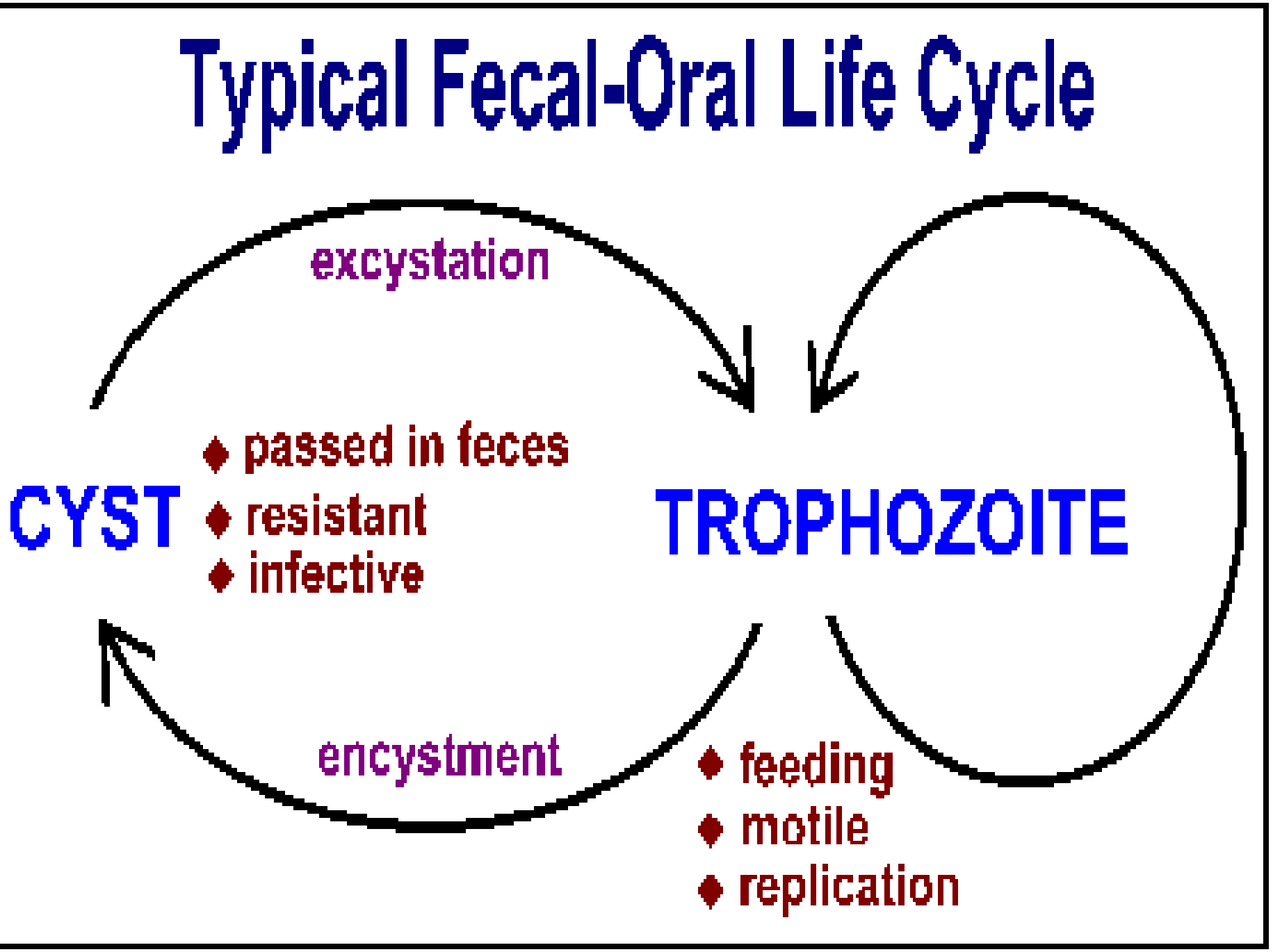
•

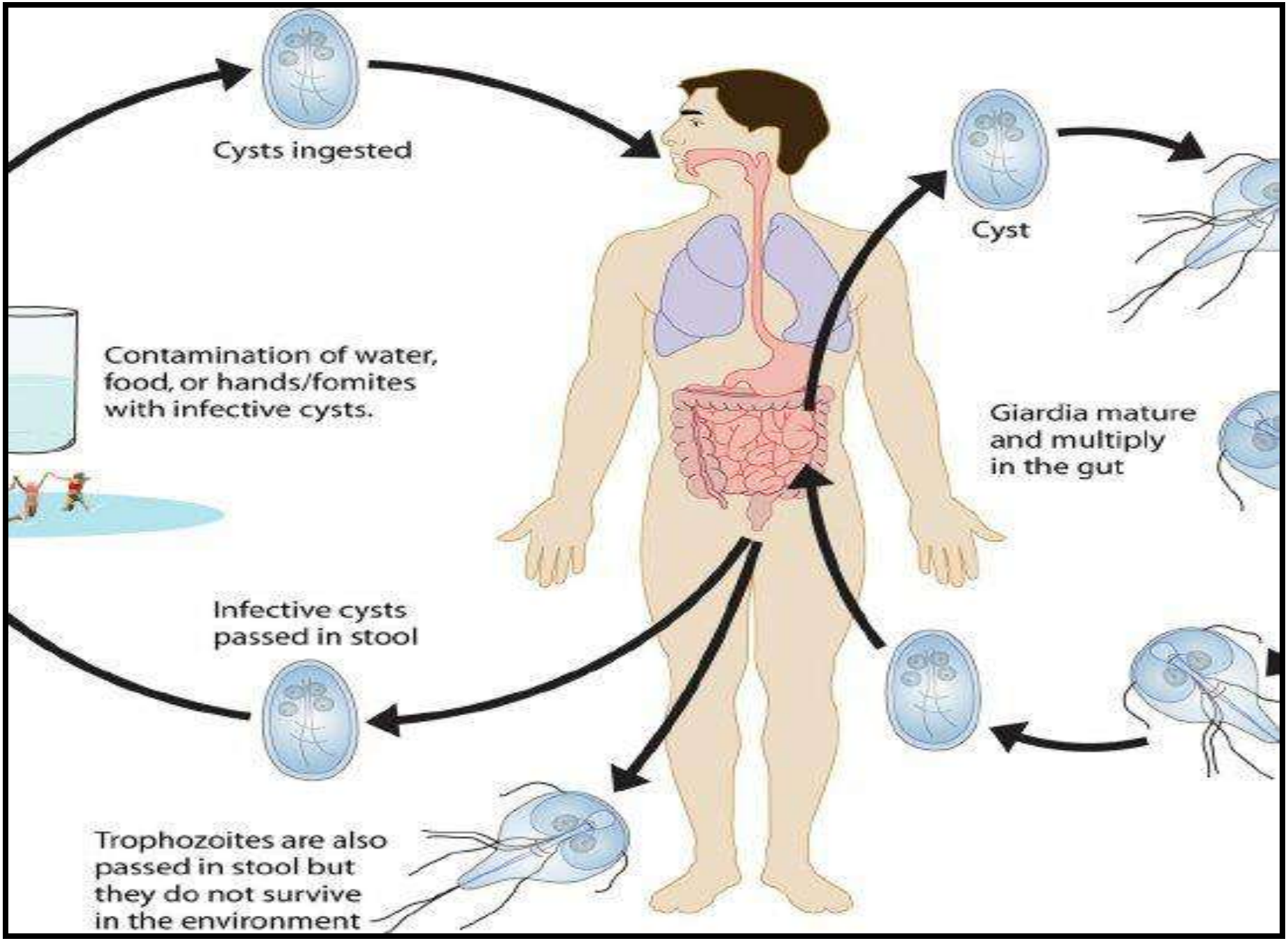
- They resemble a **pear split lengthwise**, **wide and possess eight flagella, 2 nuclei—one on each side of the longitudinal axis**—and **two claw-shaped median bodies**
- **G. intestinalis produces oval cysts with 4 nuclei, flagella, and claw-shaped median bodies.**
- **The cysts (and, less frequently, trophozoites) are excreted in stool.**

Life cycle


Ingestion of dormant cysts
Excystation the trophozoite emerges to an active state of feeding and motility
the trophozoite undergoes asexual replication through longitudinal binary fission
Only about a third of infected people exhibit symptoms.
Both cysts and trophozoites found in the feces
Only the cysts are capable of surviving outside of the host.
The Giardia cyst can survive for weeks to months in cold water

Typical Fecal-Oral Life Cycle




Cysts ingested
Cyst
Contamination of water, food, or hands/fomites with infective cysts.
Giardia mature and multiply in the gut
Infective cysts passed in stool
Trophozoites are also passed in stool but they do not survive in the environment

Pathogenesis & clinical manifestations.

- In the **small intestine, G. intestinalis can cause inflammation as well as other morphological changes and malabsorption.**
- **The course of infection is frequently asymptomatic.**

- Patients with symptomatic infections experience chronic and recurrent diarrhea, steatorrhea, and signs of malabsorption as well as upper abdominal pains, vomiting, occasionally fever, and weight loss.

• Diagnosis

- ❑ Standard diagnostic method is stool examination to detect cysts and (more rarely) trophozoites.
- ❑ ELISA kits are now also available to detect Giardia-specific structural and soluble antigens in stool samples.

• Therapy

- ❑ **Nitroimidazole** compounds are used for chemotherapy of infections, for instance **metronidazole, and tinidazole**.

Antigairdiasis drugs

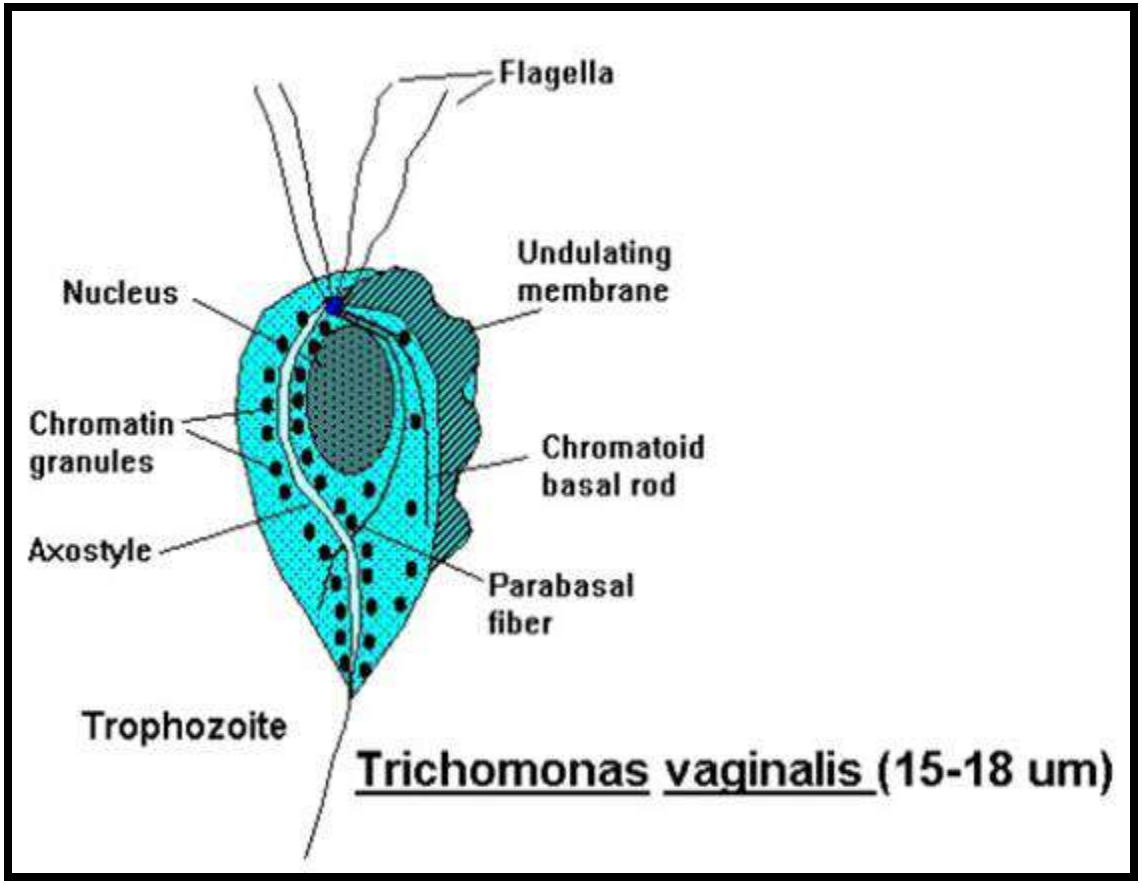
2-Trichomonas vaginalis

Causative agent of trichomonosis

- **Trichomonas vaginalis is a frequent flagellate species that occurs worldwide and is transmitted mainly by sexual intercourse.**
- It **causes vaginitis in women and urethritis in men**

Parasite, life cycle, and epidemiology.

- **Trichomonas vaginalis** is a pearshaped protozoon .



- T. vaginalis colonizes the mucosa of the urogenital tract and reproduces by longitudinal binary fission.

- Trichomonads **do not encyst**, although rounded, nonmotile forms.

- Humans are the sole reservoir of T. vaginalis.

- The parasites are transmitted mainly during sexual intercourse.

Clinical manifestations.

- In women, T. vaginalis primarily colonizes the vaginal mucosa, more rarely that of the cervix.
- In about 20–50% of cases the infection is asymptomatic, but **vaginitis** can develop after an incubation period of two to 24 days.
- The **infection results in** production of a purulent, thin, yellowish discharge in which trichomonads, pus cells, and bacteria are found.

Therapy

- It is **always necessary for both sexual partners to receive treatment**
- **Effective nitromidazole preparations for oral application— in women vaginal application include metronidazole, and tinidazole.**
- These substances **are contraindicated in early pregnancy.**

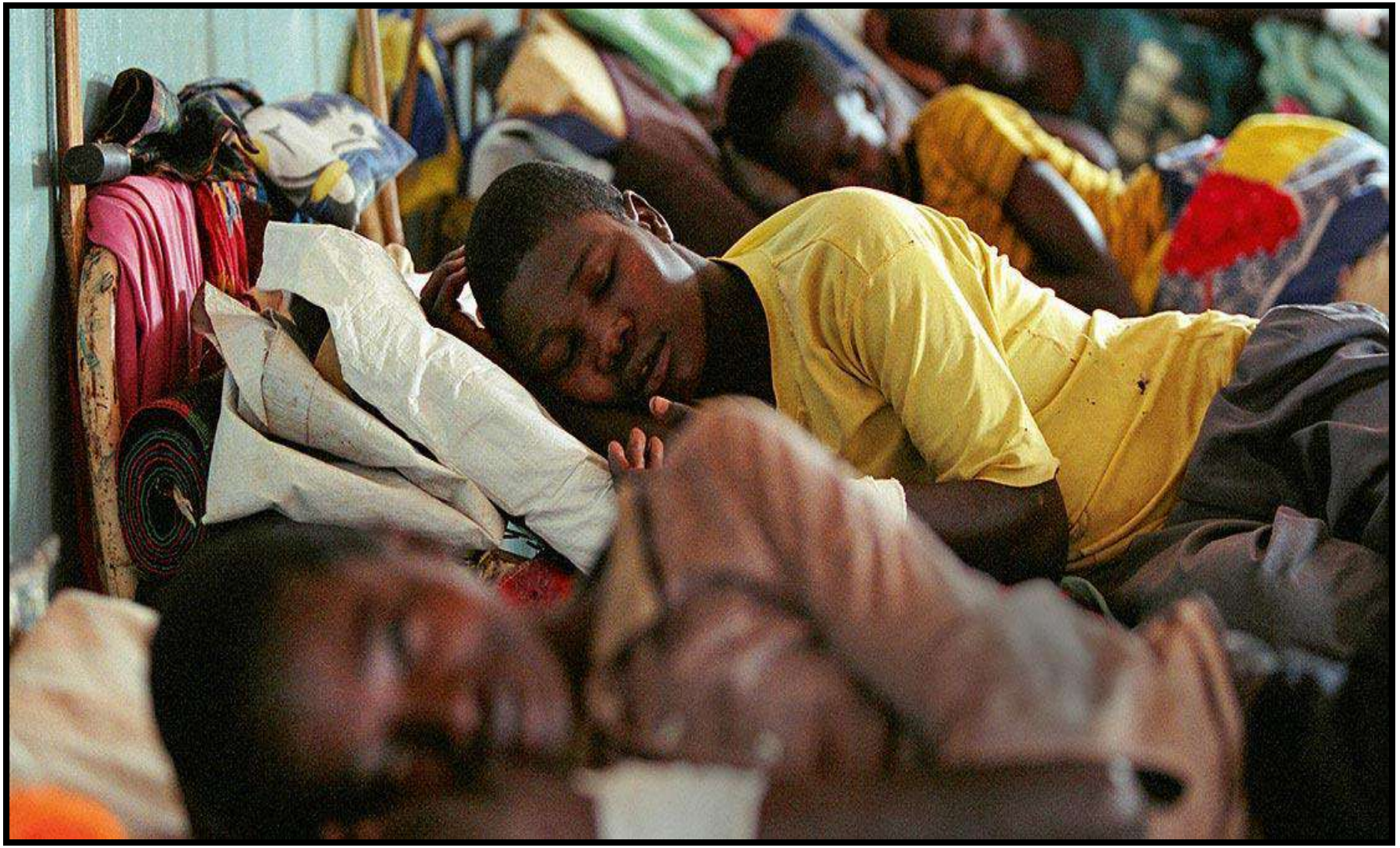
3-Trypanosoma

- Causative agents of African trypanosomosis (sleeping sickness) are Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense
- In a chronic form (T. gambiense) the disease occurs mainly in western and central Africa,
- whereas the acute form (T. rhodesiense) is predominately distributed in eastern and southeastern Africa.

- **The trypanosomes** are **transmitted by the bites of tsetse flies (Glossina).**
- **Antelopes and other wild or domestic animals serve as reservoir hosts of varying significance.**

Tsetse flies

sleeping sickness



Therapy

- Medical treatment of sleeping sickness is highly problematical, since only a small number of effective drugs are available, serious side effects are fairly frequent and drug-resistant trypanosomes are to be expected.

- In stage 1, T. gambiense infections are mainly treated with pentamidine, whereas T. rhodesiense infections are treated with suramin.

- These drugs are not effective in the second stage (cerebrospinal fluid-positive cases), so that the arsenic compound melarsoprol, a relatively toxic substance, must be used in these cases.
- The worst side effect of this substance is a potentially lethal encephalopathy observed in 1–10% of patients treated with melarsoprol.
- Eflornithine is used for treating the late stage of the T. gamibiense infection.

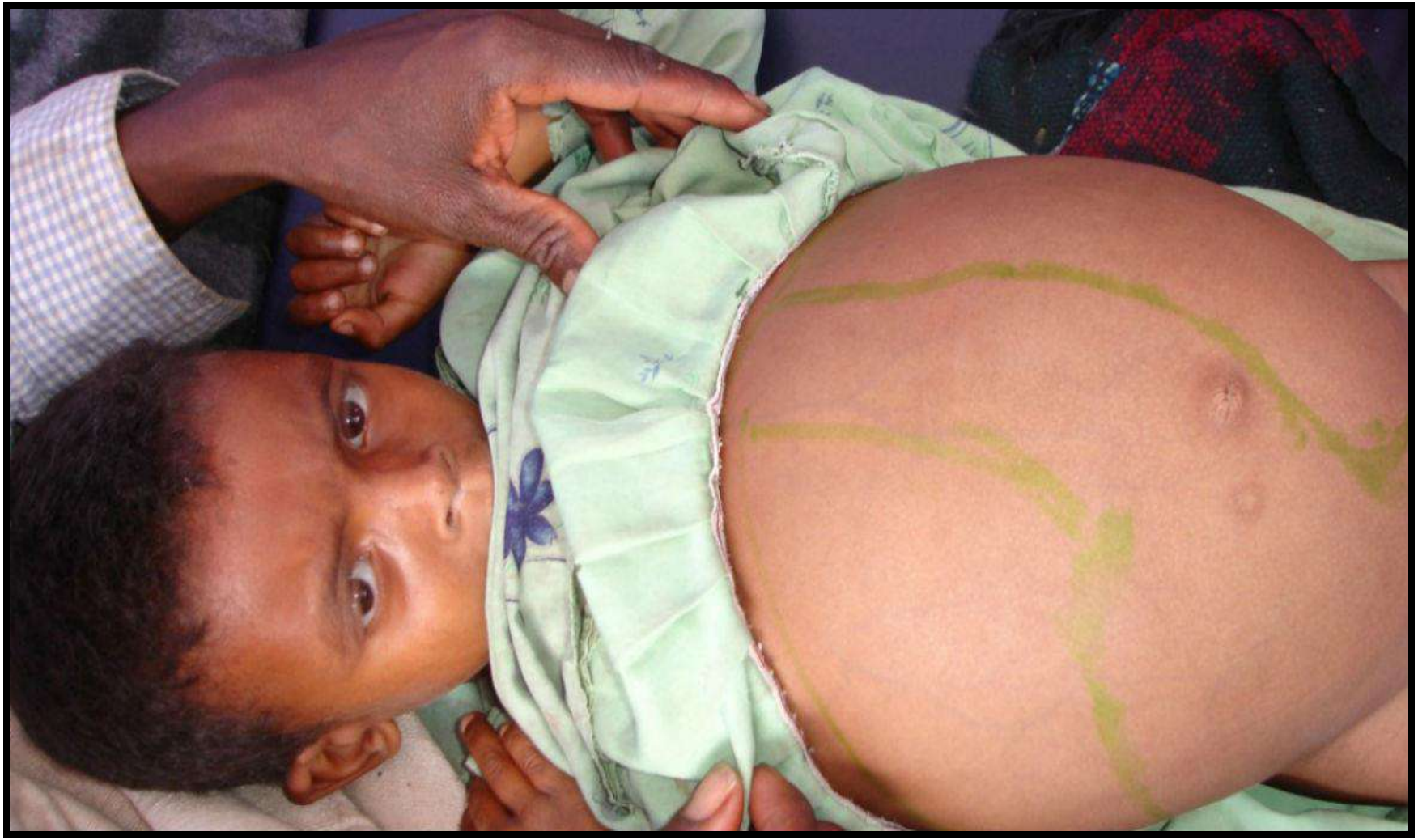
4-Leishmania

Causative agent of leishmanioses

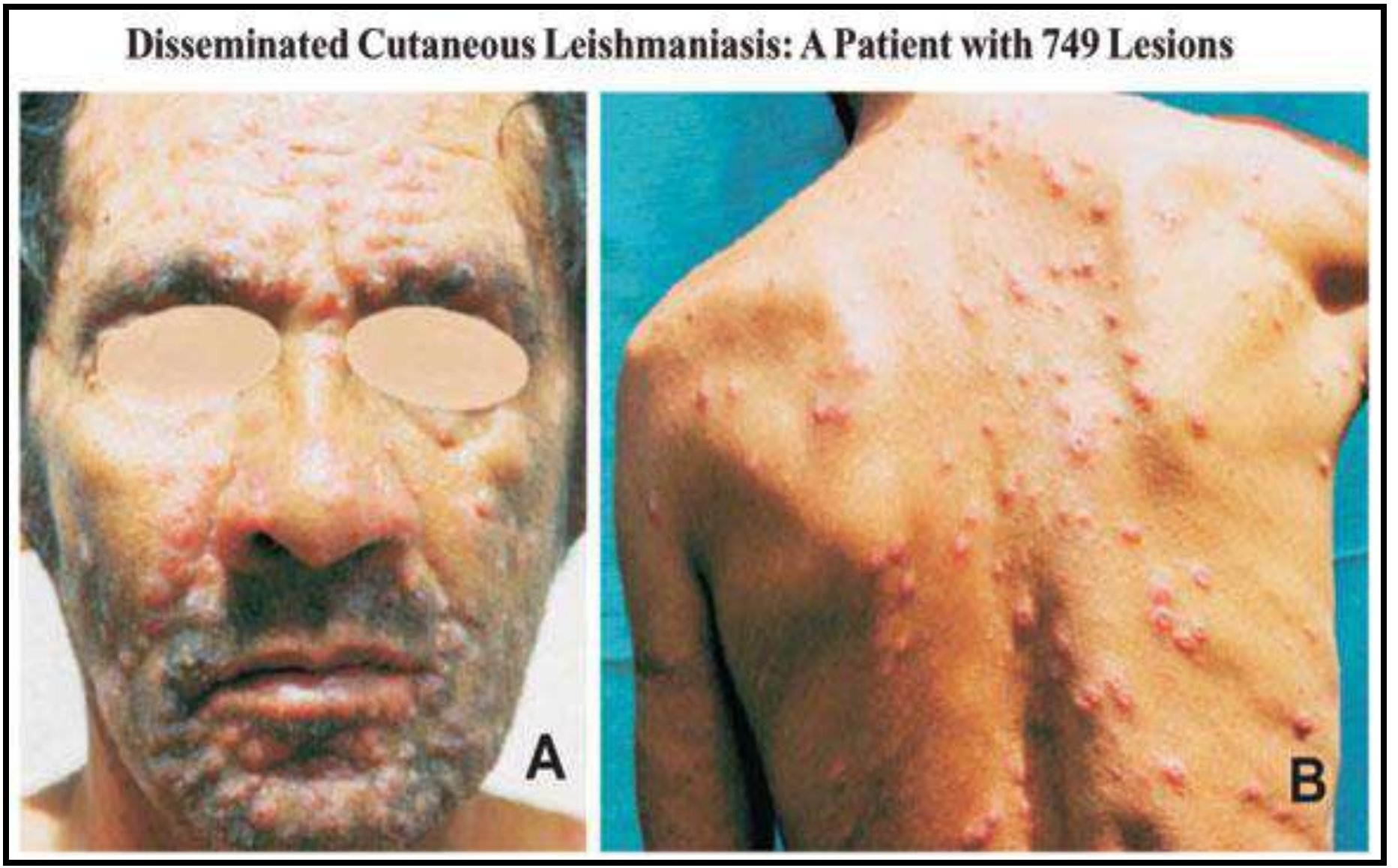
Leishmanias are transmitted by **sandflies (Phlebotomidal)** and cause the following main forms of leishmanioses in warm regions:

- **visceral leishmanioses (VL),**
- **cutaneous leishmanioses (oriental sore) (CL),**
- **and mucocutaneous leishmanioses (MCL).**

Visceral leishmanioses (VL)



Cutaneous leishmaniasis



Leishmania donovani

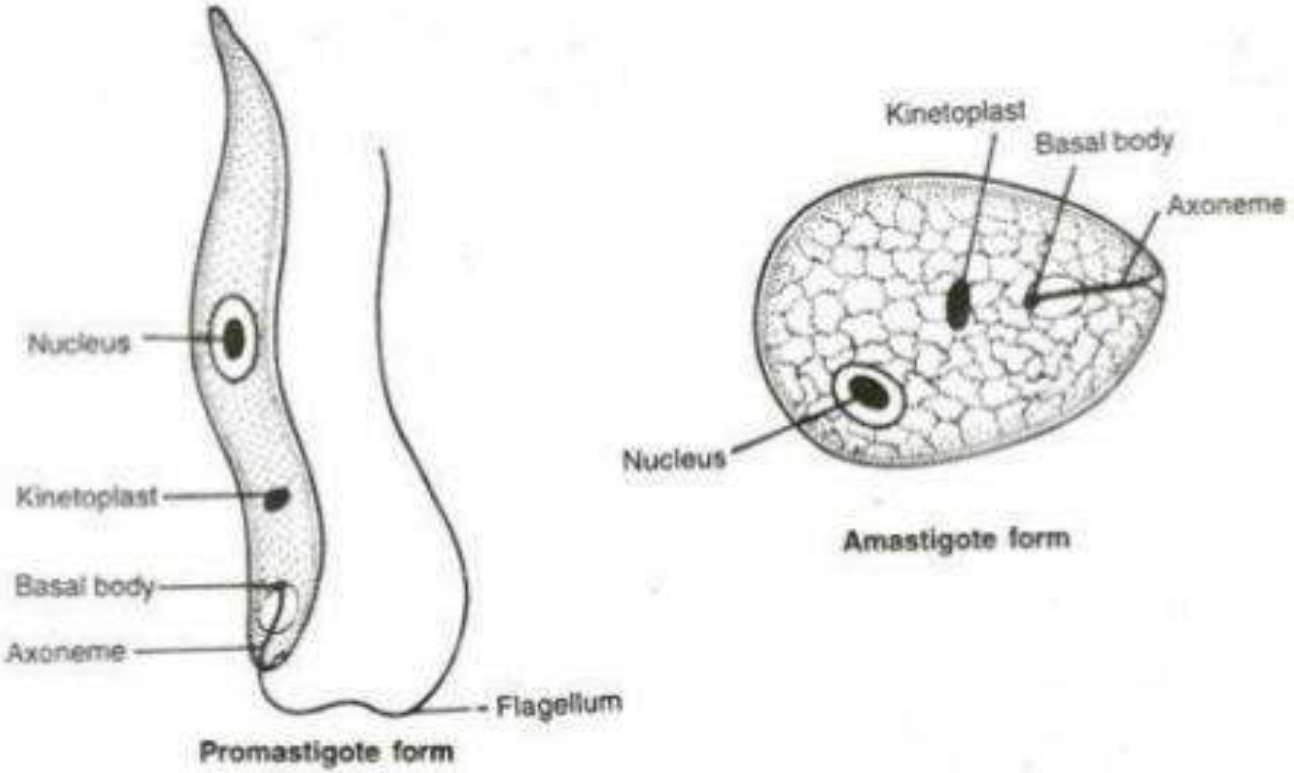


Fig. 178. Morphological forms of *Leishmania donovani*

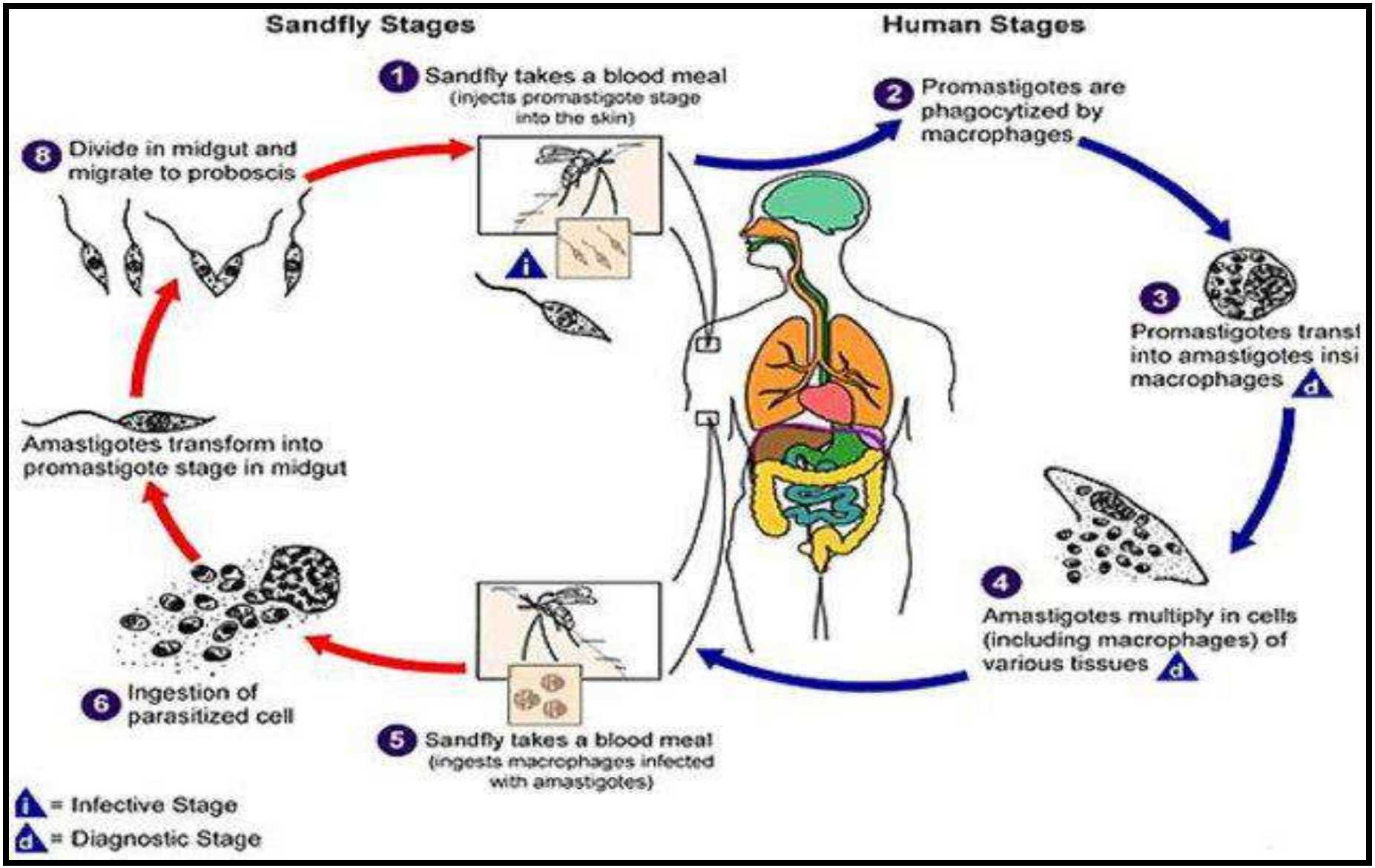
Parasites and life cycle.

- The leishmania species are transmitted by femal mosquitoes of the genera “sandflies” .

- The **amastigote stages** of the parasite ingested by the insect with a blood meal are transformed in its intestine into **slender, flagellate promastigote forms** which multiply and migrate back into the proboscis.

- At tropical temperatures this process takes 5- 8 days.
- When infected sandflies take another bloodmeal the promastigote forms are inoculated into a new host (humans or other vertebrates).

Life cycle of leishmania



Therapy

- Treatment of VL is usually done with **pentamidine.**
- The recurrence rate is relatively high, especially in HIV patients.
- **Miltefosine**, a newly developed and well tolerated antitumor has proved effective against VL.

Chapter 12

Medical Parasitology

Protozoa

B- Sarcodina

(psuedopodia)

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Entamoeba histolytica

- Causative agents of amebosis (**entamebosis, amebiasis).**
- One of the various amebic species that parasitize the human intestinal tract, **Entamoeba histolytica is significant as the causative agent of the worldwide occurring entamebosis, a disease particularly prevalent in warmer countries.**

- The vegetative stages (trophozoites) of E. histolytica live in the large intestine and form encysted stages (cysts) that are excreted with feces.
- The infection is transmitted by cysts from one human to another.

- The trophozoites of E. Histolytica can penetrate into the intestinal wall and invade the liver and other organs hematogenously to produce clinical forms of amebosis, most frequently intestinal ameboses (amebic dysentery) and hepatic amebosis ("amebic liver abscess").

- Diagnosis of an intestinal infection is primarily confirmed by detection of the parasites in stool.

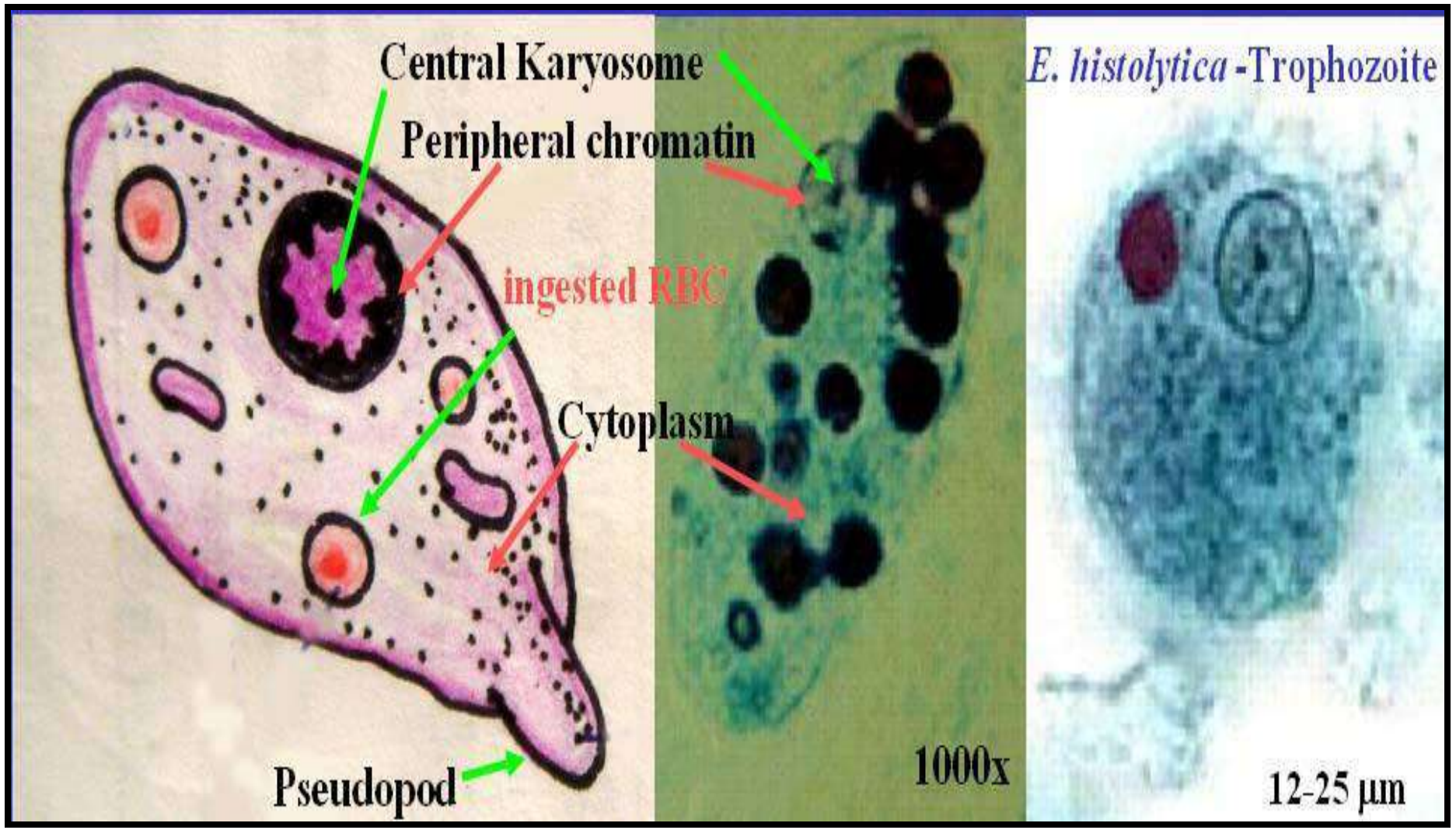
- If an invasive, intestinal or extraintestinal infection with E. histolytica is suspected, a serological antibody test can also provide valuable information.

- The trophozoites of E. histolytica are cells of variable shape and size that usually form a single, broad pseudopod (protrusion of cell membrane and cytoplasm) that is often quickly extended in the direction of movement.
- The spherical, nonmotile cysts have a resistant cyst wall.

- The nucleus divides once to produce the binuclear form and later once again to produce the infective tetranuclear cyst .

- The cysts are eliminated in the stool of infected persons, either alone or together with trophozoites.

Trophozoite of entamoeba histolytica



Cyst of entamoeba histolytica

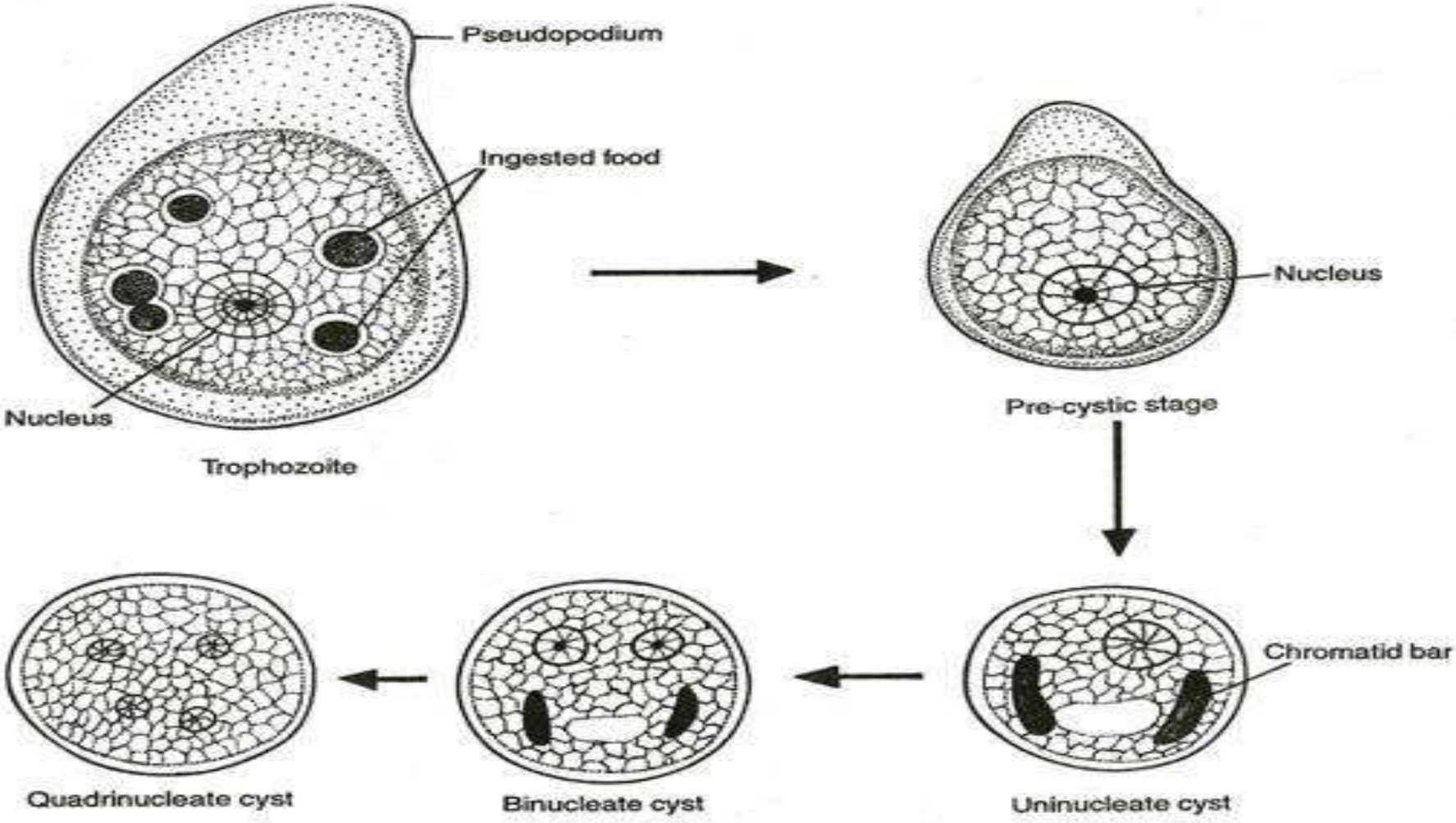
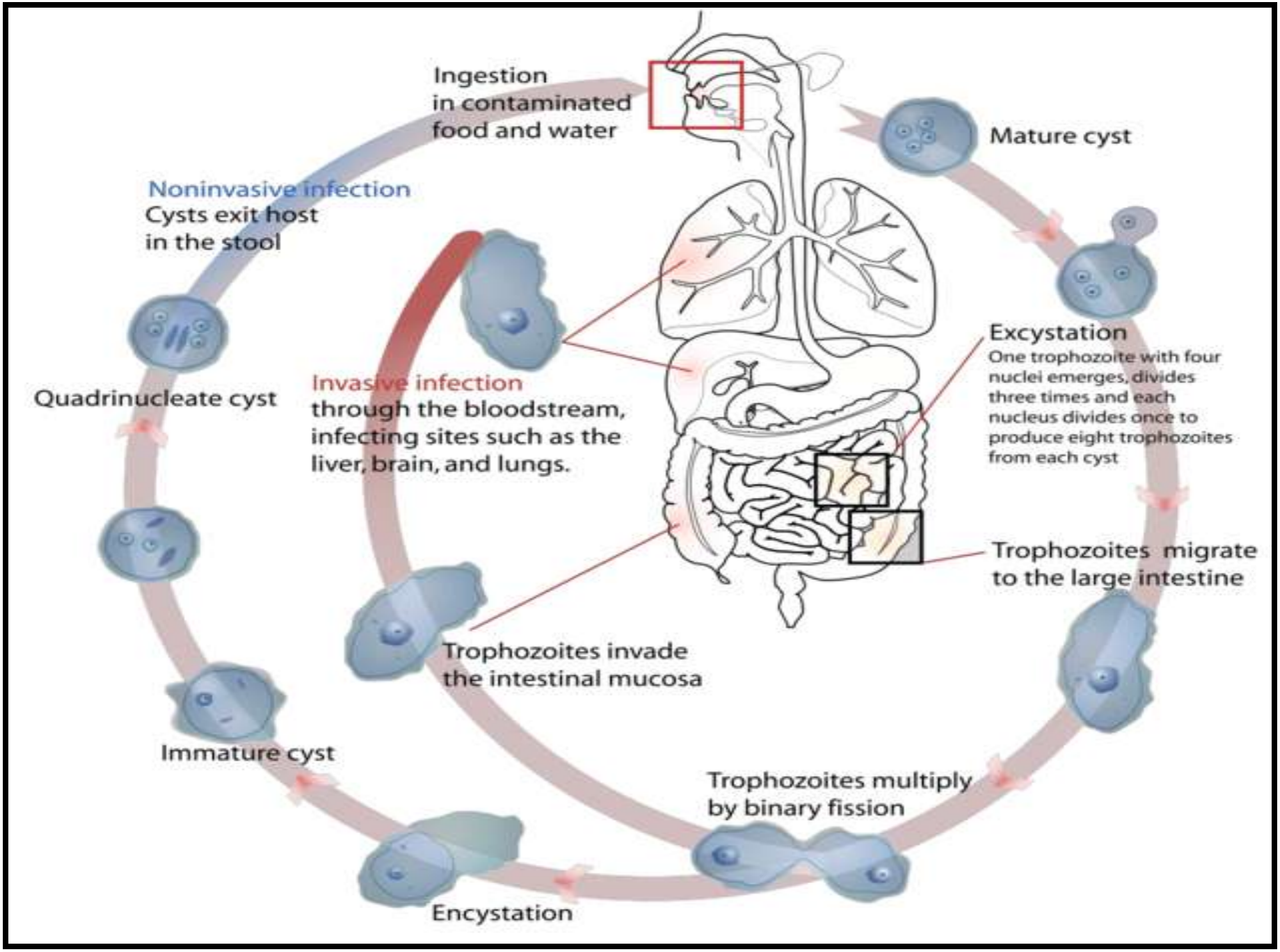


Fig. 174. Stages of life cycle of *Entamoeba histolytica*.


Ingestion
in contaminated
food and water
Mature cyst
Noninvasive infection
Cysts exit host
in the stool
Excystation
One trophozoite with four nuclei emerges, divides three times and each nucleus divides once to produce eight trophozoites from each cyst
Quadrinucleate cyst
Invasive infection
through the bloodstream, infecting sites such as the liver, brain, and lungs.
Trophozoites migrate to the large intestine
Trophozoites invade the intestinal mucosa
Immature cyst
Trophozoites multiply by binary fission
Encystation

Extraintestinal amebosis

- E. histolytica can disseminate to other organs from the intestinal wall, most particularly to the liver .
- As a result of the destruction of parenchymal cells, small necrotic foci, so-called abscesses, form and gradually become larger and can even affect major portions of the organ.

- Liver abscesses sometimes perforate into the pleural space or lung; less often a hematogenous dissemination of amebas results in an invasion of the spleen, brain, and other organs.

Clinical manifestations

- Clinical symptoms can develop as early as two to four weeks after infection with E. histolytica or after asymptomatic periods of months or even years.

- **Intestinal forms**

- Asymptomatic intestinal form. E. histolytica can colonize the intestinal mucosa, reproduce, and persist for long periods without becoming invasive or causing any changes.

- The invasive **intestinal form results from the invasion of the intestinal wall by the pathogenic E. histolytica and reflects large intestine disease.**
- The **ulcers sometimes perforate into the peritoneal cavity.**

- The acute disease usually begins with abdominal discomfort and episodes of diarrhea of varying duration, at first mushy then increasing mucoid, including blood-tinged, so-called "red currant jelly stools" in which amebas can be detected, including trophozoites containing erythrocytes.

- In such cases, antibodies are usually present in serum.
- The symptoms may **abate** spontaneously, but fairly often a recidivating chronic colitis develops that can last for months or even years.

Extraintestinal forms

- Extraintestinal forms develop because of hematogenous dissemination of E. histolytica originating in the intestine.
- The most frequent form is the so called "liver abscess," which may develop in some infected persons.

- The liver abscess causes remittent fever (sometimes high), upper abdominal pain, liver enlargement, elevation of the diaphragm, general weakness, and other symptoms.
- Large liver abscesses that are not treated in time are often lethal.
- Other forms of extraintestinal amebosis are much rarer and include involvement of the lungs, brain, and skin.

Therapy

- Nitromidazole derivatives are effective against symptomatic intestinal and extraintestinal forms of amebosis.

- On the **other hand, amebicides with only luminal activity are effective against asymptomatic intestinal amebosis (e.g., diloxanide furoate)**

Clinical Uses

Amebiasis

Metronidazole or tinidazole

The drug of choice in the treatment of all tissue infections with *E histolytica*.

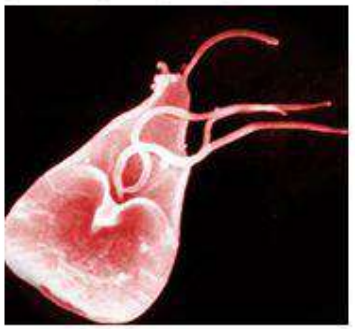
Not effective against luminal parasites and so **must be used with a luminal amebicide to ensure eradication of the infection**.

Giardiasis

Metronidazole is the treatment of choice

Efficacy after a single treatment is about 90%

Tinidazole is equally effective.

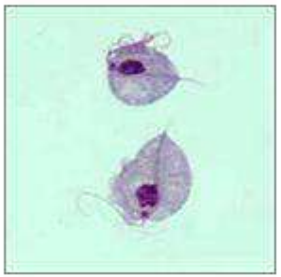


Giardia lamblia

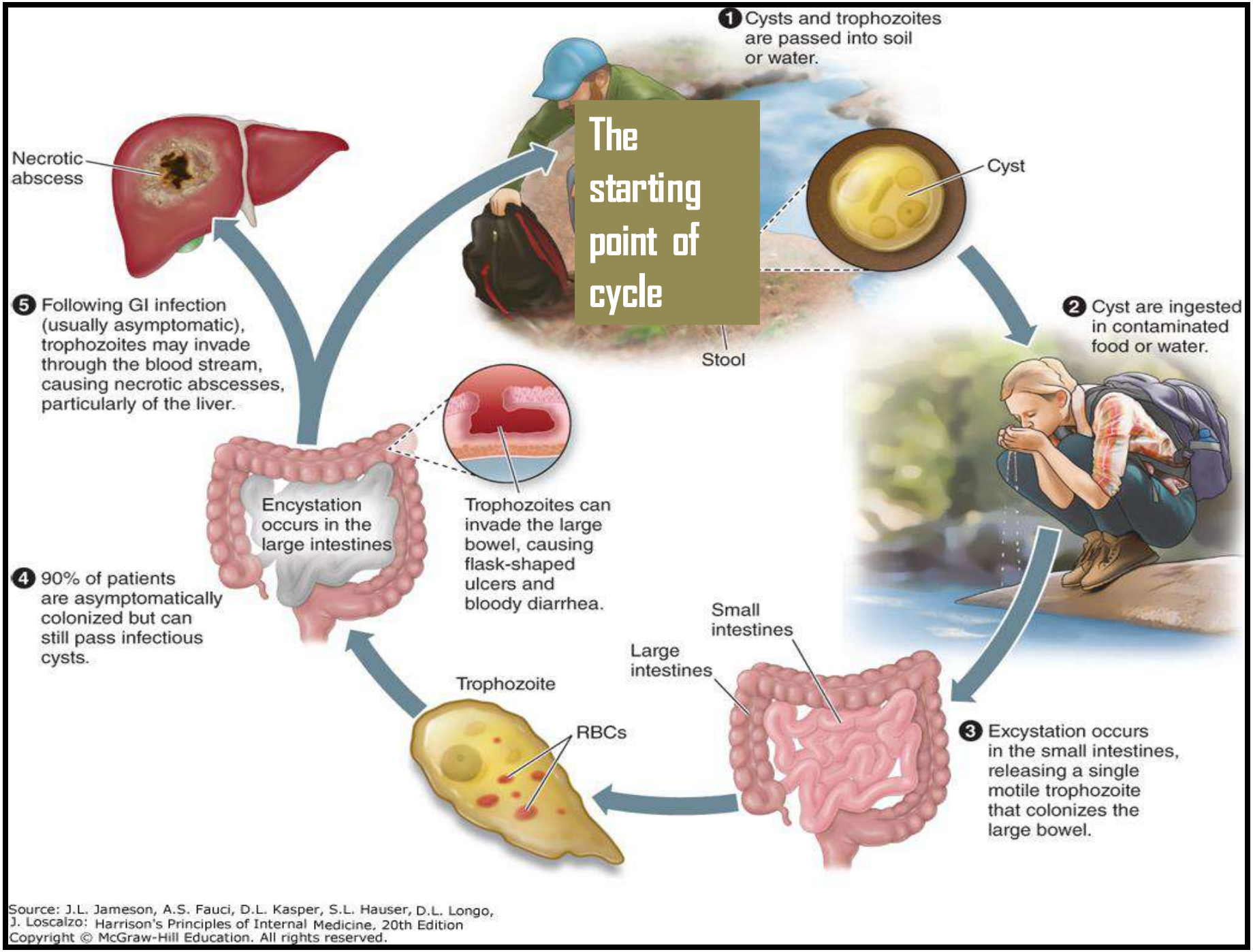
Trichomoniasis

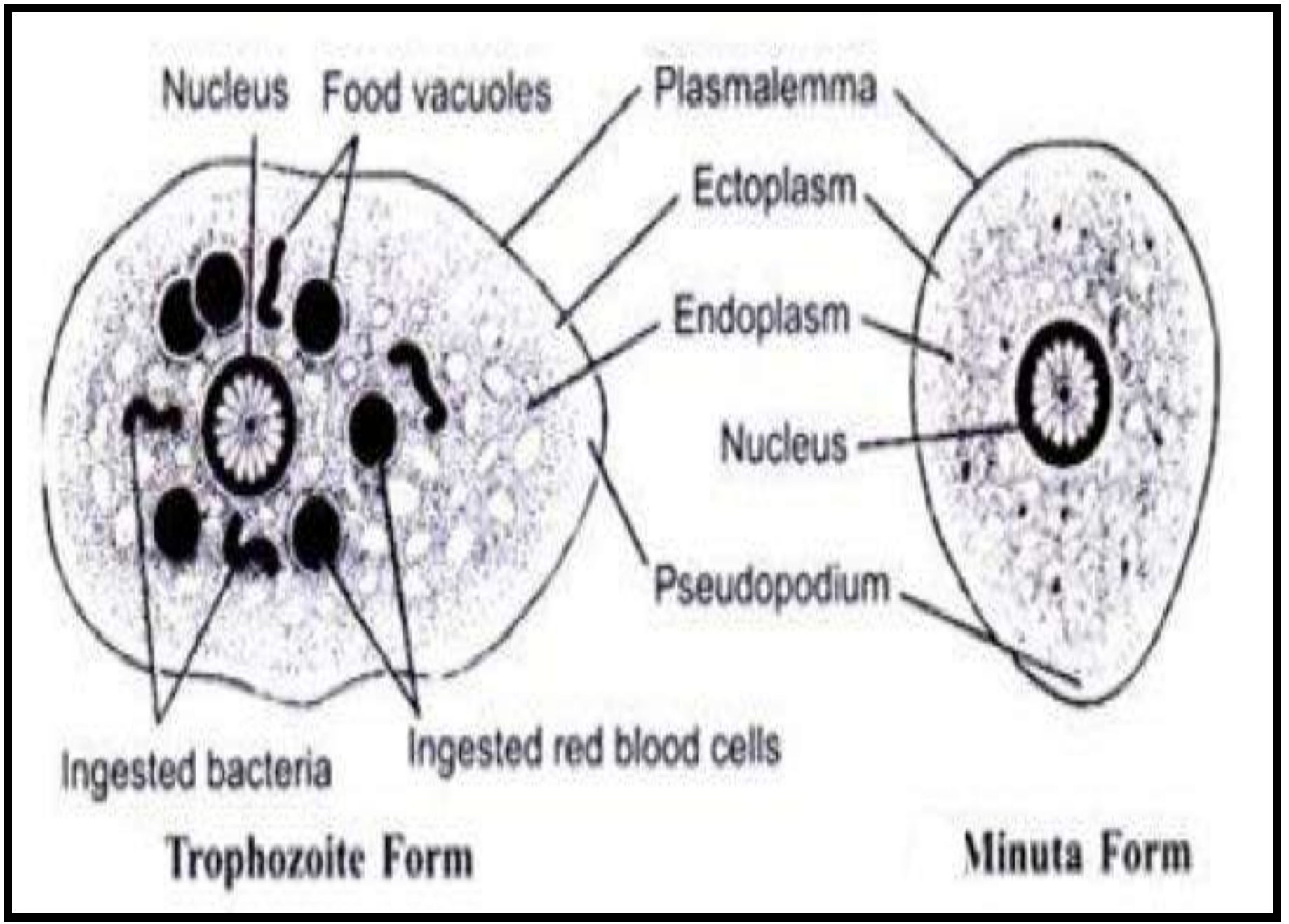
Metronidazole is the treatment of choice.

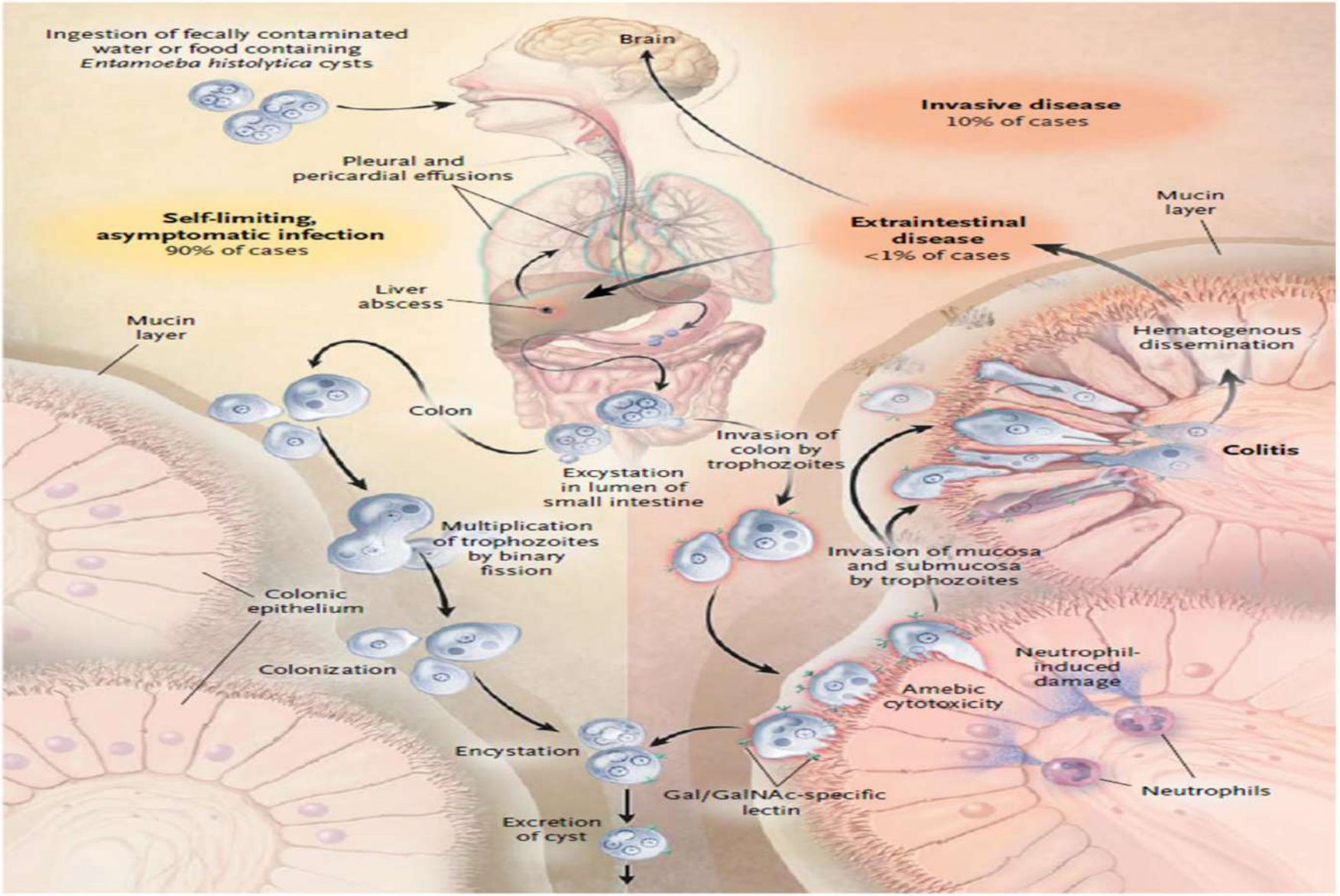
A single dose of 2 g is effective.



Trichomonas vaginalis




Nucleus
Food vacuoles
Plasmalemma
Ectoplasm
Endoplasm
Nucleus
Pseudopodium
Ingested bacteria
Ingested red blood cells
Trophozoite Form
Minuta Form

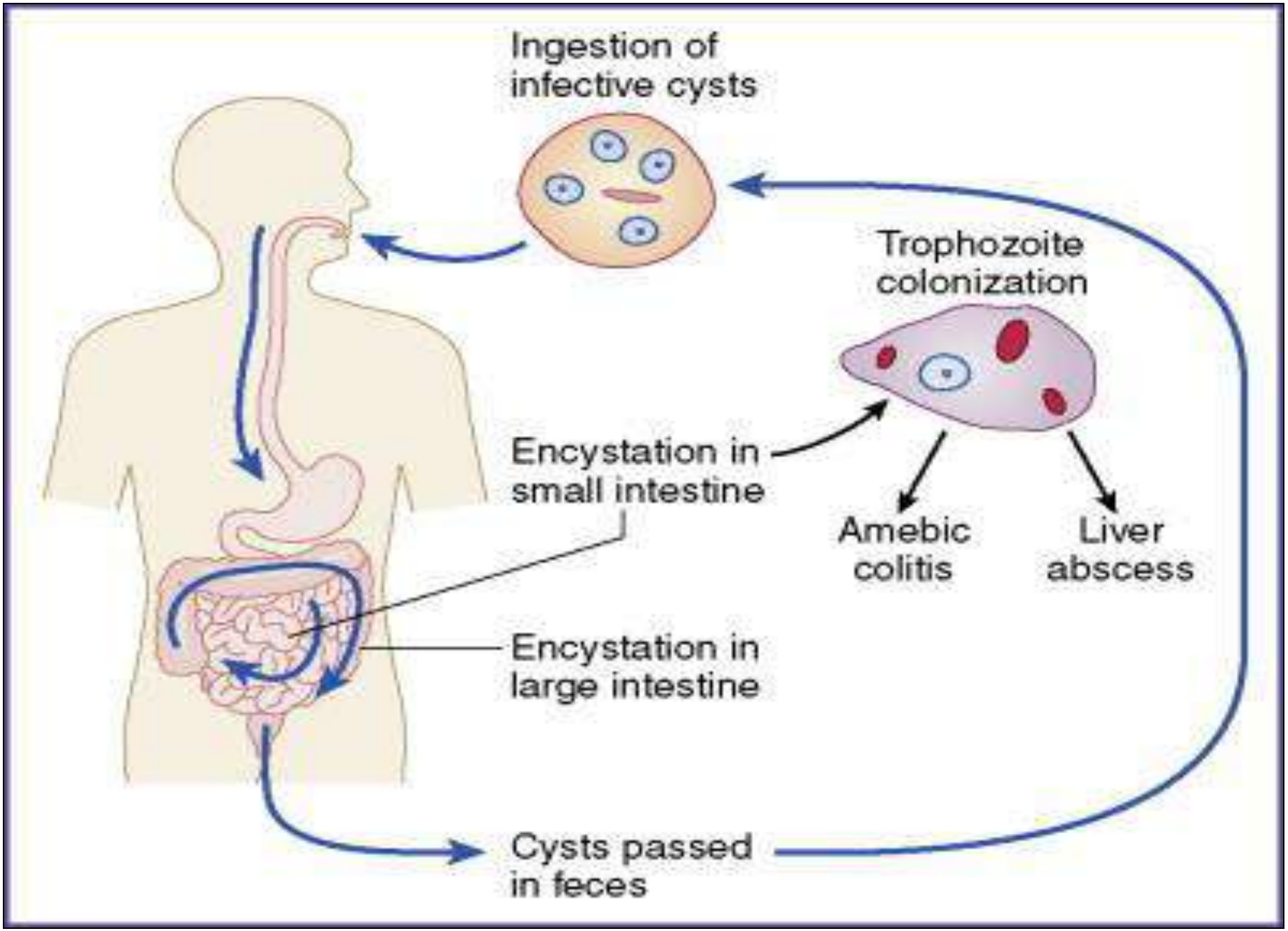

Ingestion of fecally contaminated water or food containing *Entamoeba histolytica* cysts
Brain
Invasive disease
10% of cases
Pleural and pericardial effusions
Self-limiting, asymptomatic infection
90% of cases
Extraintestinal disease
<1% of cases
Mucin layer
Liver abscess
Mucin layer
Hematogenous dissemination
Colon
Invasion of colon by trophozoites
Colitis
Excystation in lumen of small intestine
Multiplication of trophozoites by binary fission
Invasion of mucosa and submucosa by trophozoites
Colonic epithelium
Neutrophil-induced damage
Colonization
Amebic cytotoxicity
Encystation
Gal/GalNAc-specific lectin
Neutrophils
Excretion of cyst

Infections due to *E histolytica*

- **Intestinal disease**
  - Asymptomatic infection
  - Symptomatic noninvasive infection
  - Acute proctocolitis(inflammation of the rectum and colon)
  - Fulminant colitis (coming on suddenly with great severity) with perforation
  - Toxic megacolon (dialated colon)
  - Chronic nondysenteric colitis
  - Ameboma (an inflamed, tumor like, spreading nodule)
  - Perianal ulceration
- **Extraintestinal disease**
  - Liver abscess
  - Pleuropulmonary disease
  - Peritonitis
  - Pericarditis
  - Brain abscess
  - Genitourinary disease

Pathogenesis of Amebiasis

- NON-INVASIVE
  - ameba colony on intestinal mucosa
  - asymptomatic cyst passer
  - non-dysenteric diarrhea, abdominal cramps, other GI symptoms
- INVASIVE
  - necrosis of mucosa → ulcers, dysentery
  - ulcer enlargement → dysentery, peritonitis
  - metastasis → extraintestinal amebiasis


Ingestion of infective cysts
Trophozoite colonization
Encystation in small intestine
Amebic colitis
Liver abscess
Encystation in large intestine
Cysts passed in feces

Chapter 13

Medical Parasitology

Protozoa

C- Sporozoa

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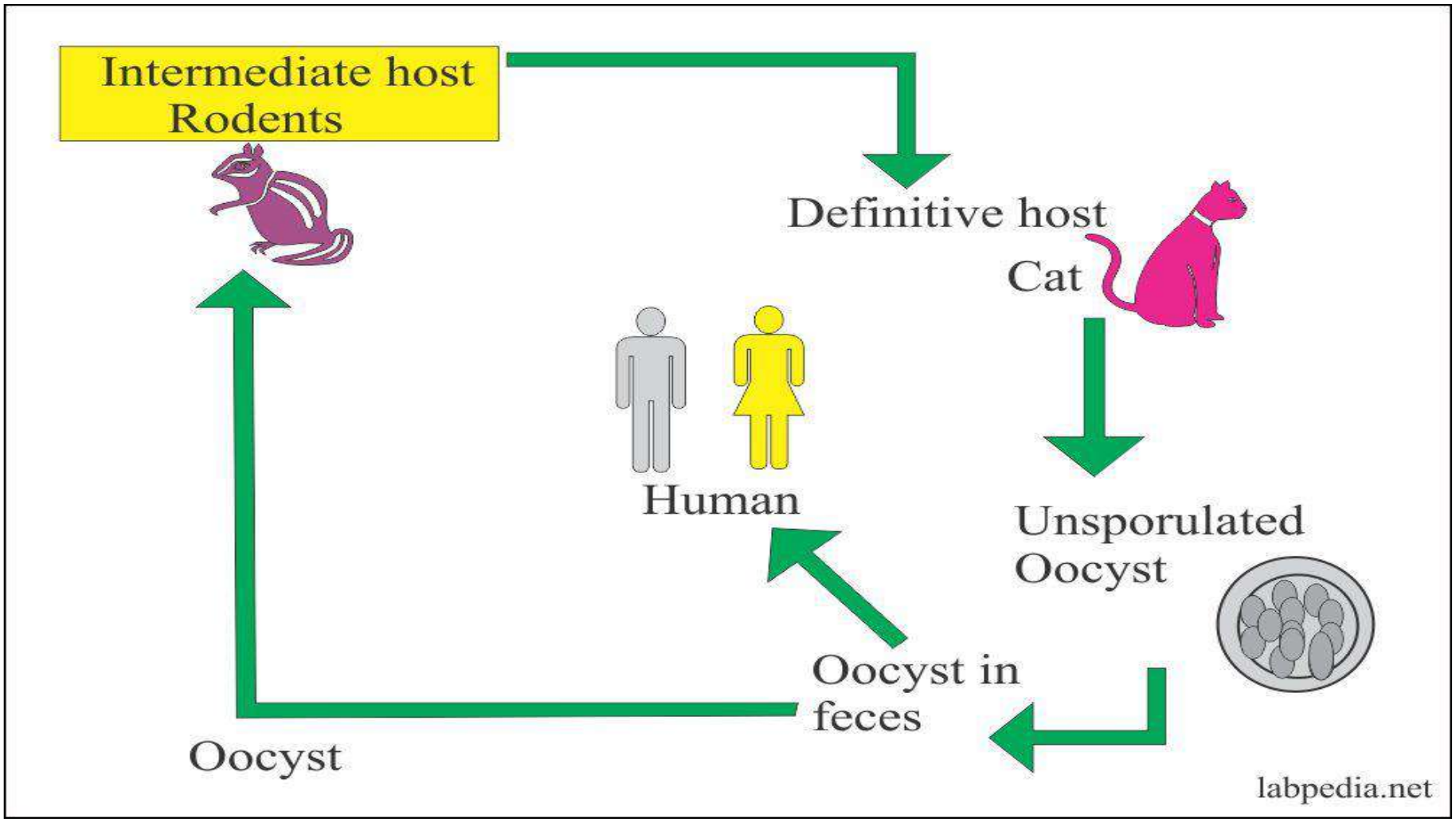
A-Toxoplasma gondii

- Causative agent of toxoplasmosis
- Toxoplasma gondii is the causative agent of a zoonosis that occurs worldwide with high prevalences
- Humans are infected by ingesting oocysts excreted by the definitive hosts (cats) or
- by eating unprocessed meat containing Toxoplasma cysts.

- If a women contracts toxoplasmosis for the first time during pregnancy, diaplacental transmission of the pathogen to the fetus is possible with potential severe consequences (for example malformations, eye damage, clinical symptoms during childhood).

Life cycle


Intermediate host
Rodents
Definitive host
Cat
Human
Unsporulated
Oocyst
Oocyst in
feces
Oocyst
labpedia.net

Therapy & Prevention

- spiramycin daily for 4 w from diagnosis to the end of the 15th week of gravidity.
- Toxoplasma cysts remain viable and infectious in meat for up to three weeks at 4 C.
- Deep-freezing to –20 C kills bradyzoites within three days, heating to 70 C is lethal to them within a few minutes.

- Toxoplasma oocysts show considerable environmental resistance, but can be killed rapidly by heat (70 C).
- Pregnant women should eat only meat that has been thoroughly heated or deep-frozen.
- Cats can be fed canned (boiled) meat to protect them from infection.

B- Plasmodium

- Causative agent of malaria

- **Malaria,** the most frequent tropical parasitosis.

- The infectionis caused by plasmodia (Plasmodium vivax, P. ovale, P. malariae, P. falciparum) transmitted by the bite of Anopheles mosquitoes.

- An infection initially presents in nonspecific symptoms (headache, fatigue, nausea, fever).

- Untreated malaria tropica (caused by P. falciparum) can quickly develop to a lethal outcome.
- Prophylactic measures are essential for travelers to regions where malaria is endemic (prevention of mosquito bites, chemoprophylaxis)

Occurrence

- Malaria is one of the most significant infectious diseases of humans.
- According to the WHO (2000, 2004), the disease is currently endemic in more than 100 countries or territories.
- About 2.4 billion people (40% of the world's population) live in malarious regions.

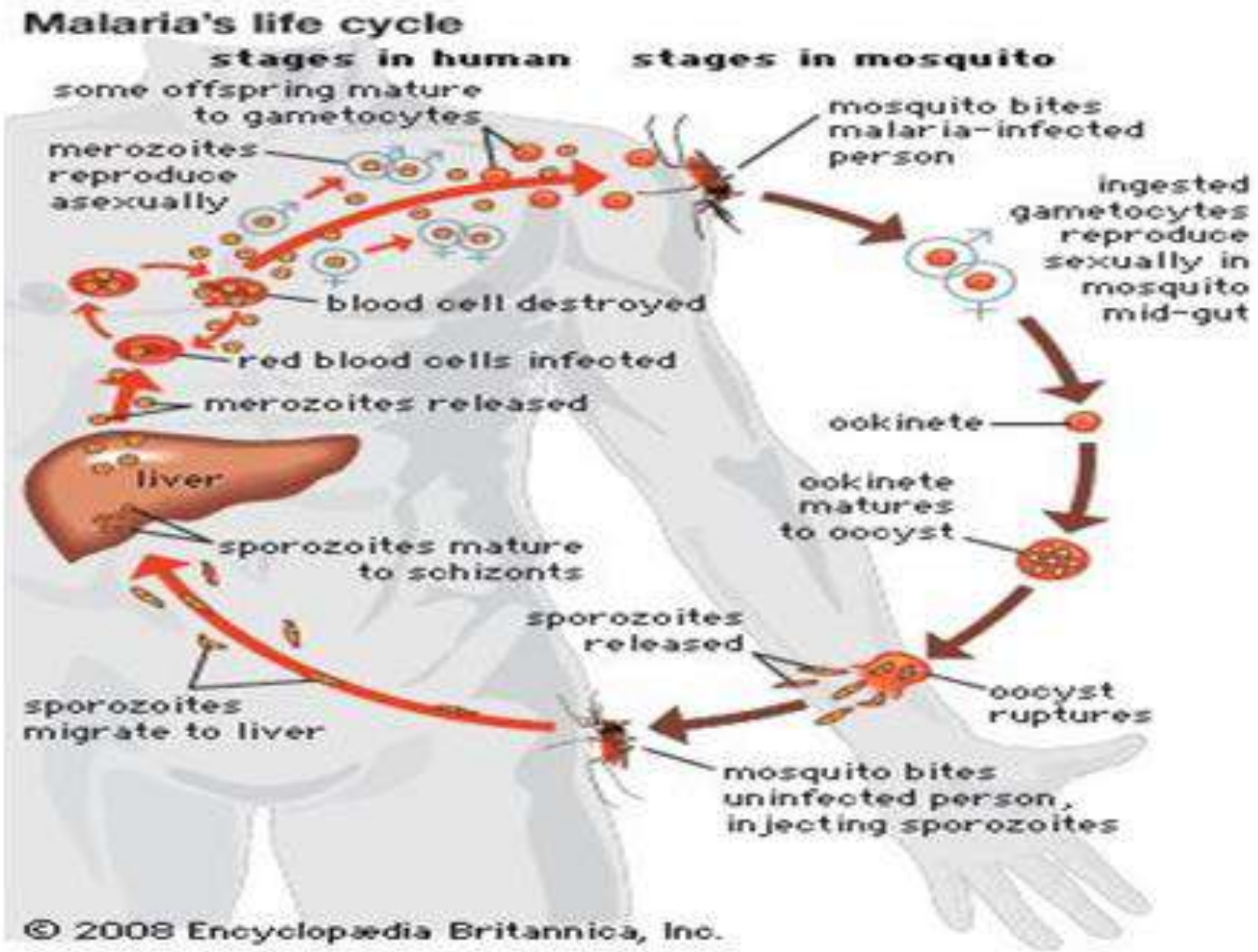
- The annual incidence of malaria worldwide is estimated to be **300–500 million** clinical cases, with about 90% of these occurring in sub-Saharan Africa (mostly caused by P. falciparum).
- Malaria alone or in combination with other diseases kills approximately 1.1–2.7 million people each year, including 1 million children under the age of 5 years in tropical Africa.

Parasites.

4 Plasmodium species infect humans and cause different types of malaria:

- **Plasmodium vivax:** tertian malaria (malaria tertiana)
- **Plasmodium ovale:** tertian malaria (malaria tertiana)
- **Plasmodium malariae:** quartan malaria (malaria quartana)
- **Plasmodium falciparum:** malignant tertian malaria (malaria tropica).

- These **Plasmodium species can be identified and differentiated from each other by** light microscopy in stained blood smears during the erythrocytic phase of the infection in humans .


Malaria's life cycle
stages in human
stages in mosquito
some offspring mature to gametocytes
merozoites reproduce asexually
blood cell destroyed
red blood cells infected
merozoites released
liver
sporozoites mature to schizonts
sporozoites migrate to liver
mosquito bites malaria-infected person
ingested gametocytes reproduce sexually in mosquito mid-gut
ookinete
ookinete matures to oocyst
oocyst ruptures
sporozoites released
mosquito bites uninfected person, injecting sporozoites
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Clinical manifestations & Incubation periods.

- The clinical manifestations of malaria are caused by the asexual erythrocytic stages of the plasmodia .
- The incubation periods vary, depending on the Plasmodium species involved, from 7 to 35 days after infection.
- These periods can, however, be extended by weeks or even months, particularly if the infection is suppressed by prophylactic medication.

- The clinical manifestations of malaria depend on a number of different factors, above all the Plasmodium species and immune status of the patient.
- The Plasmodium species with the most pronounced pathogenicity is Plasmodium falciparum, which causes "malignant tertian malaria" (malaria tropica), whereas the other Plasmodium species cause milder forms ("benign malaria").

• Classic malarial paroxysm

- After an initial rise in temperature to about 39 c, peripheral vasoconstriction causes a period of chills (lasting for about 10 minutes to one hour), then the temperature once again rises to 40–41 C (febrile stage 2-6 h), whereupon peripheral vasodilatation and an outbreak of sweating follow.

- These bouts occur mainly in the afternoon and evening hours.
- Once the paroxysm has abated and the fever has fallen, the patient feels well again until the next one begins.
- In severe malaria tropica, however, circulatory disturbances, collapse, or delirium may occur without fever (algid malaria).

Types of malaria

- **Tertian malaria (malaria tertiana)**

- caused by **P. vivax or P. ovale:**
- **Incubation period**: 9to 20 days, also several weeks or months.
- **Parasitemia:** generally low level, up to a maximum of 1–2%.
- **Course:** usually benign ("benign malaria").
- Febrile stage 3-4 h, **again 48 hours later.**

Quartan malaria (malaria quartana)

- caused by **P. malariae**
- **Incubation period:** 15–40 days (usually longer than with other species).
- **Parasitemia:** generally low level, up to a maximum of 1%.
- **Course:** usually benign. Febrile stage 4-5 hours, again **72 hours later**.

- **Malignant tertian malaria (malaria tropica)**
- Caused by **P. falciparum**
- **Incubation period**: **7- 15 days** **or longer.**
- **Parasitemia:** often at **very high level, up to 20%** or more!
- **Course:** initial symptoms often more pronounced than in other types.
- Rapid, severe course in nonimmune persons.
- **High lethality rate if untreated** **(50–60% in persons from central Europe).**

Diagnosis.

- Etiological confirmation of a clinical diagnosis is obtained by detecting malarial parasites in the blood .
- Stages of P. falciparum, P. vivax, and P. ovale can be found in blood 5-8 days after the infection at the earliest.
- P. malariae not until after 13–16 days.
- DNA detection by means of (PCR)can be used to identify the different Plasmodium species for research purposes.

Therapy

Drugs used in treatment of Malaria

- Cinchona alkaloids: quinine,quinidine
- 4 aminoquinolines:
  chloroquine, hydroxychloroquine, amodiaquine,
- 8 aminoquinolines: primaquine, tafenoquine, bulaquine
- quinoline methanol: mefloquine, halofantrine, lumefantrine
- Antifolates:
  - pyrimethamine , proguanil, sulfadoxine
- Antibiotics: tetracycline, doxycycline, clindamycin
- Hydronaphthoquinone: Atovaquone
- Qinghaosu compounds: Artesunate, artemether, arteether

Control Strategy for Malaria

Kill asexual forms

Prevent development within mosquito

? Vaccine

Plasmodia

The Agent

Kill sexual forms

Chloroquine + Primaquine

Early diagnosis and treatment

Prevent breeding

Avoid water logging; kill larvae

Prevent entry

Close doors/windows

Deny blood meal

Protect from mosquito bites

Prevent biting

Kill adult mosquitoes

Nets, repellents

Man

The Host

Mosquito

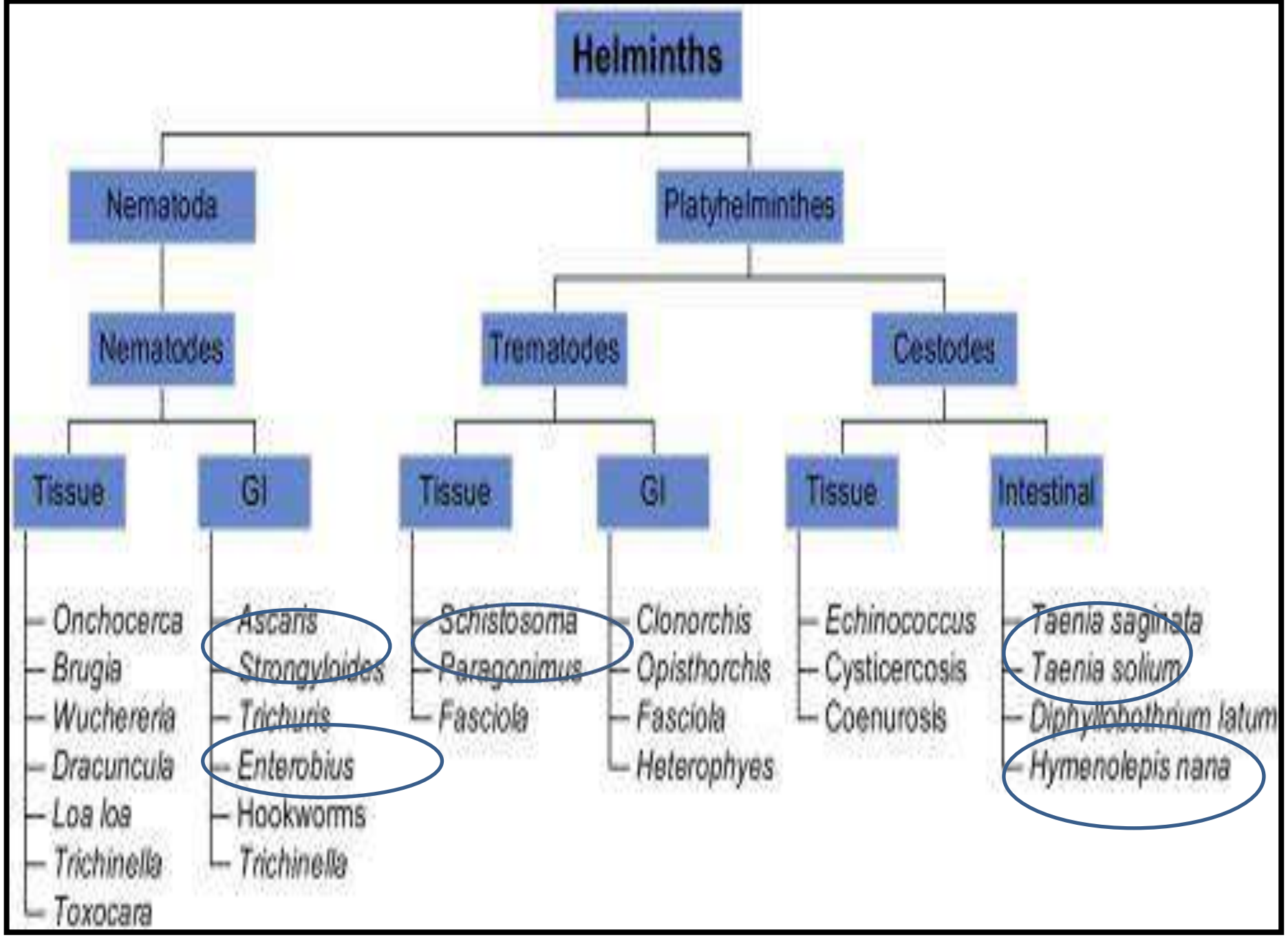
The Vector

Chapter 14

Helminths

Medical Parasitology

Dr. Mohammed Hussien Taleb


Helminths
Nematoda
Platyhelminthes
Nematodes
Trematodes
Cestodes
Tissue
GI
Tissue
GI
Tissue
Intestinal
Onchocerca
Brugia
Wuchereria
Dracuncula
Loa loa
Trichinella
Toxocara
Ascaris
Strongyloides
Trichuris
Enterobius
Hookworms
Trichinella
Schistosoma
Paragonimus
Fasciola
Clonorchis
Opisthorchis
Fasciola
Heterophyes
Echinococcus
Cysticercosis
Coenurosis
Taenia saginata
Taenia solium
Diphyllobothrium latum
Hymenolepis nana

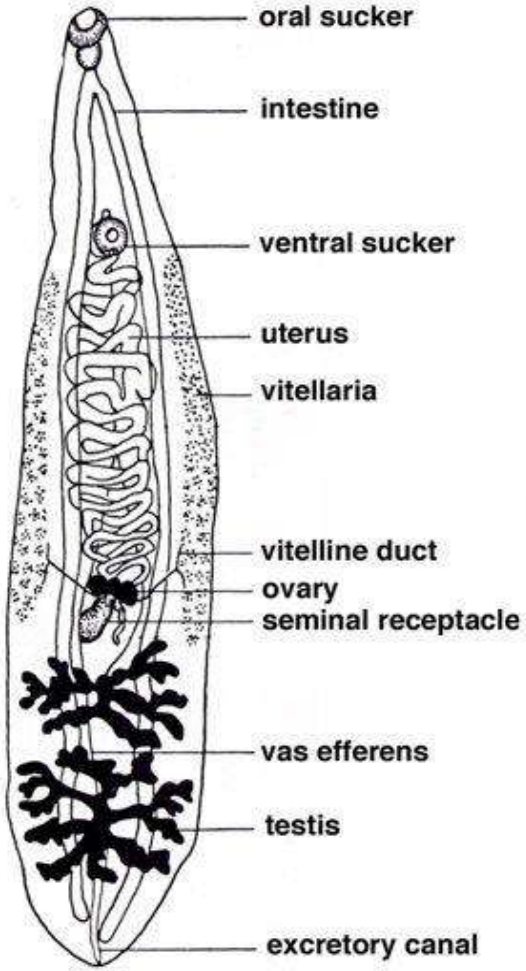
A-Trematoda (Flukes)

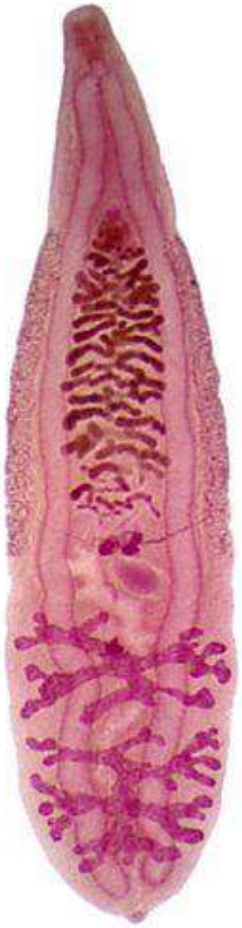
Most of the trematode species that parasitize humans are dorsoventrally flattened with an oval to lancet shape, although others have different shapes such as the threadlike schistosomes.

Most species are hermaphroditic, only the schistosomes have separate sexes.

Snails are the first intermediate hosts; some species require arthropods or fish as second intermediate hosts.

trematodes or flukes - know your worm





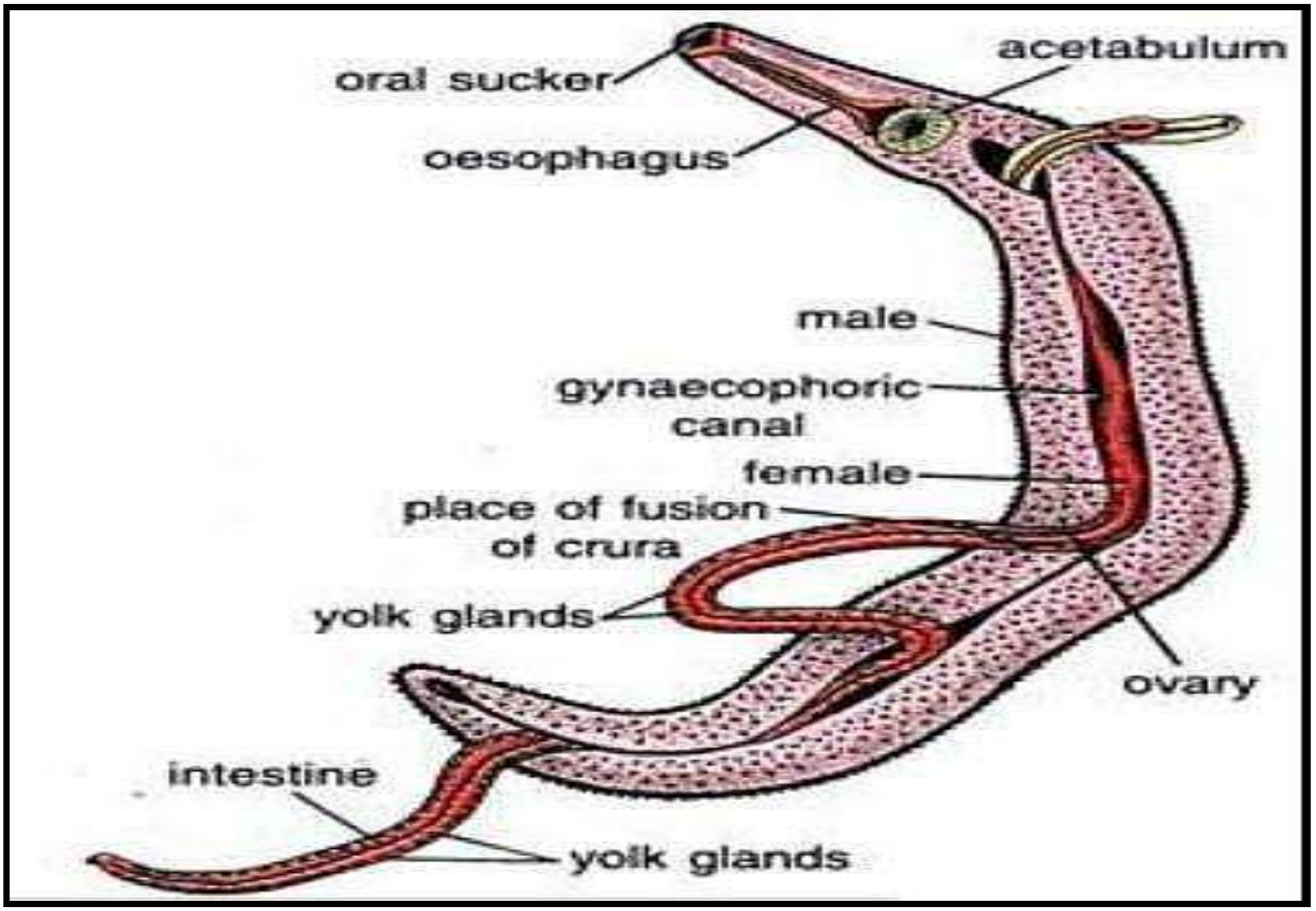
- Digenea posses two suckers (oral and ventral acetabulum) which they use to attach within the host
- Oral sucker contains the mouth
- Muscular pharynx permits the worm to pump food into the blind ending gut
- Most trematodes are hermaphrodites (they are male and female, and cross as well as self-fertilization occurs)

1-Schistosoma (Blood Flukes)

- Causative agents of schistosomosis or bilharziosis
- **Schistosomosis** (bilharziosis) is one of the **most frequent tropical diseases with about 200 million infected persons.**
- The occurrence of **schistosomosis depends on the presence of suitable intermediate hosts (freshwater snails).**
- **Human infections result from contact with standing or slow-moving bodies of water (freshwater) when Schistosoma cercariae penetrate the skin.**

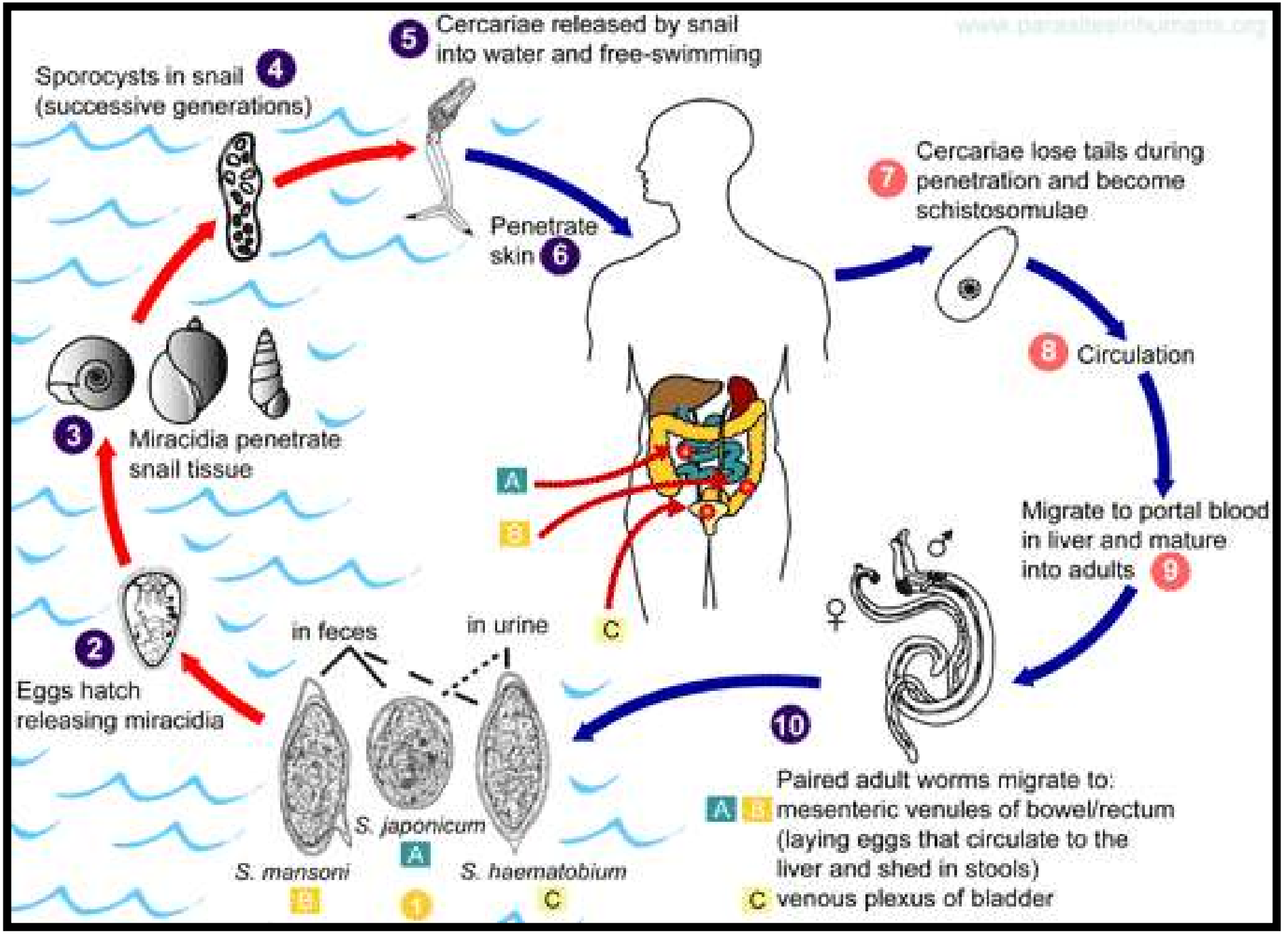
- Schistosoma hematobium causes urinary schistosomosis;
- S. mansoni, S. japonicum, of intestinal schistosomosis and other forms of the disease.
- Diagnosis can be made by detection of either Schistosoma eggs in stool or urine or of specific antibodies in serum.

Male and female of schistosoma



Parasite species and occurrence.

- Also known as bilharziosis after the German physician **Th. Bilharz,** who discovered **Schistosoma hematobium in human blood vessels in 1851**.
- The **most** **important species pathogenic to humans are Schistosoma hematobium**


Sporocysts in snail (successive generations)
4
5
Cercariae released by snail into water and free-swimming
Penetrate skin
6
7
Cercariae lose tails during penetration and become schistosomulae
8
Circulation
Migrate to portal blood in liver and mature into adults
9
3
Miracidia penetrate snail tissue
2
Eggs hatch releasing miracidia
in feces
in urine
A
B
C
10
Paired adult worms migrate to:
A B mesenteric venules of bowel/rectum (laying eggs that circulate to the liver and shed in stools)
C venous plexus of bladder
S. japonicum
A
S. mansoni
B
S. haematobium
C

- **Urinary schistosomosis (urinary bilharziosis).**
- **Causative agent: S. hematobium.**

- Incubation 10–12 weeks or longer, morbidity rate as high as 50–70%.
- Hematuria (mainly in the final portion of urine),
- micturition discomfort,
- ulcers and calcification of the bladder wall,
- pyelonephrosis and hydronephrosis.

- **Intestinal schistosomosis (intestinal bilharziosis) &others.**

❑ Causative agents: mainly S. mansoni and S. japonicum,

❑ Incubation 4 to 13weeks (acute phase), months to years (chronic phase).

Therapy

- The drug of choice for treatment of schistosomosis is **praziquantel,** which is highly effective against all Schistosoma species and is well tolerated.
- Oxamniquine is effective against S. mansoni.

2-Fasciola species

- Fasciola hepatica (Common Liver Fluke)
- Fasciola hepatica and F. gigantica are frequent bile duct parasites of domestic ruminants.
- In their life cycle freshwater snails act as intermediate hosts.
- Humans become accidentally infected when they eat plants (e.g., watercress) to which infectious parasite stages (metacercariae) adhere.

Clinical manifestations.

The infection may run an inapparent course or, after an incubation period of 4-6 weeks, become symptomatic with abdominal pain, hepatomegaly, fever, leukocytosis and eosinophilia (acute phase), or hepatocholangitic symptoms (chronic phase) and anemia.

- Occasionally, the parasites also migrate into other organs than the liver.

Therapy

- The drug of choice is triclabendazole
- The infection can be avoided by not eating raw watercress and other plants that may be contaminated with metacercariae.

B-Cestoda (Tapeworms)

- The life cycle of cestodes include one or two intermediate hosts.
- Humans can also be infected by larval stages of various tapeworm species (cysticerci, metacestodes).
- These stages develop in body tissues and generally cause considerably greater pathological damage than the intestinal cestode stages.

1-Taenia species

- **Causative agents of taeniosis &cysticercosis**
- **Taeniosis is a small intestine infection of humans** caused by Taenia species.
- In the case of **T. saginata**, the intermediate hosts are **cattle**, in the musculature of which **metacestodes (cysticerci)** develop and can be **ingested by humans who eat raw beef.**

1-Taenia saginata (Beef Tapeworm)

- Causative agent of T. saginata taeniosis

- **Occurrence.**

- This species occurs worldwide; the number of infected humans is estimated to be between 40 and 60 million..

Pathogenesis and clinical manifestations.

- The infection takes an asymptomatic course in about 25% of cases.
- Symptoms of infection include nausea, vomiting, upper abdominal pains, diarrhea or constipation and increased or decreased appetite.

Therapy

- The drug of choice is the highly effective praziquantel.
- Albendazole, mebendazole, and paromomycin are less reliable.

2-Hymenolepis

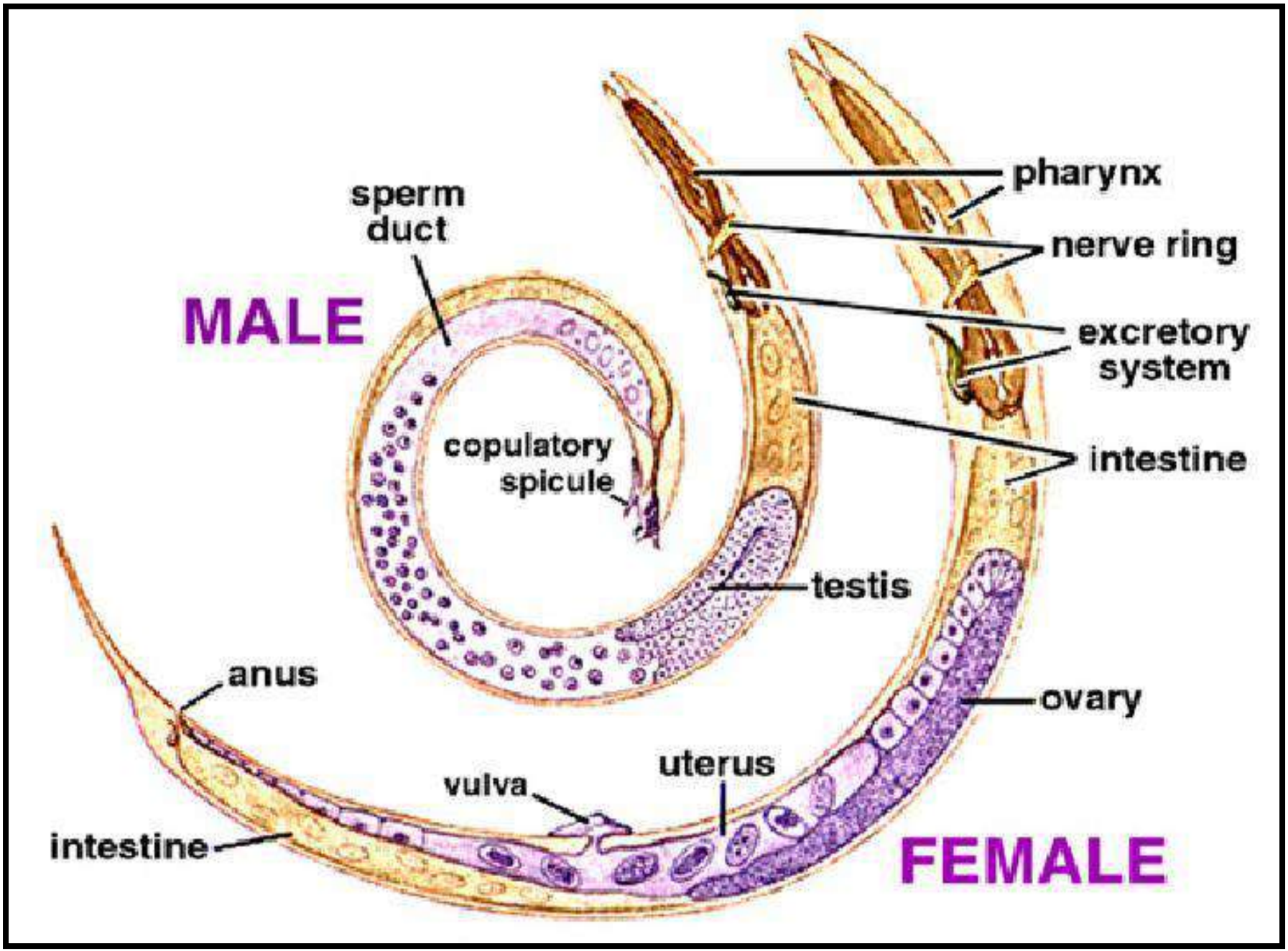
- Hymenolepis nana (Dwarf Tapeworm)
- Causative agent of hymenolepiosis
- **Occurrence**
- morphology, and life cycle.
- Hymenolepis nana, is a small intestinal parasite that occurs worldwide.
- The final hosts are rodents and humans.
- Infection results from peroral ingestion of eggs.

Therapy

- Praziquantel or albendazole are the drugs of choice.

C- Nematoda (Roundworms)

- The nematodes (nema: thread) are threadlike, nonsegmented parasites, a few mm to 1m in length, with separated sexes.
- The males are usually smaller than the females and are equipped with copulatory organs that often show features specific to each species.
- Some species require an intermediate host to complete development.


pharynx
sperm duct
nerve ring
MALE
excretory system
copulatory spicule
intestine
testis
anus
ovary
uterus
vulva
intestine
FEMALE

Intestinal Nematodes

- Ascaris lumbricoides (large roundworm), parasitize in the small intestine of humans.
- Enterobius vermicularis (pinworm) live in the large intestine.

1-Ascaris lumbricoides (Large Roundworm)

- **Causative agent of ascariosis**
- **Occurrence.**
  - The human large roundworm occurs worldwide.
  - The number of infected persons is estimated at 1.38 billion (WHO, 1998).
  - The main endemic regions, with prevalence rates of approx. 10–90%.

Therapy

- Pyrantel, mebendazole, are highly effective against the intestinal stages of Ascaris.

3-Enterobius vermicularis

- Enterobius vermicularis (Pinworm)
- Causative agent of enterobiosis (oxyuriosis)
- Parasite, life cycle, and epidemiology.

❑ **Enterobius vermicularis** which belongs to the **Oxyurida has a conspicuous white color.**

Diagnosis.

- Standard stool examination techniques are not sufficient to find the eggs.
- Egg detection by the “adhesive tape method” has proved most efficient .

*Therapy

- The following drugs are effective:
- Mebendazole & pyrantel.
- Reinfections are frequent, so that treatment usually should be repeated once or more times, extended to include all potential parasite carriers (e.g., family members, kindergarten members).

End of parasitology