A Guide To

Medical Parasitology (Gastrointestinal Module)

For 2nd Year Students

By

Staff Members of Medical Parasitology Department

Faculty of Medicine
Menoufia University
2025

Vision

To be an academically accredited college with local, regional, and international reputation for having a leading role in the field of medical education and health care provision.

Mission

The Faculty of Medicine, Menoufia University is committed to graduate a physician in accordance to the National Academic Reference Standards, who is able to meet the needs of local and regional market, skilled in conducting scientific researches that participate in developing the profession and the provided health care, and keen on continuous training and education to support the service provided to the community and surrounded environment in the frame of commitment to the ethics of the profession.

Preface

The staff members involved in teaching parasitology science welcome the 2nd year medical students to this parasitology course.

Parasitology is an important science combining the activities of physicians, pharmacists, and veterinarians.

This book **(A Guide to Medical Parasitology)** contains information that medical students of 2nd year will need during studying course of **Gastrointestinal** module.

Simple examples of parasitic life cycle for the most famous helminthic infections were illustrated.

Also, the infective and diagnostic stages, different procedures and techniques for diagnosis, specific prescribed chemotherapeutic drugs and principles of control were presented.

We would like our colleagues and students to offer their comments.

Such feedback will really enrich the subsequent editions.

The authors

Course specification

University: Menoufia

Faculty: Medicine

Course Title: Medical Parasitology

Course coordinator: Prof. Nancy Mahmoud Harba.

1- Data of the course:
Code: MFM- GIT module (2201)

Department offering the course: Parasitology Department

Programme(s) on which the course is given Bachelor's degree of medicine.

Academic year/level: second academic year.

Semester: 2nd semester of the second academic year.

Credit points: 1.5

2- Objectives of the course:
1. To supply the student with biological, epidemiological, and ecological information about the most important parasites to human.

2. To make the student understand the pathogenesis, clinical presentations, and management of these parasitic diseases.

3 – Intended learning outcomes of course (ILOs): By the end of the course, students should be able to perform:

A-Knowledge and Understanding:

a1. Describe various aspects of parasites of medical importance concerning their geographical distribution, morphology and life cycles.

a2. Discuss the pathogenesis of parasitic infections and the relation of the stage of the life cycle to pathogenesis and the clinical signs and symptoms.

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a3. Mention clinical presentations and the complications of parasitic diseases.

a4. Describe the conventional and up to date diagnostic laboratory methods to reach the accurate diagnosis of the most common parasitic diseases.

a5. Determine the effective therapeutic drugs and its doses in treating each parasitic infection.

a6. Determine the methods used for prevention and control of the most common parasites in the community.

a7. Describe the common arthropods of medical interest and explain their medical importance and the methods of combating.

B-Intellectual skills

The course aims to:

B1. Integrate basic information about life cycles, clinical picture and complications to point out the diagnostic test of choice to confirm or exclude the provisional diagnosis.

B2. Analyze theoretical information to select the most appropriate diagnosis from differential diagnosis.

B3. Diagnose and give a differential diagnosis for each parasitic disease.

C-Professional and practical skills:

c1. Draw parasites in their different stages, especially the diagnostic and infective stages, through examination of microscopic slides.

c2. Identify some parasites or their stages by naked eyes (Jars).

c3. Examine mounted slides or boxes to identify the most important arthropods of medical interest.

D-General and transferable skills

The activities done by students help them to:

d1. Use and improve their computing skills, internet search and self-learning.

d2. Apply effective communication either written or oral.

d3. Work in groups.

d4. Maintain honesty and integrity in all relations with teaching staff, colleagues and laboratory technicians.

d5. Recognize the scope and limits of their role as students and respect time factors and dates.

d6. Maintain a professional image concerning behavior, dress and speech.

Attendance criteria:

- Students should be, according to the faculty by law, attending at least 75% of the course otherwise the student will not be able to attend the exam.

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Hepatic Trematodes
Liver Flukes
(*Fasciola gigantica and Fasciola hepatica*)

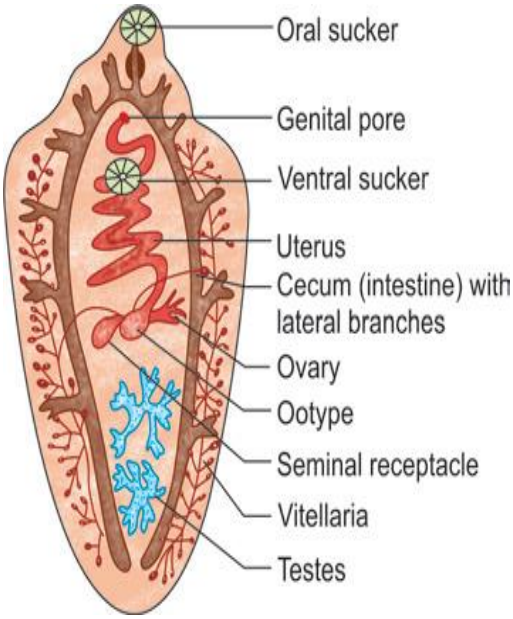
- **Geographical distribution:** worldwide, especially in tropical and subtropical countries. It was reported in 81 countries and infected 17 million people worldwide.
- **Habitat:** Bile ducts of the liver and gall bladder.
- **Definitive host:** Herbivorous animals most commonly, sheep,
- cattle, goats, camels, and buffalo. Man can be occasionally infected.
- **Intermediate host:** Snails *Lymnaea cailliaudi* and *Lymnaea truncatula.*
- **Diagnostic stage:** Immature operculated egg.
- **Infective stage:** Encysted metacercaria.

Morphology:

➢ **Adult flukes:**

• **Size:** 30 - 70 mm long by 8 - 15 mm wide.

F. hepatica is smaller.



- **Shape:** Adult flukes are flattened and leaf-like hermaphrodites (i.e., have both male and female sex organs), broader anteriorly, having an anterior cone and prominent shoulders. The worm has 2 suckers, oral and ventral. The tegument is armed with backwardly directed spines.

➢ **Egg: (Diagnostic stage)**

• **Size:** 150 x 90 um.

• **Shape**: ovoid.

• **Shell:** thin, operculated.

• **Color:** bile stained (yellowish brown).

• **Content:** embryonic cells (immature).

➢ **Encysted metacercaria (Infective stage):**

- **Size:** 0.25 mm diameter.
- **Shape:** spherical with a thick white cyst wall.

❖ **Life Cycle:**

1- In the definitive host, adult worms live in the bile ducts, lay eggs that are carried with bile to reach the intestinal lumen and passed with stool outside the body.

2- Eggs must reach water to complete the life cycle.

3- Immature eggs embryonate in fresh water and the miracidium develops and emerges from the egg to infect the snail intermediate host.

4- In the snail, the miracidium develops asexually into different stages in sequence, sporocyst, redia and finally cercaria. The cercariae are shed out of the snail, lose their tail, and encyst on aquatic vegetation as encysted metacercaria.

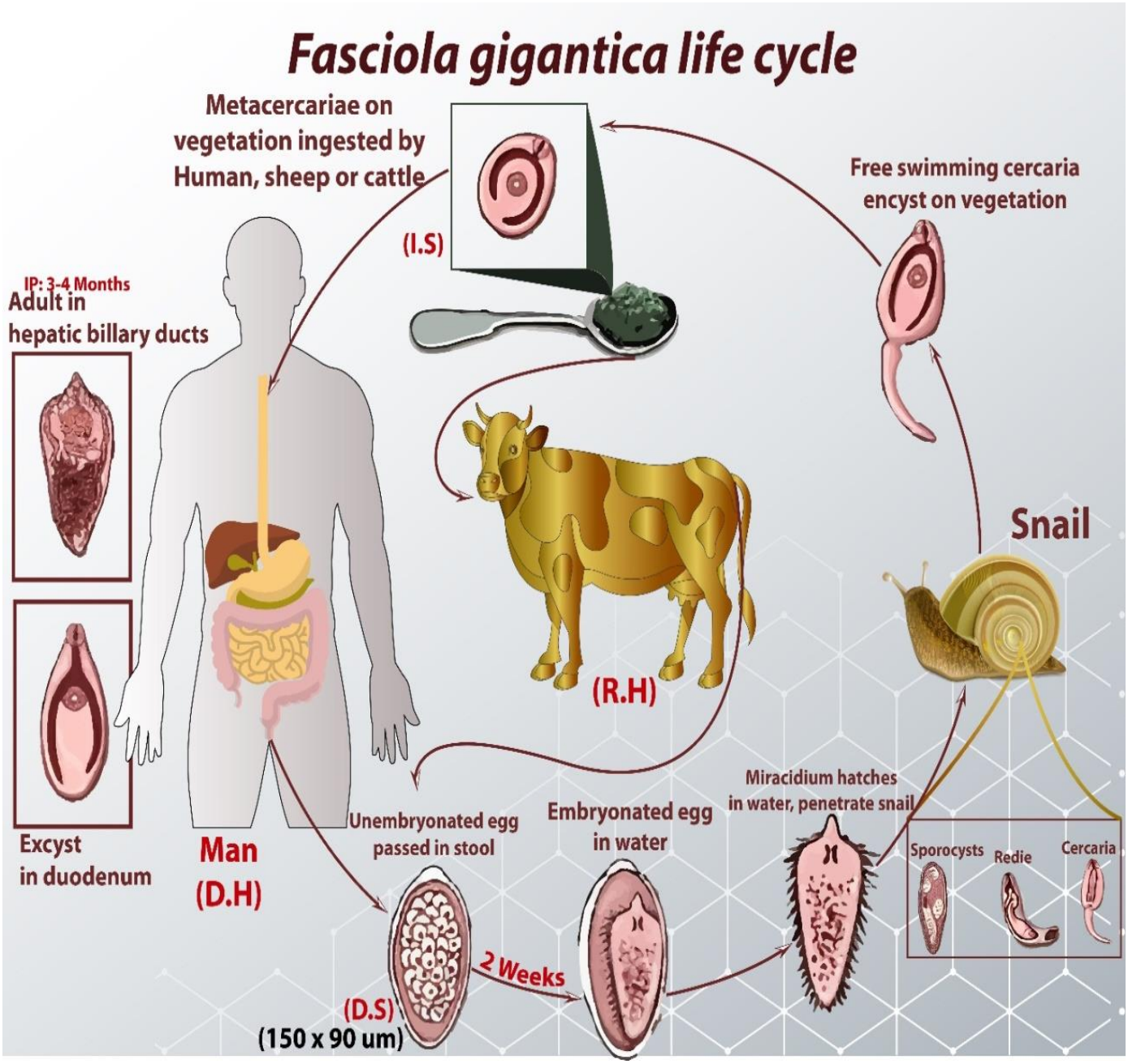
5- Encystation occurs in response to changes of environmental conditions (e.g., oxidative stress, UV light, salinity, and CO^2 concentration.

6- **Definitive hosts acquire the infection by eating contaminated vegetation with metacercaria.**

7- After ingestion, the cyst wall of metacercaria is destroyed during mastication and by the enzymatic activity of the intestinal environment.

8- The emerging juvenile flukes penetrate the intestine and migrate through the abdominal cavity and penetrate the Glisson's capsule to reach the liver.

9- In the liver, it enters its final habitat, the bile ducts, and matures into adults.

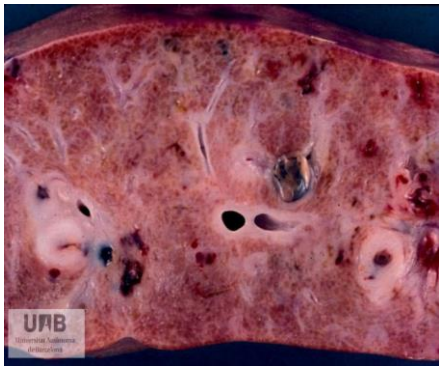


❖ **Pathogenesis:**

The pathogenesis comprises two stages, the first involves hepatic parenchyma and the second involves the bile ducts.

1- **Parenchymal phase** usually takes 2–4 months, in which inflammation, hemorrhage, necrosis, abscess formation, granulation, and fibrosis occur.

2- **Ductal phase:** Presence of adults causes excessive proliferation of the bile duct epithelium, cholangitis, and necrosis of the ductal wall resulting in biliary obstruction.



Hepatic cholangitis caused by *Faciola*

❖ **Clinical picture:**

The clinical course of human fascioliasis can be divided into two phases: acute and chronic.

1- **Acute phase:** patients may develop symptoms like prolonged febrile illness, anorexia, right upper quadrant abdominal pain, gastrointestinal disturbances, urticaria and weight loss. Ascites, hepatomegaly, splenomegaly, and anemia also occur.

2- **Chronic phase:** patients may develop symptoms of biliary obstruction such as biliary colic, epigastric pain, jaundice, and right upper quadrant abdominal tenderness.

3- In severe cases, migrating parasites may erode into blood vessels, causing huge, life-threatening subcapsular liver hematomas.

4- Young children are especially susceptible to the disastrous long-term complications associated with malnutrition and anemia such as stunting and poor neurocognitive maturation.

5- **Ectopic Infection:** is not frequent, but can occur in peritoneal cavity, intestinal wall, lungs, subcutaneous tissue.

❖ **Diagnosis:**

- **Clinical:** history and clinical manifestations
- **Laboratory:** There is no gold standard test to diagnose fascioliasis.

A. Stool examination: detecting parasitic eggs is confirmatory. However, it is of low sensitivity and not useful for diagnosis of acute infection.

B. Serological tests: for antibody and antigen detection are of value during the migratory stage of the worms and ectopic infection.

C. Eosinophilia.

D. Ultrasound and CT.

E. Molecular diagnosis: A nested-PCR was developed for detection of parasitic DNA in human stools and urine samples.

❖ **Treatment:**

1- **Biothionol:** 30 -50 mg/kg on alternate days for 10 to 15 doses.

2- **Triclabendazole:** 10 mg/kg in two doses separated for 12 to 24 hours.

3- **Nitazoxanide**: is a good alternative in case of triclabendazole failure especially in the chronic stage of infection. It is given as 500 mg twice a day for 7 days in adults.

❖ **Prevention and control:**

1- Anthelmintic Therapy:

• Prophylactic anthelminthic therapy is given to the animal host.

• Mass drug administration to decrease the prevalence of fascioliasis in human especially in high burden countries.

2- Health education.

3- Snail control.

Halzoun
(Nasopharyngeal linguatulosis)

- Definition: It is a rare allergic condition that manifests as an acute edematous reaction affecting the upper respiratory tract and nasopharyngeal mucosa after the consumption of raw or undercooked viscera (e.g., liver and lymph nodes) of sheep, goat, camel, and other herbivores.

- Distribution: mostly prevalent in Eastern Mediterranean countries. Many cases were recorded in Lebanon because raw liver is a traditional Lebanese dish that is popularly served as part of the Lebanese "Mezza".

- Causative organisms:

1- *Fasciola* adult worm: the living worm attaches to the mucosa of the pharynx by its suckers. This causes edematous congestion of the pharynx and larynx resulting in dysphagia and suffocation.

2- *Linguatula serrata* or tongue worm's nymph:

• Adult worms are found in the respiratory tracts and sinuses of the carnivorous, especially foxes, dogs, and cats.

• The embryonated eggs are expelled either with nasal secretions or in feces of the definitive host.

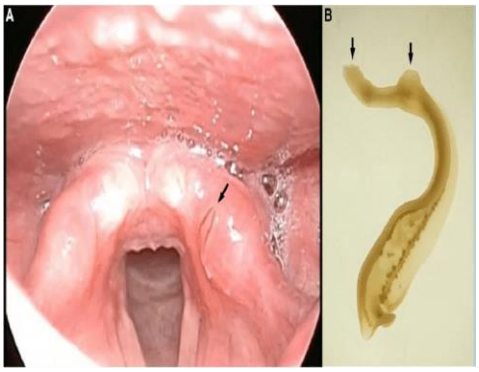
• When ingested by an intermediate host such as sheep, goats, cattle and camels, the embryos migrate to the mesenteric lymph nodes and various organs, where they feed on blood and fluids and molt to become mature nymphal stage.

• The nymph may remain alive in the intermediate host for at least two to three years, where they become encapsulated.

• **<u>Human infection occurs by eating the encysted third stage larva (nymph) in raw or undercooked liver, lymph nodes or other visceral organs.</u>**

• The nymphs cling to the soft palate, pharynx, or larynx of the human host before being swallowed or when vomited causing inflammatory reaction.

❖ **Clinical picture:**



- Manifestations are usually mistaken for other for typical allergies.
- They usually occur in the form of nasopharyngeal discomfort, coughing and sneezing.
- Other less common symptoms: pain, itching in the external auditory canals, tinnitus, hoarseness, lacrimation, coryza, hemoptysis, epistaxis, submandibular oedema, temporary hearing loss, and papular rash.

❖ **Treatment:**

1. Gargling with alcoholic drinks to lose the worm from the pharynx.
2. Giving emetic drugs.
3. Picking up of the worm by forceps.
4. Tracheostomy if suffocation occurs.

N.B: False fascioliasis (pseudo-fascioliasis): refers to the presence of *Fasciola* eggs in the stool without an actual infection but from recent ingestion of infected livers of sheep or cattle containing adult worms with eggs. It must be excluded from a real infection by repeated stool analysis one week after a liver-free diet.

▶ For more information, you can visit this website:

https://www.youtube.com/watch?v=y68MuNQRTAUhttps://www.youtube.com/watch?v=y68MuNQRTAU

1- A 40-year-old patient attended the hospital complaining of pain in the right hypochondrium, nausea, and frequent passage of loose stool. His body temperature was 38.5 ^{o}C and his skin was slightly jaundiced. Complete blood picture showed high eosinophilia. **Which of the following organisms is the most likely cause?**

a) *Ancylostoma duodenale.*
b) *Fasciola gigantica.*
c) *Heterophyes heterophyes.*
d) *Taenia saginata.*
e) *Trichuris trichiura*

2- A 44-year-old female attended to the emergency room complaining of colicky pain in her right upper quadrant with yellowish discoloration of her sclera and skin.

Which of the following parasites is suspected to be the cause of this condition?

a) *Balantidium coli.*
b) *Enterobius vermicularis.*
c) *Faciola gigantica.*
d) *Strongyloides stercoralis.*
e) *Taenia saginata.*

Intestinal cestodes
Diphyllobothrium latum
(Fish tapeworm or broad tapeworm)

- **Geographical distribution:**

  Cold-water regions e.g., Russia, Finland, and areas around Swiss, Italian, and French lakes where raw or undercooked perch is consumed. The risk of infection in non-endemic countries is rising because the culture of eating raw fish dishes is increasing (e.g., sushi, lightly pickled fish, smoked fish, gefilte fish).
- **Habitat:** Jejunum and ileum.
- **Definitive Host:** Man
- **Reservoir Host:** dogs, cats, foxes, bears, and pigs.
- **Intermediate Host:** *D. latum* passes through 2 intermediate hosts.
  - **First intermediate host:** copepods (including Cyclops and Diaptomus species).
  - **Second intermediate host:** Many cold-water fish species e.g., perch, pike, salmon, ruffe, and burbot.

Diagnostic stage: un-embryonated eggs and less common proglottids.

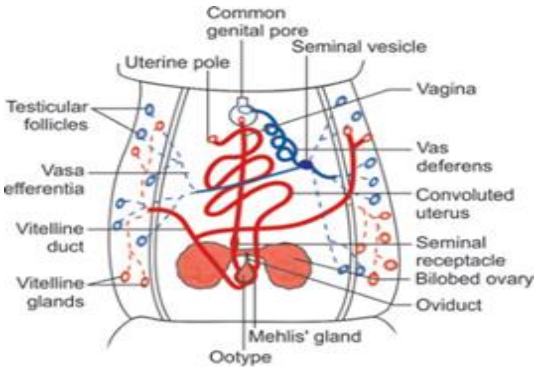
Morphology:

- **Adult worms:**

1. **Size:** from 2 to 25 meters in length (3000–4000 segments)
2. **Scolex:** almond elongated with two slit-like attachments grooves (bothrium) on the dorsal and ventral surfaces.
3. **Segments (proglottids):** have a fleshy consistency and are ivory in color (sometimes with central darkening). They are classified as follows:

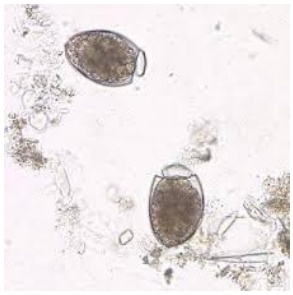
- **Neck:** A proliferative zone present posterior to the scolex.
- **Immature segments:** follow the neck with undeveloped genitals.

- **Mature segments:**



- *D. latum* is a hermaphrodite with well-developed male and female genitals in each segment.
- The proglottids are wider (12 mm) than they are long (4 mm).
- Centrally located rosette-shaped uterus with a single genital pore that opens in the middle of the ventral surface through which the mature proglottids release the eggs that are continually produced.

➢ **Egg: (diagnostic stage)**



- **Size:** range 55-75 µm long x 40-55 µm wide.
- **Shape:** ovoid.
- **Shell:** Smooth, thick with an operculum on one end and a characteristic terminal knob on the opposite end which differentiates *D. latum* from other operculated eggs e.g., Fasciola and Paragonimus.
- **Colour**: yellowish brown.
- **Content**: immature.

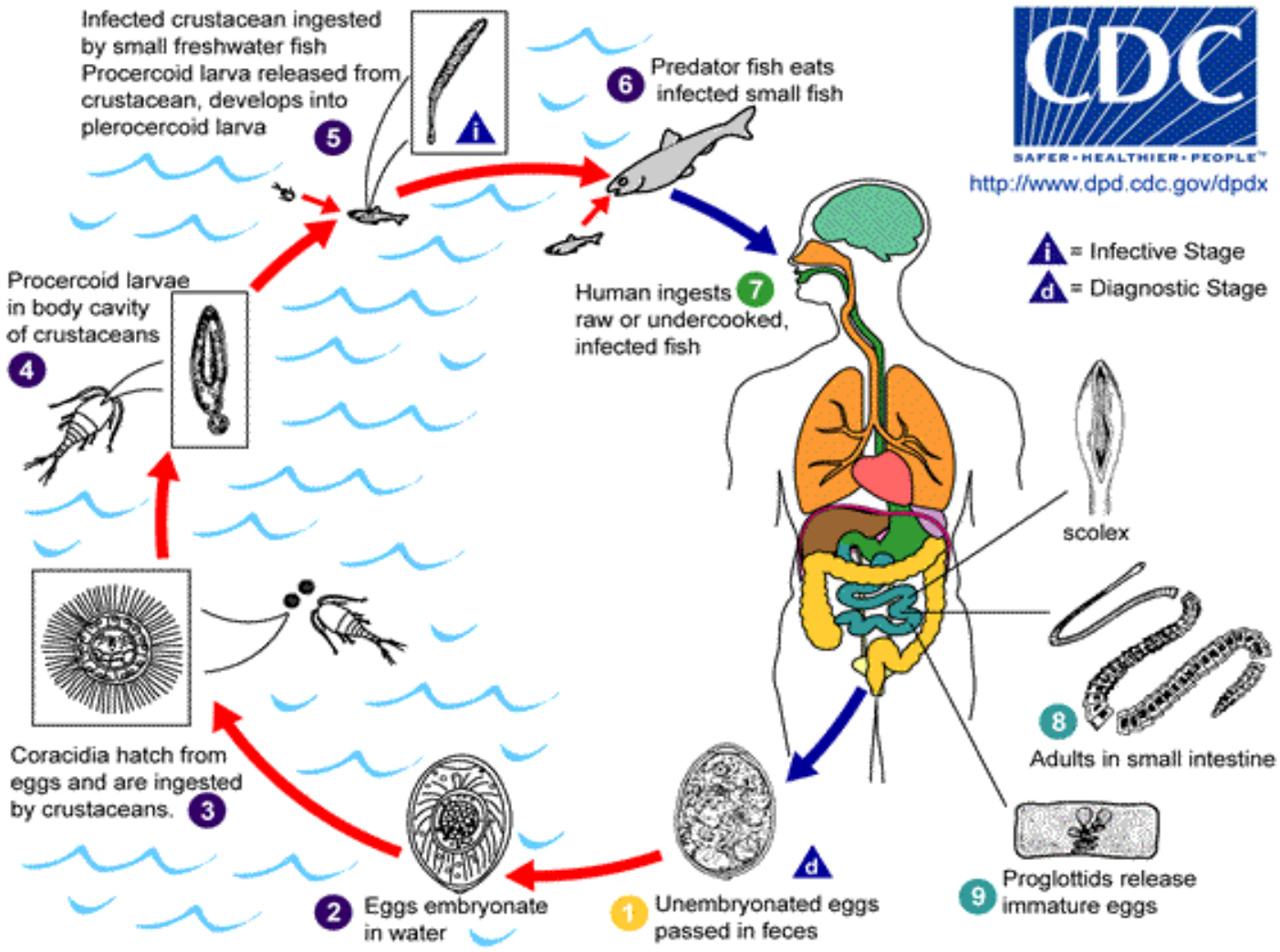
➢ **Plerocercoid larvae (spargana) (Infective stage):**

- 5–20 mm long.
- Ribbon-shaped larvae with an evaginated, bothriate scolex.
- The body of the larva shows the beginning of strobilation.

❖ **Life cycle:**

1. Adult worms live in the small intestine of man and other reservoir animals.
2. The hermaphrodite adult worm self-fertilizes, and the eggs are passed in stool. The distal strobila also break off and are passed singly or in chains during defecation.

3. Feces must reach fresh or salt water rapidly for an egg to hatch and release the free-swimming embryo (i.e., coracidium) which can infect many species of copepods.
4. Coracidium penetrates the copepod intestinal wall and develops into the procercoid larva.
5. Freshwater fish ingest the infected copepods, and procercoids enter the fish tissues and develop into the plerocercoid stage.
6. **<u>Humans are usually infected by eating raw or poorly cooked fish, (e.g., raw salted or marinated fish fillets, sushi, and sashimi).</u>**
7. In the small intestine, the scolex emerges from the plerocercoid and attaches to the intestinal mucosa and develops rapidly into adult tapeworms.



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❖ **Clinical Picture:**

Name of the Diseases: Diphyllobothriasis.

Clinical manifestations:

- Despite the large size of *D. latum* most infected cases are usually asymptomatic.
- Clinical manifestations occur in only 20% of infected patients and are usually as follows:

1. Diarrhea, intermittent digestive discomfort, and abdominal pain are the most common symptoms.
2. Other symptoms include fatigue, constipation, headache, weight loss, salt carving, and allergic reactions.
3. Pernicious anemia occurs in 2% of prolonged (3-4 years) or heavy infections because of parasite-mediated dissociation of the vitamin B12 intrinsic factor complex in the gut lumen (making vitamin B12 unavailable to the host). Also, adult worms consume more than 80% of vitamin B12 that is ingested by the host. It is often associated with low platelets or white blood cell counts.
4. Neurologic manifestations with prolonged infections of old aged patients, including glossitis, decreased vibration sense, paresthesia, and central scotoma.
5. Rare massive infections can result in intestinal obstruction.
6. Migrating segments can cause cholecystitis or cholangitis.

❖ **Diagnosis:**

- **Clinical:** history and clinical manifestations
- **Laboratory:**

1- Stool examination: for detection of un-embryonated (immature) eggs and less frequently the proglottids. Because egg discharge is periodic in intestinal diphyllobothriasis, the diagnosis can be missed by examining a single stool sample.

2- Mild eosinophilia of 5-15% can occur.

❖ **Treatment:**

1. **Praziquantel:** A single oral dose of 10-25 mg/kg.
2. **Niclosamide:** A single dose (child <10 kg, 0.5 g; child 10-35 kg, 1 g; adults, 2 g).
3. **Cobalamin injections and oral folic acid** are indicated for patients with clinical or laboratory evidence of vitamin B12 deficiency.

   N.B.

   Stool samples from infected patients who have had drug treatment should be examined. To ensure that the treatment was successful, the expected findings consist of the passage of the scolex and the absence of (new) proglottids.

❖ **Prevention and control:**

The main preventive measure is to alter food handling and eating habits because regulations for proper disposal of human feces alone have no impact on the contamination of water supplies with eggs from animal sources. These measures include:

1. Cooking fish to an internal temperature of at least 63°C.
2. Freezing fish at −35°C or below and storing at −20°C for 24 hours kills plerocercoids and renders the meat safe for consumption.
3. Canned fish is safe, but smoked fish is safe only if the fish is frozen appropriately before smoking to kill any parasites. It can be also killed by placing the fish in a concentration of brine (12% NaCl).

Taenia saginata
(Beef tapeworm, Unarmed tapeworm)

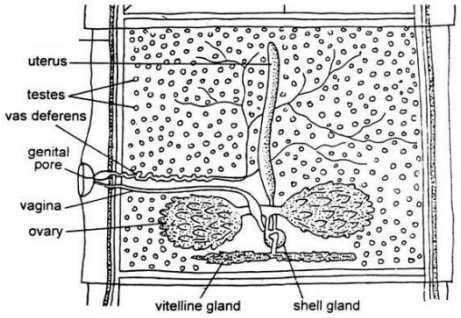
- **Geographical distribution:** worldwide with the highest prevalence recorded in Southern and Eastern Africa.
- **Habitat:** upper third of the small intestine.
- **Definitive hosts**: Man is the only definitive host.
- **Intermediate hosts**: cattle.
- **Morphology:**

- **Adult worms**:

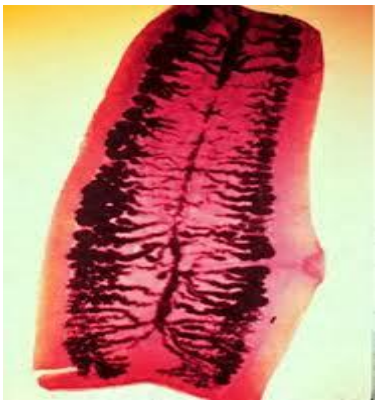
1. **Size:** 2–5 meters long. Sometimes it can reach 10 meters long.
2. **Scolex:** has 4 muscular **suckers with no hooks** followed by a neck region.
3. **Segments:** Following the neck, a chain of 1,000 to 2,000 proglottids are arranged as immature, mature and gravid segments.

- **Mature segments:**

- Broader than long.
- Each proglottid is an independent reproductive unit, as it contains both male and female genitals.
- After fertilization (that can occur within the same segment, different segments or different worms), the resulting embryos are then encapsulated in a protective keratin shell.



- **Gravid segments:**

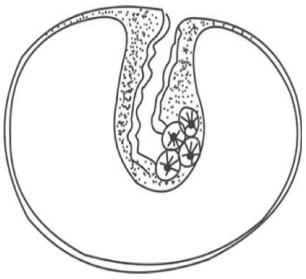


- Narrower and longer than mature segments.
- Formed after progressive enlargement of the uterus and compression of other organs of the mature segment.
- Uterine branches are between 15and 20 branches, with an average of 18.
- Gravid segments contain thousands of infective oncospheres (encapsulated embryos).
- The gravid proglottids detached from the distal end of the worm are highly motile.
- They often crawl from the anus during the day when the host is most active.
- Their **<u>independent</u>** emergence from the anus (i.e., without stool) is the principal cause of symptomatology.

➢ **Eggs: (Diagnostic stage)**

- **Size**: 28-40 µm.
- **Shape**: spherical.
- **Shell**: thick and known as an **embryophore**
- on which distinct radial striations reside.
- **Colour**: yellowish brown.
- **Content**: mature i.e., hexacanth embryo with three pairs of hooklets.

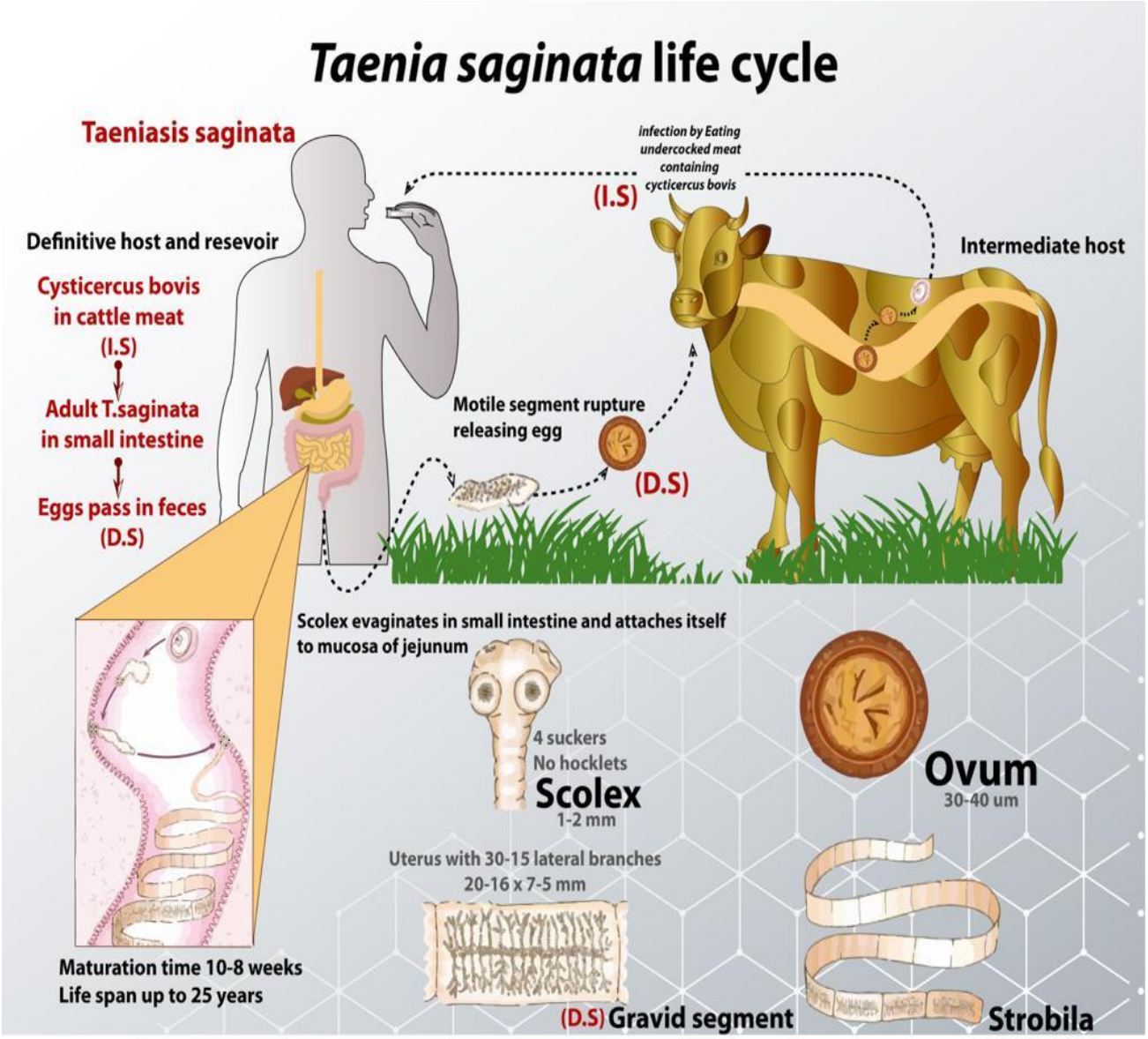
➢ **Larva (cysticerci or cysticercus *saginata*): (Infective stage)**



- **Size:** 5-10 mm in diameter
- **Shape:** translucent fluid-filled bladder with an invaginated scolex, observed as a white opaque sphere suspended within the vesicle. The bladder wall contains various cell types surrounded by loose connective tissue.

❖ **Life cycle:**

1. The adult tapeworm lives in the small intestine of humans.
2. 10 to 12 weeks after infection, the tapeworm begins to eliminate gravid proglottids which are shed in stool.
3. When infective eggs are ingested by cattle in contaminated vegetations or water, the oncospheres are liberated, penetrate the intestinal wall, and are carried with the circulation to various tissues especially muscles, mostly the heart and masseter muscles, where they develop into larvae or cysticerci.
4. **Humans are infected by ingesting raw or undercooked beef infected with cysticercus *saginata*.**
5. The larvae evaginate in the small intestine of the host by the effect of the digestive juices and bile.
6. The evaginated larvae attach to the intestinal wall and burrow through the intestinal villi with its unarmed rostellum, while simultaneously anchoring to neighboring villi by all four suckers.



❖ **Clinical picture:**

Disease: Taeniasis *saginata*, Beef tapeworm disease

1. Asymptomatic: Most infected
   patients typically remain asymptomatic.
2. Symptoms of irritable bowel, particularly abdominal pain. Also, nausea, distension and anorexia, diarrhea, or constipation.
3. The most specific complaint is the discomfort and anal pruritus caused by the proglottids independently crawling out of the anus.

❖ **Diagnosis:**

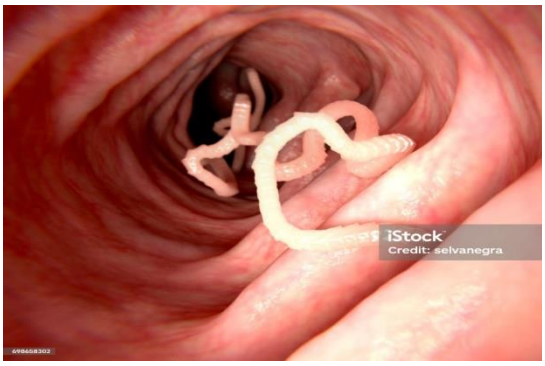
- **Clinical:** history and clinical manifestations
- **Laboratory:**

  1- Stool examination for egg detection.

  2- The perianal swab method may also be effective to detect proglottids.

  3- Antigen detection by ELISA.

  4- Preparation of specific DNA probes for the detection of *Taenia* eggs in stool samples.



❖ **Treatment:**

1. **Praziquantel:** 10 mg/kg as single dose.
2. **Niclosamide:** adults and children over 6 years: 2 g. Children aged 2–6 years: 1 g in a single dose.
3. **Albendazole:** 400 mg for 3 consecutive days.

❖ **Prevention and control:**

1. Beef inspection for the presence of cysticerci is the best preventive measure.
2. Beef must be thoroughly cooked, especially in areas of endemicity, to at least 56 °C internal temperature.
3. Freezing at –10 °C for 10 days is usually lethal to *Taenia* cysticerci, but they can withstand 70 days at 0 °C.
4. Proper sanitation practices.
5. Proper disposal of human feces whenever feasible.
6. Preventing irrigation of vegetables with water contaminated with human feces.
7. Treatment of infected persons.
8. Health education.

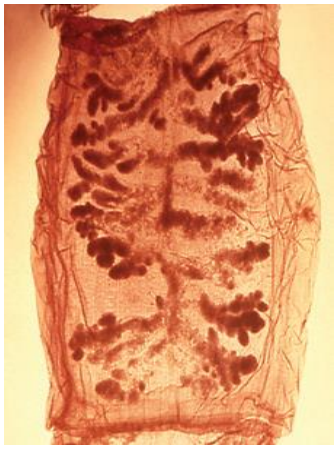
Taenia solium
(Pork tapeworm, armed tapeworm)

- **Geographical distribution:** Worldwide. It is endemic in many countries of Central and South America, South Africa, and Asia.
- **Habitat**: upper third of small intestine.
- **Definitive host:** Humans.
- **Intermediate host**: Pigs in taeniasis and man in cysticercosis.
- **Morphology:**

➢ **Adult worms:**

- **Size:** smaller than *T. saginata* (2-3 meters).
- **Scolex:** has four large suckers with **a rostellum carrying two rows of hooks**.
- **Segments:**

- The total number of proglottids is **less than 1,000**.
- The immature proglottids are wider than long.
- The mature proglottids are approximately square.
- The gravid segments' number of lateral uterine branches are between **7 and 13 branches.**
- Detached proglottids are much **less motile** and consequently less likely to be noticed than those of *T. saginata*.

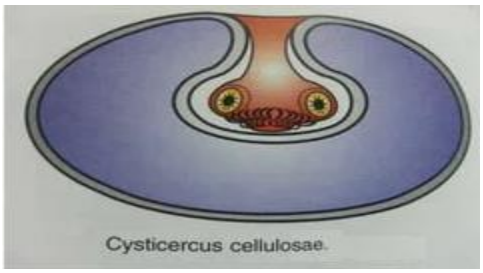


➢ **Eggs (Diagnostic and infective stage)**:

The eggs of *T. solium* and *T. Saginata* are indistinguishable by standard parasitology procedure.

➢ **The larva (cysticerci, cysticercus *solium*): (Infective stage):**

Similar to *T. Saginata* but the scolex is hooked.



❖ **Life cycle:**

Similar to *T. Saginata* except for

1. Pig is the intermediate host.
2. Eggs of *T. solium* are infective to humans and if ingested (either by autoinfection or external infection) cysticercosis can occur where man will be the intermediate host.

❖ **Clinical picture:**

Common associated diseases:

1. Taeniasis *solium* (pork tapeworm infection).

2. Cysticercosis.

1) Taeniasis *solium*:

- Infection is most commonly asymptomatic or causes mild disease.
- Children are often more symptomatic than adults.

1. Anal pruritus caused by the escaping proglottids. Most patients become acutely aware of the infection by noting the passage of proglottids in their feces.
2. Other symptoms include nausea, change in appetite (both increase and decrease have been reported), weakness, and weight loss.
3. Less commonly headache, constipation, dizziness, and diarrhea also occur.

2) Cysticercosis:

- Can affect any organ of the body. The presence of cysticerci in the brain represents the most frequent parasitic infection of the human nervous system and the most common cause of adult-onset epilepsy throughout the world.
- Symptoms depend on the site involved. Manifestations appear as malfunction of the affected organ.

❖ **Diagnosis:** similar to *T. saginata*.

1. The eggs of *T. solium* and *T. Saginata* can be distinguished by Ziehl-Neelsen staining or using molecular techniques.
2. The species can be also distinguished by a passed gravid proglottid based on the number of uterine branches.

❖ **Treatment**

- Similar to *T. saginata* although a purgative should be used after niclosamide treatment because it causes disintegration of the worm in the small intestine. This increases the risk of autoinfection.

❖ **Prevention and control:** similar to *T. saginata.*

▶ For more information, visit this website:

https://www.youtube.com/watch?v=2SLhWgHEIc8

▶ The main differences between *T. saginata* and *T. solium*

Characteristic	***T. saginata***	***T. solium***
Common name	Beef tape worm	Pork tapeworm
Disease	Taeniasis saginata	Taeniasis *solium*. Cysticercosis.
Morphology: **1)Adult Scolex** Number of Suckers	Four	Four
Rostellum	Absent	Present
Hooks	Absent	Present
Gravid Proglottids Shape	Longer than wide.	Square
Number of lateral branches on each side of uterus	15-30	7-15
2) Egg	The same morphology	The same morphology
3) Cysticerci	-cysticercus *saginata* -Scolex without hooks	-cysticercus *solium* -Scolex with hooks
Definitive host	Man	Man
Intermediate host	Cattle	Pig, may be man
Mode of infection	-ingesting raw or undercooked beef infected with larvae	-ingesting raw or undercooked pork infected with larvae - ingestion of *T. solium* eggs -Autoinfection
Infective stage	cysticercus *saginata*	-cysticercus *solium* -egg
Diagnostic stage	-Egg -Gravid segment -Mature segment	-Egg -Gravid segment -Mature segment

1- A 5-year-old girl attended with her mother complaining of perianal itching. Stool examination revealed rounded eggs with thick striated wall. **Which of the following is the causative organism?**

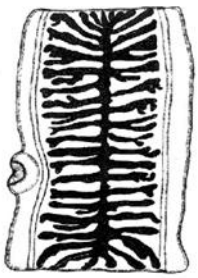
a. *Ancylostoma duodenale.*
b. *Ascaris lumbricoides.*
c. *Enterobius vermicularis.*
d. *Strongyloides stercoralis.*
e. *Taenia saginata.*

2. **Which of the following animals represents a source of infection to man regarding the shown parasite?**

Cats.
Cattle.
Dogs.
Migrating birds.
Pigs.

3. A 45-year-old male patient attended the clinic of internal medicine complaining of abdominal colic with nausea and vomiting. His stool examination revealed the shown structures.

Which of the following organisms will be your diagnosis?



Dipylidium caninum.
Hymenolepis diminuta.
Hymenolepis nana.
Taenia saginata.
Taenia solium.

Hookworms

Species:

A. The two most common types of hookworms that infect humans are:

1. *Ancylostoma duodenale:* known as the old-world hookworm.
2. *Necator americanus*: known as the New World hookworm or the American killer.

Ancylostoma duodenale
(Old World hookworm)

- **Geographical distribution:** It is common in tropical, subtropical, and temperate regions.
- **Habita**t: small intestines, mostly the jejunum
- **Definitive Hos**t: Man (the only natural host).
- I**ntermediate Host**: No intermediate host.
- **Diagnostic stage** :immature eggs.
- **Infective stage**: Third stage filariform larva.
- **Morphology:**

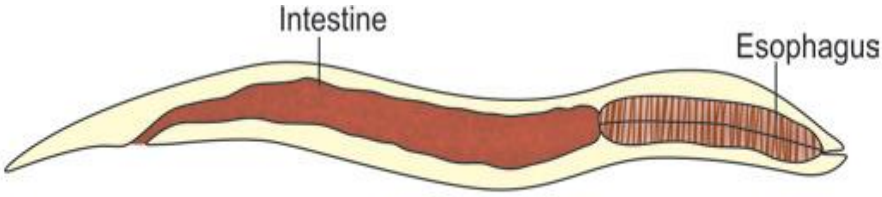
➢ **Adult worms**

- Small cylindrical worms with a cup shaped toothed buccal capsule which carries a pair of glands that secrete anticoagulant peptides into the worm's buccal cavity, esophagus, and the attachment site.

➢ **Eggs (diagnostic stage):**

- **Size:** 60 x 40 µm.
- **Shape:** Oval.
- **Shell:** Thin. There is a clear space between the segmented ovum and eggshell.
- **Color:** translucent.
- **Content:** Freshly laid eggs contain a developing embryo in the early stages of cleavage (4-8 cells).

➢ **The 3rd stage filariform larva (infective stage).**



- 500—700 µm long.
- Sheathed.
- Sharply pointed tail.
- Ratio between the length of esophagus to intestine: about 1:2.

Life cycle

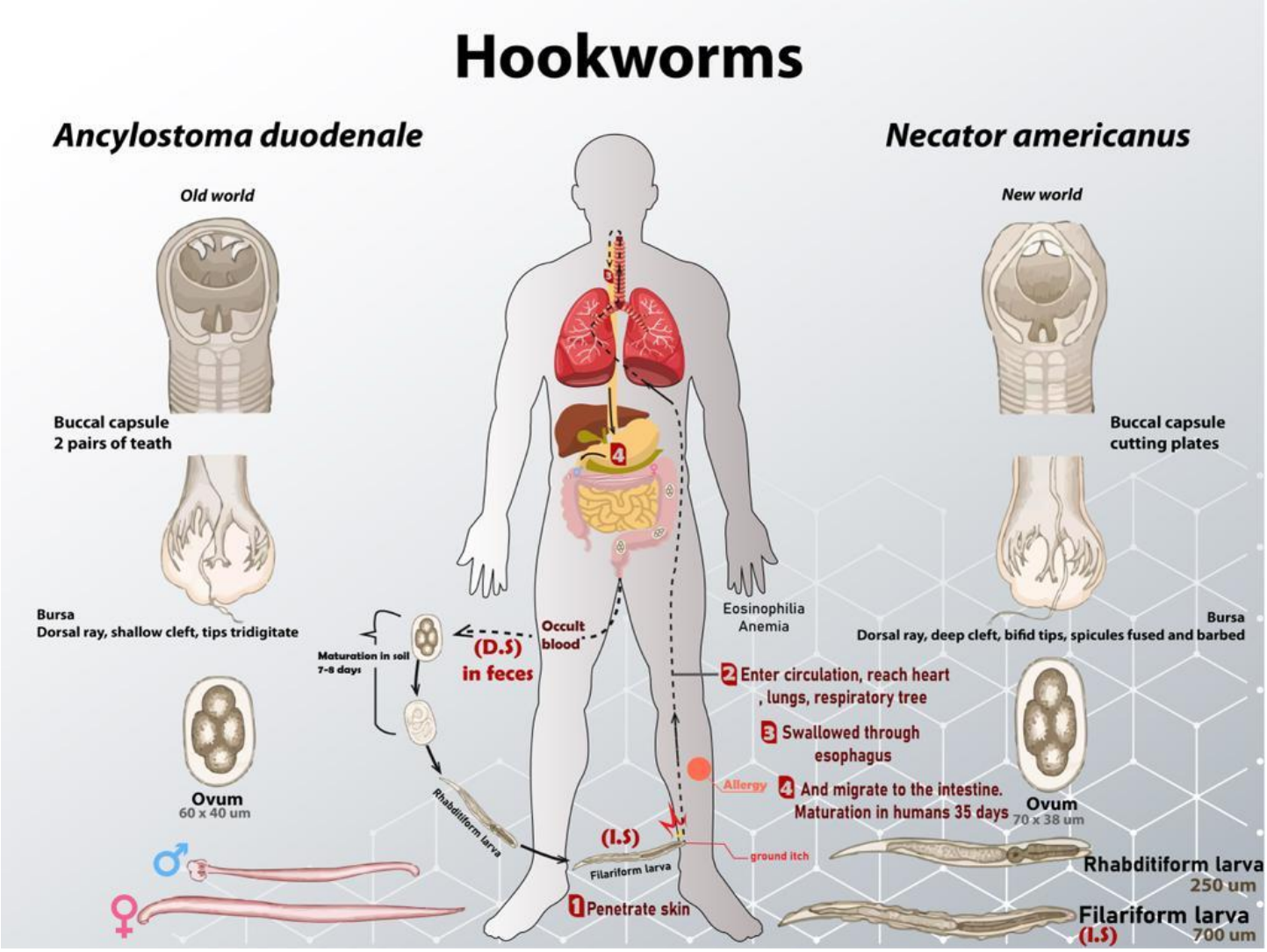
1. Adult worms live in the small intestine of man. They attach themselves to the intestinal mucosa using their buccal capsule.
2. After fertilization, the female worm lays eggs which are passed out in feces of infected persons.
3. In the soil, under favorable environmental conditions (moisture, warmth, and shade), the embryo develops inside the deposited eggs and rhabditiform larvae hatch, feed on bacteria and other organic matter.

4. After 5 to 10 days, they moult twice to become the third-stage infective filariform larvae.
5. **Man can be infected by:**

a. Active penetration of 3rd stage filariform larvae to his exposed skin (especially hands, feet, arms, and legs). Larvae usually gain access through hair follicles or abraded skin.

b. Oral route, where the filariform larva, contaminating vegetables or fruits, penetrates the buccal mucosa to reach the venous circulation.

6. Following skin penetration, the larvae enter subcutaneous venules and lymphatics, migrate to the right side of the heart and to the lungs.
7. Inside the lungs, they break through the pulmonary capillaries to reach the alveoli, where the third moult occurs, ascend along the bronchial tree to the trachea, traverse the epiglottis to the oesophagus and are swallowed to reach intestine.
8. In heavy infections, not all the filariform larvae are kept inside the lung. Part of them can reach the left side of the heart to be distributed with the general circulation to many of human viscera **(visceral larva migrans**).
9. The larvae which reach the small intestine undergoes the final moult to form adults which mature and mate.



❖ Pathogenesis and Clinical Picture:

Pathogenicity is extensive involving the skin, lungs, and small intestine. Intensity depends on the number of worms in the intestine (worm burden) and the nutritional state of the patient.

❖ **Disease: Ancylostomiasis.**

Some people can develop an *Ancylostoma* infection without any symptoms.

1. **The invasion stage**:

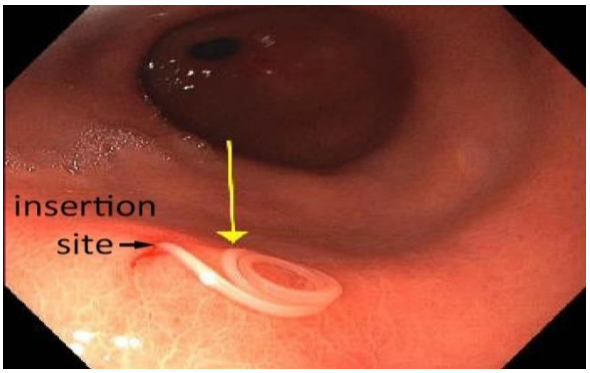
The filariform larvae penetration of skin results in hypersensitivity reaction which manifests as local irritation, intense itchiness, and vesicular erythematous rash lesions. These are followed by linear serpiginous, and elevated ridges due to the larvae tunneling phenomenon and vesicular rash lesions that are called **ground itch or creeping eruption**.

2. **The pulmonary stage**

a. Larval migration through lung tissue causes a pulmonary hypersensitive response called **verminous pneumonia (eosinophilic pneumonia or Loeffler's syndrome)** which presents clinically in the form of cough, sneezing, bronchitis, and hemoptysis.

b. Visceral larva migrans: can occur in heavy infections.

3. **The intestinal stage:**



The adult worms cause mechanical injury to the mucosa by their buccal capsule and teeth during their blood meal.

a. A light hookworm infection causes abdominal pain, loss of appetite, and geophagy (pica, urge to eat soil).

b. Heavy infections occur most often in children and include dyspepsia, nausea, epigastric distress, acute enteritis with uncontrollable diarrhea and foul stools.

c. Chronic infection results in:

- Hypoproteinemia: which leads to

- Malnutrition with physical and cognitive impairment due to protein deficiency.
- Anasarca (oedema of the face and lower limbs) and abdominal distension from ascites.

  N.B. The skin of infected individuals acquires a sickly yellowish color, sometimes referred to as 'chlorosis' The combination of anemia and protein losses, together with chlorosis, is a reason why, chronic hookworm infection was sometimes known as the 'yellow puffy disease.

- Iron deficiency anemia due to blood loss

The mechanisms by which hookworms induce blood loss are multifactorial:

1. Actively feeding adult worms.
2. Leakage around the attachment site of the hookworm in the gut of the host.
3. The secretion of parasite-derived anticoagulants,

❖ **Diagnosis:**

- **History and clinical picture.**
- **Laboratory:**

A. Direct methods:

1. **Stool examination:** to detect eggs.
2. **Stool culture**: to detect 3rd stage filariform larva.
3. **Baermann's technique** for examination larvae in soil or stool.

B. Indirect methods

1. **DNA-based tools** for diagnosis of infection could serve as a molecular approach for accurate diagnosis of hookworm in the feces.

❖ **Treatment:**

A. Antihelminthic treatment.

1. **Albendazole:** 400 mg orally as a single dose.

2. **Mebendazole:** 100 mg orally twice a day for 3 days or 500 mg as a single dose.
3. **Pyrantel pamoate:** 11 mg/kg (maximum dose of 1 g) orally once a day for 3 days.
4. **Levamisole:** 4 mg/kg body weight as a single dose.

B. Symptomatic treatment.

1. Iron supplements: should be administered with mild or moderate anemia and continued for about 3 months to replenish depleted iron stores.
2. Severe anemia may require blood transfusions.
3. High protein diet and vitamins.

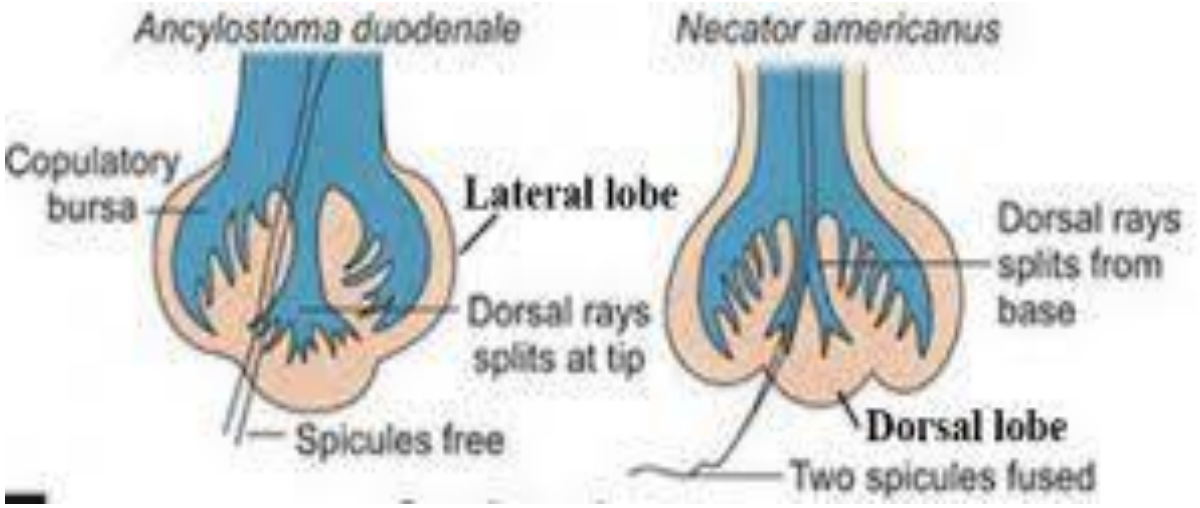
❖ **Prevention and control:**

1. Treatment of patients and carriers.
2. Sanitation and proper use of sanitary latrines.
3. Wear shoes, gloves and avoid walking bare footed.
4. Avoid using fertilizer made from human feces.
5. Hygiene measures such as hand washing and avoid defecation on the ground.

Necator americanus
(New world hookworm or American hookworm)

▶ Differences between *A. duodenale* and *Necator americanus:*

Characteristics of adult worm	**Ancylostoma Duodenales**	**Necator americanus**
Distribution	North Africa specially Egypt, Europe, Middle & Far East	Central and South Africa, Central and South America & Far East
Common name	Old world hookworm.	New world hookworm.
Size	Larger and thick Female 12mm Male 10mm	Shorter and more slender Female – 10 mm Male- 5-6 mm
Shape	Single curve looks like 'C'	Double curve looks like 'S'
Anterior end	Bends in the same direction as body curvature	Bends in the opposite direction to the body curvature
Buccal capsule	Larger and oval. 6 teeth, 4 hook like teeth on the ventral surface. 2 knob-like teeth on dorsal surface	Small and round. 4 cutting chitinous plates- 2 on ventral surface and 2 on dorsal surface.
Copulating bursa	Circle in shape. Broader than long & trilobed. Dorsal ray is single, bifurcated at the tip, bifurcation is tripartite. The total number of rays is 13.	Oval in shape. Longer than broad & bilobed. Dorsal ray is split from base. Each bifurcation is bipartite. Total no of rays 14.
Vulva position	opens at junction of middle and posterior 1/3 Copulation Y-shaped	In front the middle of the body Copulation T-shaped
Daily egg output	20.000 eggs/ female	10.000 eggs/female
Pathogenesis	blood loss by feeding worm is higher (0.5 cc of blood daily/parasite)	blood loss is lower (single worm can consume 0.03 cc of blood/ day)
Life span	live on average 1–3 years	live 3–10 years



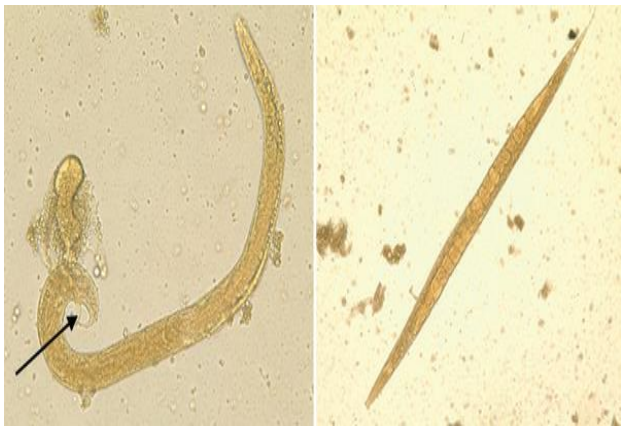
Strongyloides stercoralis
(Dwarf thread worm)

- **Geographical distribution:** Mainly in Tropical & subtropical regions especially in areas of low sanitation and poor hygienic measures.
- **Habitat:** The parasitic female lives in the human small intestine (duodenum and jejunum) while the free-living female and male worms freely multiply in the environment.
- **Definitive host:** Man, dogs, and cats.
- **Diagnostic stage:** Rhabditiform larva.
- **Infective stage:** 3rd stage filariform larva.

Morphology

➢ **Adult worms:**

- Cylindrical thread-like worm.
- The size of parasitic females is larger than the free-living ones.
- The size of both free-living and



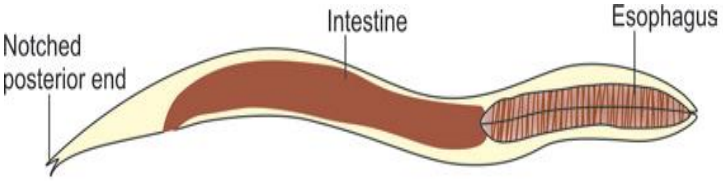
➢ **Eggs:**

- **Size:** 50–70 x 30 µm.
- **Shape:** oval.
- **Shell:** thin.
- **Color:** translucent.
- **Contents**: mature (larva).

➢ **Rhabditiform larva (L1) [diagnostic stage]:**

- **Size:** 200-250 µm long.
- **Shape:** short buccal cavity, a double bulb esophagus and large genital primordium.

➢ **Third stage filariform larva (L3) [infective stage]:**



- **Size:** 630 µm long.
- **Shape:** long cylindrical esophagus and a notched tail.

❖ **Life Cycle:**

Strongyloides stercoralis is a unique parasite as it can develop by a direct parasitic life cycle or an indirect free-living life cycle by alternation of generations.

1. Parasitic females are embedded by its anterior ends into the submucosa of small intestine.
2. Eggs are produced and hatch inside the small intestine.
3. Larvae pass with stool to the soil and moult to form the 3rd stage infective filariform larva.
4. The behavior of the larvae is determined according to the environmental conditions.

- **Indirect development:** in favorable environmental conditions, the soil L3 completes its life cycle without a host. They moult twice to develop into adults which lay their eggs in the soil to hatch larvae that moult into adults and so on.
- **Direct development**: In unfavorable environmental conditions the larvae starve and try to find a host to live.

5. **Humans can be infected by:**

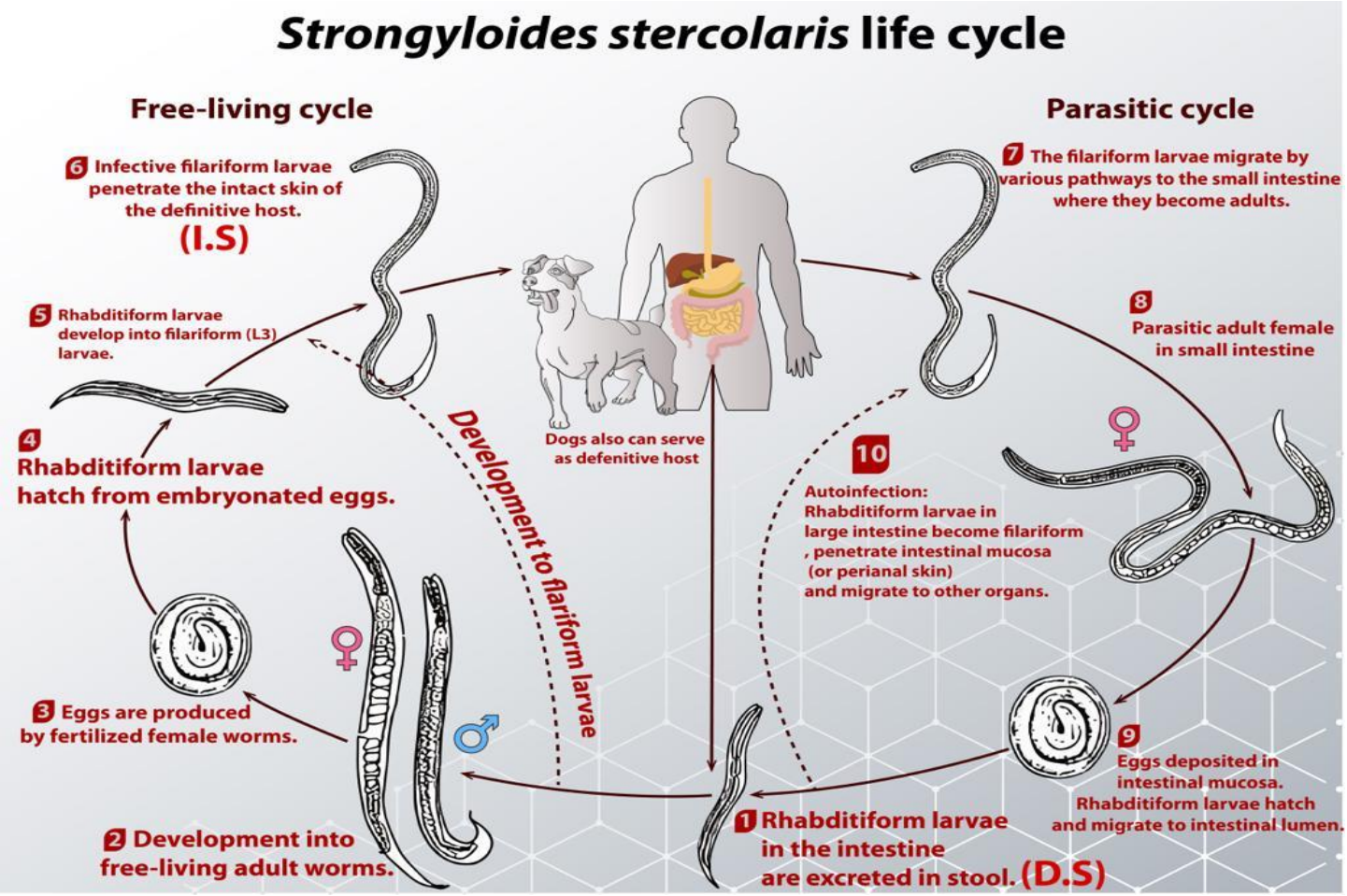
a. **Penetration of skin by L3 during walking bare footed.**

b. **Penetration of mucous membrane of the mouth by eating food contaminated with L3.**

c. **Autoinfection: Some L1 larvae don't pass with feces but they develop into L3, penetrate the intestinal wall (internal autoinfection) or perianal skin (external autoinfection).**

d. **Hyper-infection: can occur in patients receiving corticosteroids which increase the moulting rate of L1 larvae in the gut.**

6. L3 find their way to the circulation and are carried by blood to lungs → pass into alveoli→moult→ascends to trachea → pharynx → esophagus → small intestine.
7. In the small intestine, the filariform larvae undergo their final moult to form male and female adults, which mate and lay eggs.



❖ **Clinical picture:**

- *Strongyloides stercoralis* is an opportunistic parasite. As long as there is an intact immune system, the host can control the parasitic burden, and the organism may persist for years at the initial site of entry. The manifestations usually appear in immunocompromised patients.
- The disease has cutaneous, pulmonary, and intestinal phases.

1. **The cutaneous phase:**

- **Ground itch:** At the site of entry of infective larva→ congestion, hemorrhage, swelling, itching, edema, maculopapular or urticarial rashes usually on the feet. This may spread to the buttocks and waist areas.

- **Larva currens:** linear, pathognomonic serpiginous urticarial rash in the perianal region (**Cutaneous larva migrans**).

2. **The pulmonary phase:**

- Due to larval migration in lungs.
- Manifests by bronchopneumonia with cough, dyspnea, and wheezes.
- Larva is found in sputum.

3. **The intestinal phase:**

- Female worms become embedded in the mucosa or submucosa leading to epigastric pain, diarrhea with blood & mucus, nausea and weight loss, malabsorption of fat, proteins, and vitamin B12.
- **Hyperinfection** can occur due to steroids, immunosuppressive therapy, malignancy, hypochlorhydria, diabetes and AIDS. It leads to colitis, malabsorption, ulcerative enteritis, and paralysis of small intestine (**paralytic ileus**).
- **Disseminated strongyloidiasis** where migrating larvae invade GIT and migrate to CNS, lungs, heart, and kidneys. Passage of enteric flora with larvae can lead to Gram-negative sepsis with subsequent pneumonia, meningitis, CNS invasion and brain abscess→high mortality.

❖ Diagnosis

- **History and clinical picture.**
- **Laboratory:**

1. **Stool examination:** for detection of rhabditiform or filariform larvae in stool.
2. **Culture of stool samples**: nutrient agar is the most effective in larvae isolation (method of choice).
3. **Baermann funnel technique, Charcoal culture method.**
4. **Sputum** should be examined for larvae with wet mounts.

5. **Serological diagnosis** can be used to follow-up the efficacy of treatment since there is a fall in parasite specific IgG.
6. Diagnosis of strongyloidiasis in stool using **coproantigen** has excellent specificity.
7. **Duodenal aspiration (Entero-test)** is a very accurate technique in hyper-infection because the larvae passed in feces may decrease.
8. **Molecular diagnosis:** PCR is 100% specific and of high sensitivity.
9. **Eosinophilia** 10 – 40%

❖ Treatment:

1. **Ivermectin:** 200 mg/kg single dose.
2. **Thiabendazole:** 25 mg/kg twice daily for 3 days.
3. **Albendazole**: 400 mg twice daily for 3 days.
4. **Mebendazole:** 100 mg twice daily for 3 days.

   N.B. Treatment should be repeated after a week to avoid relapse of the intestinal phase, especially in immunocompromised patients.

❖ Prevention and Control:

1. Personal prophylaxis: wearing shoes to protect skin from contact with contaminated soil (farmers).
2. Health education.
3. Avoid defecation in soil.
4. Sanitary disposal of human feces.
5. Avoid use of human feces as fertilizers or disinfection before use.
6. Mass treatment of patients.
7. High-risk groups, such as immunosuppressed, farmers, immigrants from endemic areas, and mentally handicapped should be examined periodically and treated.

▶ For more information, visit this website:

https://www.youtube.com/watch?v=iQMwziCLyCs

Trichostrongylus colubriformis
(Snake-like worm)

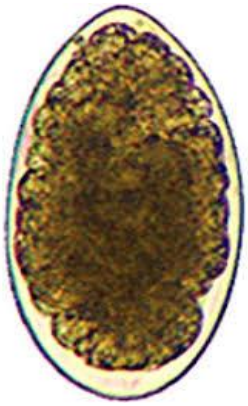
- **Name:** Greek: *tricho* = fine hair; *strongylos* = rounded; *colubriformis* = snake-like.
- **Distribution:** Worldwide, more common in agriculture communities.
- **Habitat:** small intestine.
- **Definitive host:** Man (infected occasionally).
- **Reservoir host:** All plant-feeding vertebrates except for fish. Most common reservoir hosts are the large herbivorous animals e.g., sheep and cattle.
- **Diagnostic stage:** Immature eggs in stool.
- **Infective stage:** The 3rd stage filariform larva.

❖ **Morphology:**

➢ **Adult worms:**

- Thread like nematode.
- Thin anterior part and thick posterior part.
- Rudimentary buccal capsule.
- Sex is separate.

➢ **Egg (diagnostic stage):**



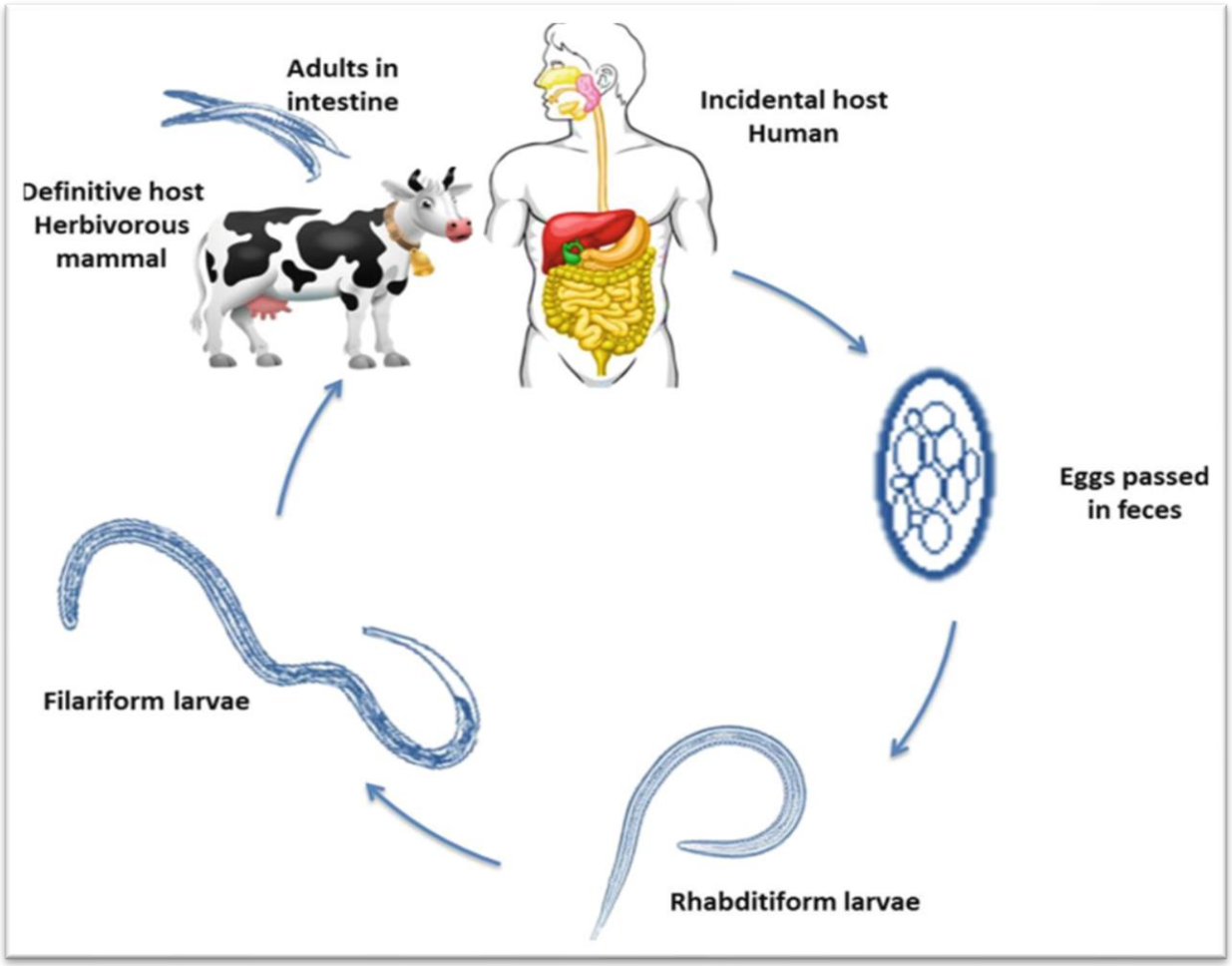
- **Size:** 90 x 40 µm.
- **Shape:** Oval, one pole is pointed and the other is rounded (shape of hen's egg).
- **Shell:** Thin.
- **Color:** Translucent.
- **Content:** Immature (morula stage, 32-cell stage).

➢ **Filariform larvae (infective stage)**:

- 600-700 µm.
- Cylindrical (filariform) oesophagus (1/4 of the body).
- Sheathed.
- Posterior end has a knob.

❖ **Life cycle:**

1. Adult worms live in the small intestine of the herbivorous animals and occasionally man with their anterior ends embedded in the intestinal submucosa.
2. Immature eggs pass out with feces.
3. Under favorable environmental conditions, the egg matures and hatches into 1st stage rhabditiform larva.
4. The 1st stage rhabditiform larva moults twice to develop 2nd stage rhabditiform larva then 3rd stage infective filariform larva.
5. **Man is infected by ingestion of 3rd stage filariform larva with vegetables and water.**
6. In the small intestine, the larva moults twice to develop into adult.



❖ **Clinical picture:**

1- Light infection: Asymptomatic.

2- Heavy infection: Intestinal disturbance, abdominal pain, anorexia, and diarrhea.

❖ **Diagnosis:**

- **Clinical:** history and clinical manifestations.
- **Laboratory:**

1. Stool examination: for detection of immature eggs.
2. Stool culture: for detection of larvae: Rhabditiform larvae take 1-2 days and filariform larvae take 3-4 days to develop.

❖ **Treatment:**

1. **Thiabendazole:** 25 mg/kg twice daily for 3 days.
2. Mebendazole, albendazole, and ivermectin are also effective.

❖ **Prevention and Control**

1. Health education.
2. Treatment of patients and reservoir hosts.
3. Proper washing of vegetables.
4. Pure water supply.
5. Avoid defecation in the soil.
6. Sanitary sewage disposal.
7. Avoid using human stools as manure.

Capillaria philippinensis

- **Geographical distribution**: most infections occur in the Philippines and Thailand. Some cases were recorded in Egypt.
- **Habitat**: Small intestine especially jejunum.
- **Definitive Host:** Fish eating birds and man.
- **Intermediate Host**: Freshwater fish tilapia, milkfish, and carp.
- **Morphology:**

➢ **Adult worm**

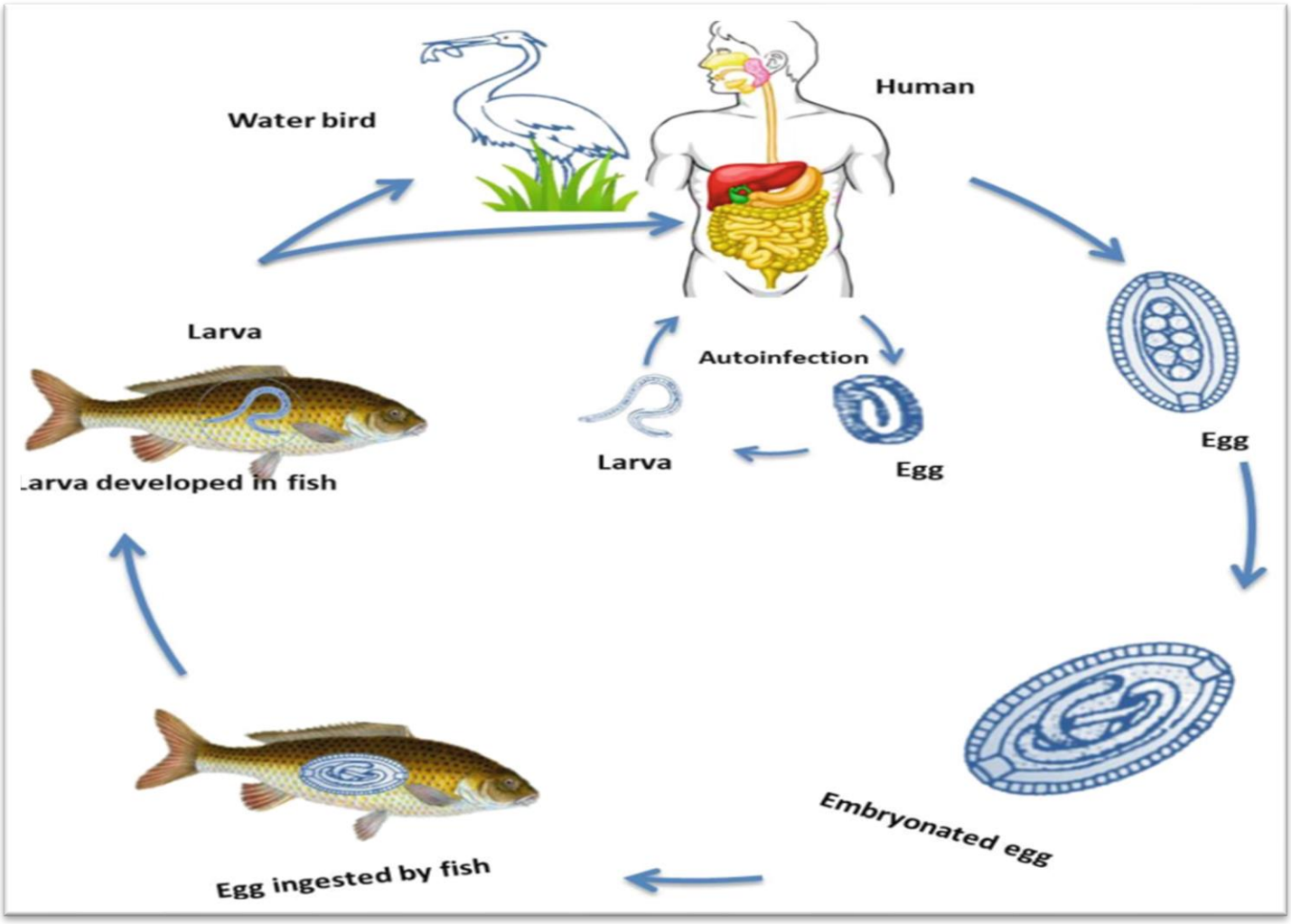
- **Shape:** filamentous thin anterior end and a slightly thicker posterior end.
- Sex is separate.

➢ **Egg:**

- **Size:** 45×20 um.
- **Shape:** peanut shaped.
- **Shell:** thick striated shell with flattened bipolar plugs.
- **Color:** Yellowish.
- **Content:** the female produces two types of eggs immature and mature eggs containing larva.

➢ **Life cycle**:

1. The adult worms live in the small intestine of man and fish-eating birds.
2. The female produces two types of eggs un-embryonated thick shelled eggs and embryonated eggs with weak thin shell.
3. **The mature thin shelled eggs hatch inside the small intestine of the host and mature into adults (i.e., internal autoinfection).**
4. Autoinfection is a part of the life cycle and leads to hyper-infection (a massive number of adult worms).
5. The eggs that pass in stool and reach water are swallowed by fish.
6. The swallowed eggs hatch and grow as larvae in the **fish intestines**.
7. **Humans acquire the infection by eating raw or undercooked small freshwater fish.**
8. Infective larvae develop into adults in the small intestine of the definitive host within 10 days, and the first-generation females produce the thin-shelled mature eggs.



❖ **Clinical picture:**

1. Diarrhea, borborygmus, abdominal pain, and edema.
2. If untreated, the symptoms are aggravated due to hyper-infection which leads to a protein-losing enteropathy with subsequent cardiomyopathy, severe emaciation, and cachexia.
3. Death may occur because of the irreversible effects of the infection which leads to protein and electrolyte disturbances. (The fatality rate of capillariasis is over 10%).

❖ **Diagnosis:**

- **Clinical: history and clinical manifestations**
- **Laboratory:**

1. **Stool examination**: for identification of eggs, larvae, or adults.

   N.B. The eggs are easily recognized under a microscope and have a characteristic shape and size. However, because the eggs are shed intermittently, multiple stool samples may be required to confirm the diagnosis.

2. **Intestinal biopsy and aspiration**.
3. **Serological tests:** are particularly useful in cases where stool examination is inconclusive or when the patient has a low worm burden.

❖ **Treatment**

1. Electrolyte replacement and administration of antidiarrheal agents.
2. **Thiabendazole (the drug of choice):** 25 mg/kg/day or 1 g/day for 30 days.
3. **Mebendazole or albendazole.**

❖ **Preventions and Control:**

1. Water sanitation.
2. Avoid eating raw fish. Cooking the fish for a short time would be sufficient to kill larvae in the intestines.
3. Early diagnosis and treatment are recommended to prevent serious disease and death.
4. Health education.

Trichinella spiralis
(Trichina worm)

- **Geographical distribution:** Cosmopolitan, mainly in pork-eating countries e.g. America and Europe.
- **Habitat:** Adult worm lives in the small intestine mainly duodenum and jejunum and larvae in the muscles of man and animals, so man and animals act as D.H & I.H.
- **D.H, I.H:** Rat, pig & Man.
- **Life cycle:**

  - The adults live in the small intestine of man (D.H), pigs, and rats (R.H).

  - After fertilization, the male dies and is expelled, while the female penetrates the intestinal wall to lay larvae (0.1 mm) in the submucosa.

  - Larvae are carried by the blood to liver → Rh side of the heart → lung →Lt side of the heart → general circulation → to all parts of the body especially the active striated muscles (eye lid, tongue, muscles of mastication, deltoid, intercostals muscles and diaphragm) where they are coiled and encysted as a result of host reaction in the long axis of muscles forming an oval cyst (0.5× 0.2 mm).

  - Larvae increase in size from 0.1 to 1mm → infective larvae within 2 weeks.

  - Man is infected by ingestion of undercooked pork containing the encysted larvae. In the small intestine, larvae are released and moult 4 times and mature to adults in 48 hours.

  - Adult female starts to lay larvae 5 days after infection.

  - Cysts are calcified in 6-12 months.

  - Pigs are infected by eating flesh from infected pig or dead pigs or infected rats.

  -Rats are infected by eating infected rat (cannibalism) or infected pig's muscles.

Fig. (18 - 1)
Female

Fig. (18 - 2)
Male

Fig. (18 - 3, 4, 5) Larvae in muscles

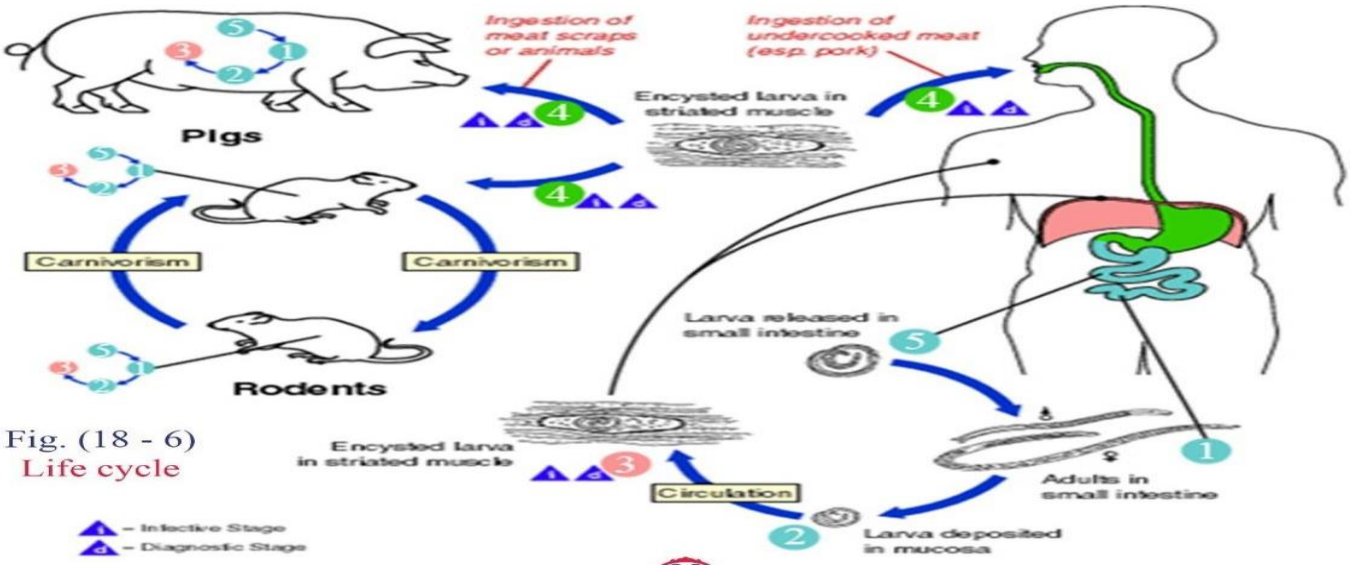


Fig. (18 - 6)
Life cycle

❖ **Pathogenesis and symptomatology:**

- **Disease: Trichinosis, trichiniasis or trichinelliasis.**
- The symptoms vary depending on the amount of encysted larvae ingested, age and host immunity.
- There are two main phases for infection: -

1) Enteral phase (affecting the intestine):

- A small worm burden is usually asymptomatic.

- A large worm burden in the intestine produces symptoms such as nausea, vomiting, dyspepsia, abdominal pain and diarrhea (like food poisoning).
- This phase occurs from 2 to 7 days after infection.
- Eosinophilia was detected early and increased.

2) Parenteral phase: (outside the intestine):

Larvae migrate in the blood → invade different tissue → initiate the inflammatory reaction and presented by:

a. Fever and urticarial rash due to toxemia.

b. Muscles: Pain and tenderness of the affected muscle:

- Eye lids → periorbital edema (a classic sign), subconjunctival hemorrhage, and difficulty in eye movement.
- Tongue → difficulty in speaking and dysphagia.
- Diaphragm and intercostal muscles → difficulty in breathing.
- Muscles of mastication → difficulty in mastication.

c. Splinter hemorrhage under the nails (a common symptom)

d. Larynx: Hoarseness of voice.

e. Heart: Myocarditis → may lead to heart failure.

f. Lung: Pneumonia.

g. Brain: encephalitis and meningitis.

h. In severe cases, death can occur 4-6 weeks after the infection and is usually caused by myocarditis, encephalitis, or pneumonia.

i. Eosinophilia: 20-50%.

❖ **Diagnosis:**

1) **History:** Eating improperly cooked meat.

2) **Clinical:** Periorbital edema, splinter hemorrhage, non-specific gastroenteritis, and muscle pain.

3) **Laboratory:**

1) **Direct:**

a. Stool examination: For larvae or adults (possible during enteral phase).

b. Blood examination for detection of larvae by mixing blood with dilute acetic acid then centrifuge and examine the precipitate (possible during parenteral phase)

c. Muscle biopsy: Must be taken from the swollen tender part of gastrocnemius, biceps or deltoid muscles. The specimen is compressed between two slides after digestion in acid pepsin to see larvae (possible during parenteral phase).

d. Trichinoscopy: To see larvae in muscle

e. X ray for calcified cyst.

f. CT for brain lesions.

2) **Indirect:**

1- **Eosinophilia** (20 - 50%).

2- **Intradermal test (Bachman test):** Intradermally injection of 0.1 c.c of 1: 10000 dilution of larval antigen in the forearm and other forearm injected by normal saline as a control test. In positive cases immediate reaction occurs within 20 minutes in the form of wheal surrounded by erythema. The test becomes positive after 2 weeks of infection and remains for several years

3- **Serological tests:** CFT, IHA, IFAT, ELISA, bentonite flocculation test (BFT) & latex agglutination test (LAT).

❖ **Treatment: -**

1) **General treatment:**

- Bed rest and fluid therapy
- Sedatives for headache and muscle pain.
- Corticosteroids to reduce inflammatory reactions.
- Cardiac and respiratory monitoring.

2) **Specific therapy:**

- Thiabendazole (Mintezol): 25 mg/kg twice daily for 10-14 days.
- Mebendazole (Vermox): 200 mg 3 times daily for 2 weeks.

❖ **Prevention and control:**

1) Treatment of the patient.
2) Avoid eating pork or proper cooking or freezing of pork.
3) Proper inspection of pig's meat in slaughterhouses by trichinoscope.
4) Treatment of pigs.
5) Proper breading places for pigs and heat treatment of garbage used in feeding pigs.
6) Rodent control.
7) Health education.

1- A 56-year-old male renal transplant patient presented with dyspnea and coughing blood. His body temperature was 38.5 ^{O}C. Laboratory investigations showed high eosinophilia.

Which of the following parasites suspected to cause these manifestations?

a) *Diphyllobothrium latum.*
b) *Strongyloides stercoralis.*
c) *Taenia saginata.*
d) *Trichostrongylus colubriformis.*
e) *Trichuris trichiura.*

2- **Which of the following drugs is suitable for treatment of a male child with iron deficiency anemia and his stool showed oval eggs with 4 cell stage?**

a) Mebendazole.
b) Metronidazole.
c) Niclosamide.
d) Praziquantel.

3- **Baermann's technique can be used in the diagnosis of infection by which of the following parasites?**

a) *Capillaria philippinensis.*
b) *Fasciola gigantica.*
c) *Heterophyes heterophyes.*
d) *Strongyloides stercoralis.*

Nematodes of large intestine

Enterobius vermicularis

(*Oxyuris vermicularis*, Pinworm, Seat-worm)

- **Geographical distribution:**

  Worldwide. It is more prevalent in the cool and temperate zones. *E. vermicularis* is the world's most common parasite, especially among children.

- **Habitat:** large intestine, especially the caecum, appendix, and adjacent portion of ascending colon.
- **Definitive Host:** Man.
- **Intermediate Host:** no intermediate host.
- **Diagnostic stage:** eggs and adult worms.
- **Infective stage:** eggs.
- **Morphology:**

➢ **Adult Worms:**

- White worms with pointed ends (pinworms).
- The mouth is surrounded by 3 retractile small lips.
- Two cephalic alae (cuticular lateral expansion).
- The esophagus has a **double-bulb** structure, a unique feature to this worm. - Sex is separate.

- **Adult Female:**

  Size: 8–13 mm long.

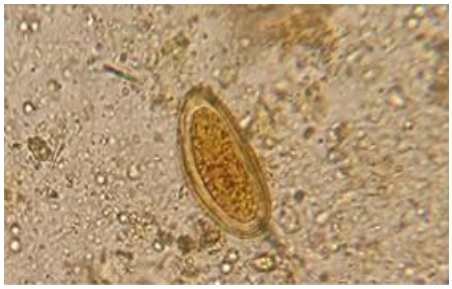
  Shape: Its posterior third is a thin straight pointed pin-like tail.

- **Adult Male:**

  Size: 2–5 mm long.

  Shape: has a curved caudal end.

➢ **Eggs (diagnostic and infective stage)**:



- **Size:** 50 x 20 µm.
- **Shape:** elongated ovoid, straight on one side, and convex on the other (planoconvex).
- **Shell:** double-layered and relatively thick with an outer albuminous layer through which the eggs stick to each other, clothing, and other objects.
- **Color:** translucent.
- **Contents:** mature (larva).

❖ **Life cycle:**

1. Adult worms live in the large intestine, especially the caecum, appendix, and adjacent portion of ascending colon.
2. After fertilization of the female worms, the males usually die and is passed out in the stool.
3. At night, the gravid female migrates down the colon to the rectum. It comes out through the anus and crawls on the perianal skin to lay its sticky eggs. Sometimes the female worm enters the urethra or vagina of female patients.
4. Crawling of the gravid female worm leads to pruritis and the patient scratches the affected perianal area. This transfers eggs of *E. vermicularis* to the fingers and under nails.
5. **Man acquires infection via one of the following:**

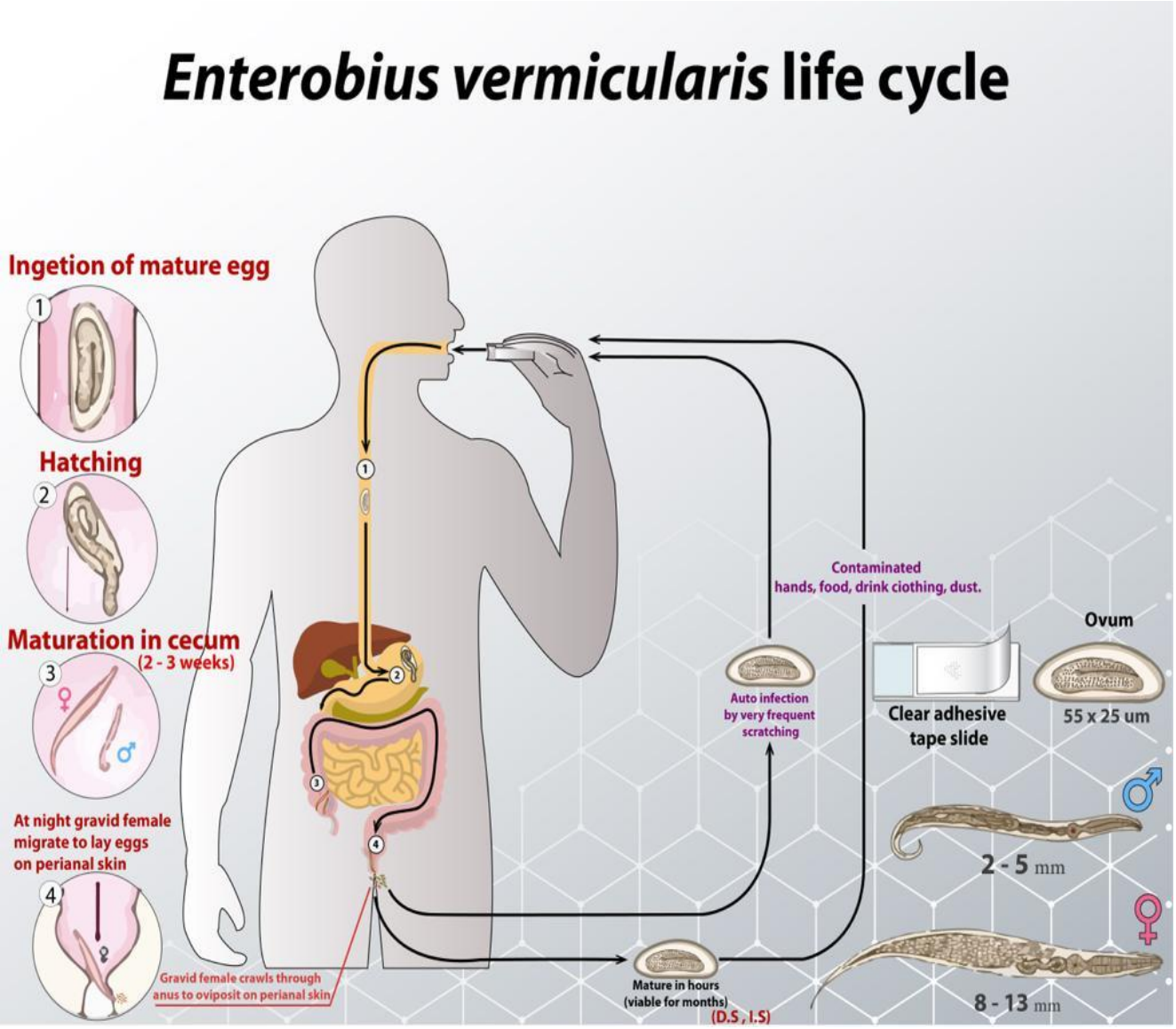
a. **Self-inoculation (transferring eggs to the mouth with hands that have scratched the perianal area). i.e., autoinfection.**

b. **Retro-infection: The eggs laid on the perianal skin immediately hatch, and the larvae migrate through the anus to develop into adults in the colon.**

c. **Through exposure to eggs in the environment (e.g., contaminated surfaces, clothes, bed linens, etc.).**

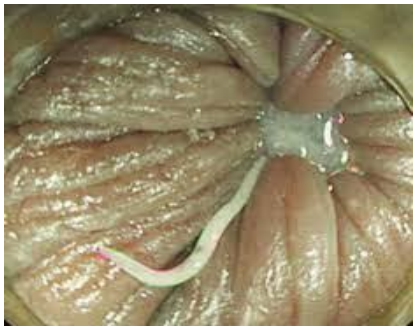
d. **Rarely, eggs may become airborne and be inhaled and swallowed.**

6. The swallowed eggs hatch in the intestine. They moult in the ileum and enter the caecum, where they mature into adults.



❖ **Clinical Picture:**

Name of the Disease: Enterobiasis or Oxyurisasis.



1. Many individuals remain asymptomatic.
2. The most striking symptom of this infection is pruritus ani which is caused by the migration of the female worms from the anus onto the perianal skin for egg deposition at night. In most infected people, his may be the only symptom.
3. As the worm migrates out at night, it causes sleep disturbances with subsequent nervousness, insomnia, and restlessness.
4. Nocturnal enuresis may occur. It is more common in female children.
5. In heavily infected females, the worm crawling into the vulva and vagina causes irritation, a mucoid discharge with pruritus vulvi.
6. It may migrate up to the uterus, fallopian tubes, and into the peritoneum. This may cause chronic salpingitis, cervicitis, peritonitis, and recurrent urinary tract infections.
7. The presence of *Enterobius* in the appendix may result in inflammation that causes appendicitis.

❖ **Diagnosis:**

- **History and clinical picture:** anal itching, irritability, and insomnia may suggest a pinworm infection.
- **Laboratory:**

A. Detection of Eggs by:

1. NIH Swab Method

The NIH swab (National Institutes of Health) has been widely used for the collection of specimens. It consists of a glass rod at one end of which a piece of transparent cellophane is attached with a rubber band. The glass rod is fixed on a rubber stopper and kept in a

wide test tube. The perianal region is swabbed with the cellophane paper then spread on a slide and examined microscopically for eggs.

2. **Scotch adhesive tape swab:**

   A piece of sticky surface tape is applied to the perianal skin. The tape is transferred to a glass slide, with the sticky side down and examined under the microscope**.**

   N.B.: Swabs should be taken in the morning before defecation or bathing.

3. **Detection of eggs in stool:** eggs can be detected in faeces in a small proportion of patients.
4. **Nail examination:** The eggs may be demonstrated in the dirt collected from beneath the fingernails of infected children.
5. **Urine examination** for eggs.

B. Detection of adult worms in the stool or perianal region.

❖ **Treatment:** In households where more than one member is infected or where repeated, symptomatic infections occur, it is recommended that all household members be treated at the same time.

1. **Albendazole:** 400 mg single dose given on an empty stomach (200 mg in children under 2 years of age), is the drug of choice. It should be repeated in 2 weeks to kill any worms that might have hatched from eggs present at the time of initial treatment.
2. **Mebendazole:** A 100 mg single dose repeated after two weeks.
3. **White precipitate ointment** to the perianal area to relieve inflammation and itching.

❖ **Prevention and control:**

1. Maintenance of personal and community hygiene.
2. Frequent washing and boiling of internal clothes and bed linen.
3. Washing hands before meals and after defecation.

4. Cleaning and cutting fingernails.
5. Children should wear tight underwear to prevent their hands from reaching the perianal area during sleep.
6. Food protection from contamination by infected hands, dust, and flies.
7. Treatment of the whole family at the same time.
8. Fly control.
9. Health education.

Trichuris trichiura
(*Trichocephalus trichiura*, Whip worm)

- **Geographical distribution:** Worldwide. More common in tropical countries.
- **Habitat:** large intestine (mainly caecum).
- **Definitive Host:** Humans.
- **Intermediate Host:** no intermediate host.
- **Diagnostic stage:** immature eggs.
- **Infective stage:** eggs containing first-stage larva.

Morphology:

- **Adults:**

The anterior end of the worm is slender, and the posterior end is thicker, giving it a "buggy whip" appearance, hence the name whipworm.

- **Eggs (diagnostic stage):**

Size: 50 x 25 µm.

Shape: barrel shaped.

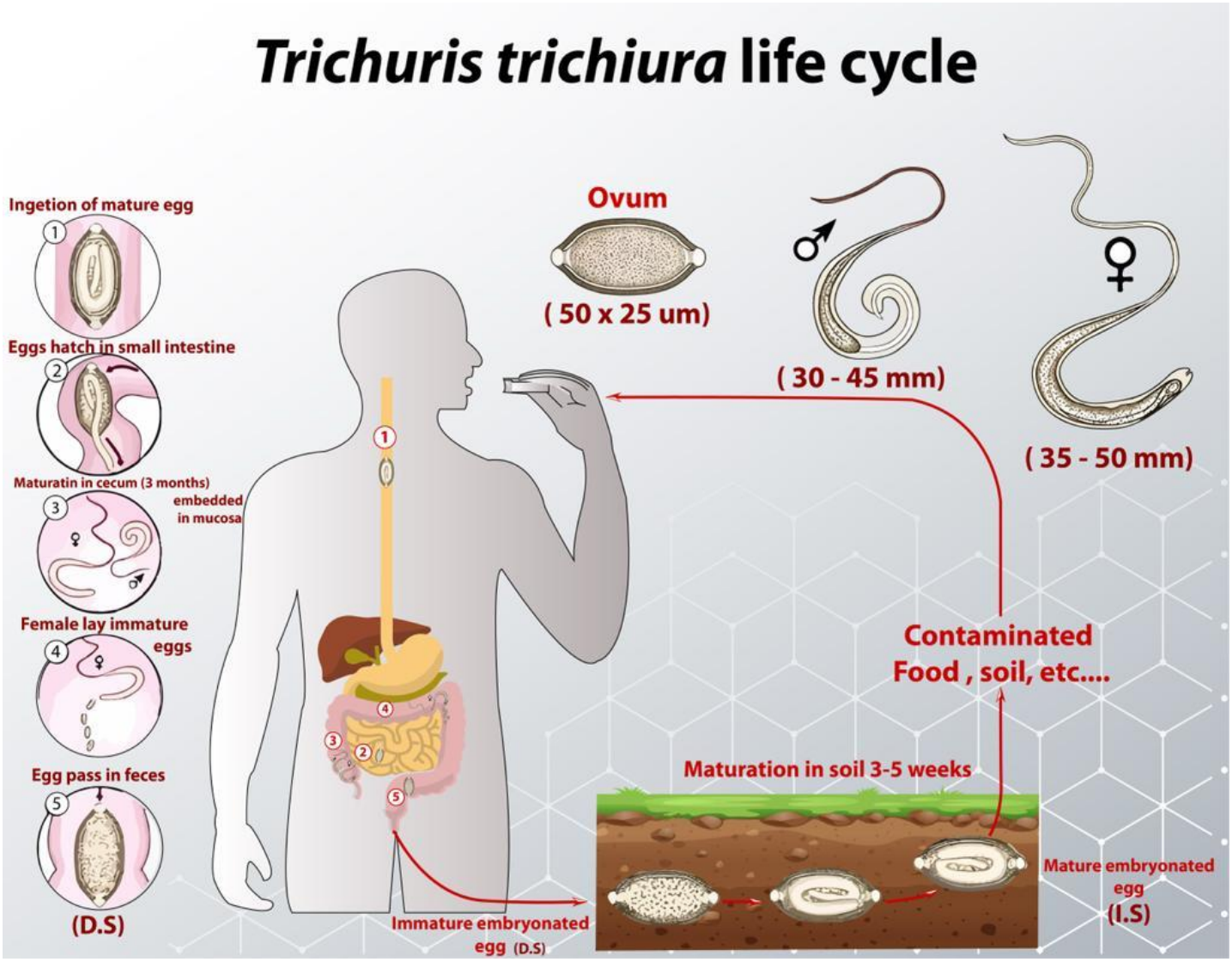
Shell: triple shell with a projecting mucus plug at each pole.

Color: brown but the plugs are colorless.

Content: immature (single ovum).

- **Life cycle:**

1. Adult worms live in the caecal wall with the thread-like anterior portion piercing the mucosa and the thick posterior end projecting out.
2. The gravid adult females lay eggs that are discharged in feces.
3. The eggs are embryonated in soil optimally under warm, moist, shady conditions where the infective 1st stage rhabditiform larva develops within the egg in 3–4 weeks.
4. **Human infection occurs when the mature embryonated eggs containing the infective 1st stage rhabditiform larvae are swallowed in contaminated food or water.**
5. The eggs hatch in the small intestine and the larva emerges through the pole of the egg and passes down into the caecum where moulting and maturation into adults occur.



▶ **For more information, visit this website:**

https://www.youtube.com/watch?v=kgKkV6NSeaA

❖ **Clinical Picture:**

Disease name: trichuriasis, Whipworm infection, or Trichocephaliasis.

1. Light infection is asymptomatic.
2. Infections of moderate to heavy worm loads present with lower abdominal pain, distention, and diarrhea.

3. Severe infection may lead to dysentery, profuse bloody diarrhea, cramps, tenesmus, and urgency.
4. *Trichuris* may also cause rectal prolapse in children with heavy infections. Prolapse results from increased peristalsis that occurs to expel the worms. The whitish worms may be seen on the prolapsed mucosa.
5. Anemia is a common finding, either microcytic hypochromic anemia due to continuous blood loss or hyperchromic pernicious anemia due to toxin production.
6. Worms may occasionally migrate to the appendix, causing appendicitis.

❖ **Diagnosis:**

- **History and clinical picture.**
- **Laboratory:**

a. Stool Examination: for detection of eggs.

b. Blood Examination: Differential leukocyte count may show up to 25% eosinophilia in the early stage of the disease.

c. Sigmoidoscopy: In heavy infection, sigmoidoscopy may show the white bodies of worms hanging from the inflamed mucosa, so it is called coconut cake rectum.

❖ **Treatment:**

1. **Mebendazole:** 100 mg twice daily for 3–5 days.
2. **Albendazole**: a single dose of 400 mg.

❖ **Prevention and control:**

1. Treatment of infected persons.
2. Proper disposal of feces.
3. Avoid consumption of unwashed fruits and vegetables and proper washing of green vegetables.
4. Pure water supply.
5. Fly control.
6. Health education.

1- Which of the following parasites can be complicated by both appendicitis and nocturnal enuresis?

a. *Ancylostoma duodenale.*
b. *Ascaris lumbricoides*
c. *Diphyllobothrium latum.*
d. *Enterobius vermicularis.*
e. *Trichuris trichiura.*

2- Which of the following complications is expected to occur in infection with the shown organism?

Brain embolism.
Cholecystitis.
Hypokalemia.
Intestinal obstruction.
Rectal prolapse**.**

3- Which of the following complications is expected to occur in infection with the shown parasite?

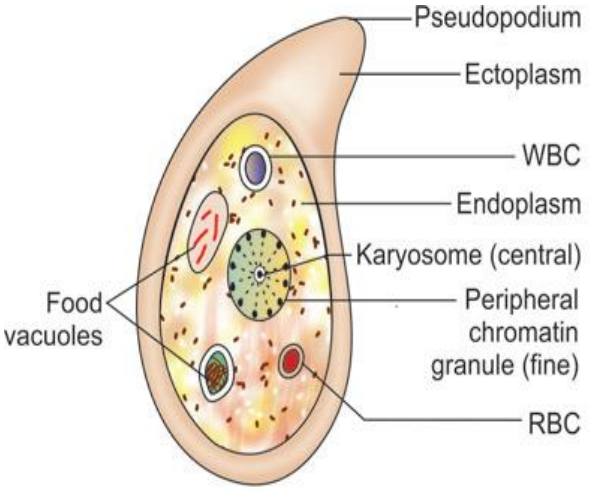
Nocturnal enuresis.
Cholecystitis.
Intestinal obstruction.
Lung abscess.
Rectal prolapse.

Intestinal protozoa

Entamoeba histolytica

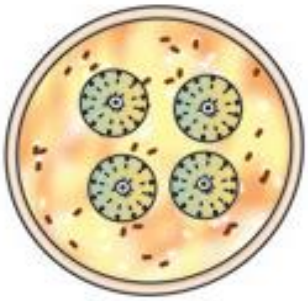
- **Geographical distribution:** worldwide disease, but it is more prevalent in the tropics and subtropics. Prevalence rates increase in areas of crowding and poor sanitation.
- **Definitive host:** man
- **Intermediate host**: no intermediate host.
- **Habitat:** large intestine (colon, rectum, sigmoid colon, and colonic flexures)
- **Infective stage**: mature quadri-nucleate cyst
- **Diagnostic stage**: trophozoites and cysts (immature and mature)
- **Morphology:**

➢ **Trophozoite**



- 12 to 60 μm in diameter (average 20 μm).
- Mononucleated.
- Trophozoites are actively motile by pseudopodia (cytoplasmic protrusions).
  The pseudopodium may be in the form of a short, blunt, and broad, or long and fingerlike.
- The nuclear membrane appears as a delicate line, on its inner surface there are peripheral chromatin dots (uniform and small). In the center of the nucleus there is a small mass of chromatin (karyosome) and between the karyosome and the chromatin dots there are thin fibrils.
- Red blood cells may be ingested by trophozoites.

➢ **Cysts**



Quadrinucleated cyst

- Rounded.
- About 15 µm in diameter.
- may contain one nucleus (mono-nucleated cyst) , two nuclei (di-nucleated cyst) or four nuclei (quadri-nucleated cyst).
- All cysts are diagnostic stages while only the mature quadri-nucleated cyst is the infective stage.
- The nucleus is similar to that of trophozoites. The immature cyst contains chromatoid bodies (stored glycogen) in the form of rods of cigar-shaped structures.

❖ **Life cycle**

1- **Man is infected by ingestion of the fully mature quadri-nucleate cyst by feco-oral route either by autoinfection or contaminated food or drink which may be direct by food handlers or indirect by *Musca domestica* (house fly).**

2- Inside the human body, the life cycle of *E. histolytica* consists of four consecutive parasitic stages, 1) the metacyst 2) trophozoite 3) precyst 4) cysts (immature and mature).

3- The cyst is resistant to gastric acid, so excystation occurs in the small intestine. The amoeba within the cyst becomes active in the neutral or alkaline pH of the small intestine.

4- The cyst wall is digested by the digestive enzymes in the intestine.

5- The amoeba becomes very active and each of the four nuclei in the emerging *E. histolytica* (metacyst) undergoes one division, thus forming eight amoebae (smaller than the trophozoites seen in the colon).

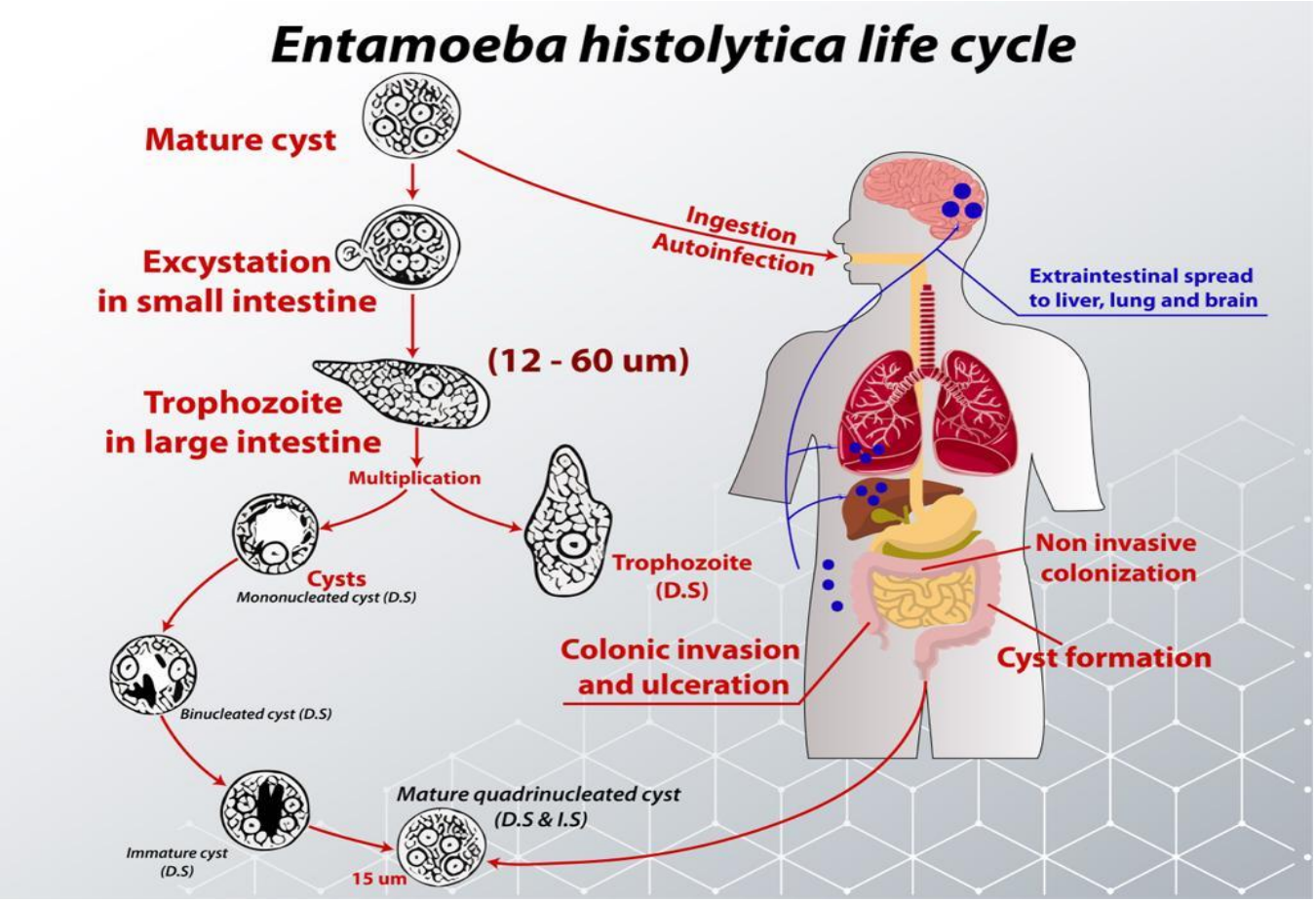
6- They are carried into the caecum where they complete their maturation to trophozoites which multiply by binary fission and the nucleus divides by mitosis.

7- As the amoebae pass down the colon, they become dehydrated and take a spherical shape to become precysts.

8- The precyst secrets a thin cyst wall forming an immature cyst. Two mitotic divisions occur, resulting in a cyst that contains four nuclei (mature cyst).

9- Mature cysts pass in the stool to the environment.

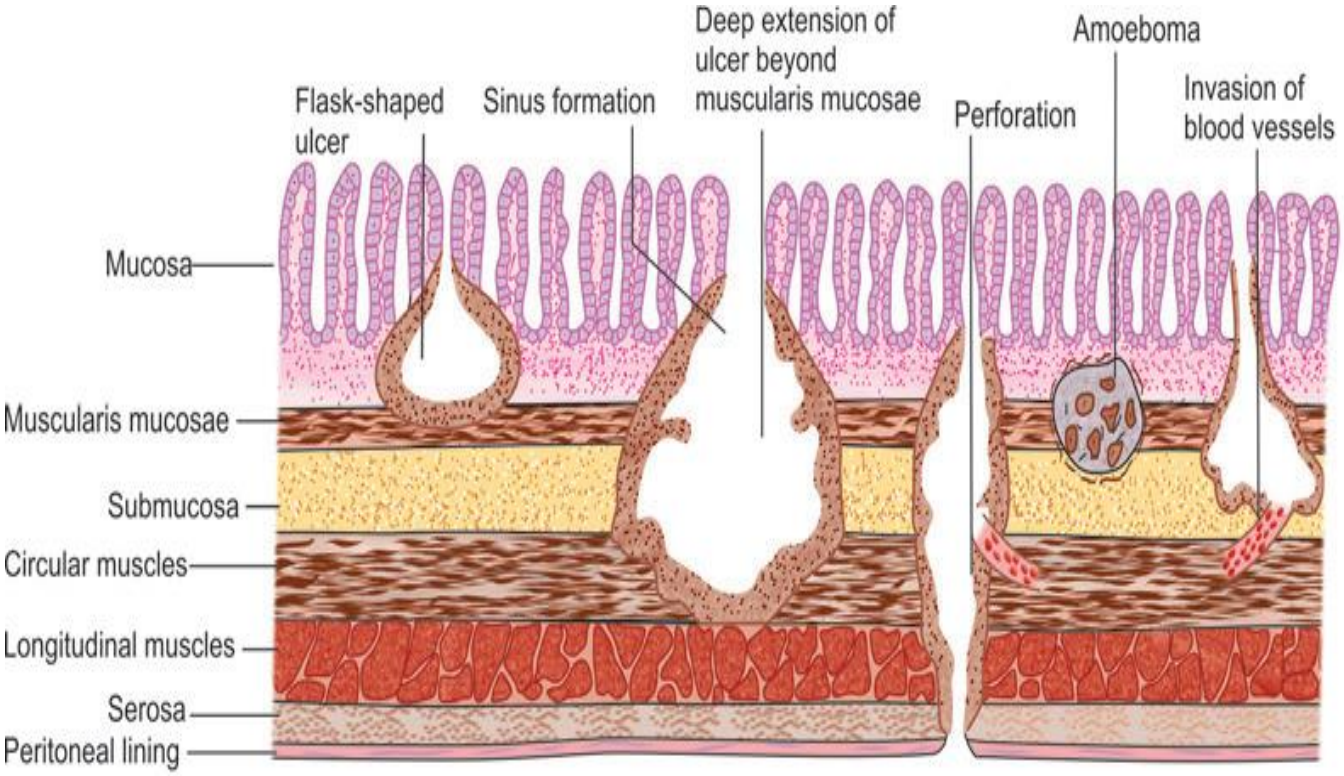
10- Cysts remain viable and infective for several days in feces and water, but they are easily killed by dehydration.



❖ Pathogenesis and Pathology:

- In cases with heavy infection, low immunity and high virulence of the organism the trophozoite of E. histolytica invade the mucosa and submucosa of the large intestine by secreting lytic enzymes causing the classic flask-shaped ulcers of the intestinal mucosa with erosion of blood vessels and bleeding

- The most common sites of amebic ulcers are caecum, colonic flexures and sigmoidorectal region
- Ulcers may be complicated by secondary bacterial infection with necrosis, sloughing, perforation, peritonitis, and appendicitis.
- Amoebae spread from the intestine to the liver occurs through portal circulation.
- In patients treated with corticosteroids, amoebic trophozoites can be found in every organ of the body, including the brain, lungs, skin, and eyes, even cartilage and bone



Pathogenesis and pathology of amebiasis

❖ **Clinical Picture:**

The pathogenic activity of *E. histolytica* depends upon

1-The host immunity

2-The virulence of the parasite (type of the strain, invasiveness, number of parasites)

3-The condition of the intestinal tract (carbohydrate diet, injury of the mucosa, stasis, bacterial flora)

- **Asymptomatic infections**

Most common E. histolytica patients may be asymptomatic cyst passers.

- **Symptomatic infections:**

a-Intestinal amebiasis

- Dysentery and diarrhea account for 90% of cases of invasive intestinal amebiasis.

1- Acute amoebic dysentery:

colicky abdominal pain, frequent bowel motions (up to 10 per day), tenesmus and dysentery (stool with blood and mucus).

- In severe cases, symptoms may begin suddenly with profuse diarrhea (over 10 stools/day), fever, dehydration, and electrolyte imbalances. (trophozoites present in stool)

2- Chronic infection:

low grade fever, recurrent episodes of diarrhea alternating with constipation (cysts present in stool).

b) Extraintestinal amebiasis

1) Hepatic (Acute hepatitis or liver abscess)

•The trophozoites living in the submucosa can reach the liver via portal circulation.

•The onset of symptoms may be gradual or sudden. There is upper right abdominal pain with fever from 38 to 39 °C in most cases.

•There may be hepatomegaly

Cross section of liver showing amoebic liver abscess

with tenderness. Liver function tests usually
remain normal or slightly elevated with jaundice (very rare).
•Amoebic hepatitis is a diffuse early stage of liver infection before abscess formation.
-There is leukocytosis of 15,000 to 35,000/ microliter
The presence of leukocytosis, a high alkaline phosphatase level, and an elevated right diaphragm suggests an amoebic hepatic abscess

Lung abscess

- Amebiasis of the lungs occurs when a liver abscess ruptures through the diaphragm.
- It may also occur via blood spreading from the intestine.
- Rarely, the pericardium can be invaded in patients with an abscess in the left lobe of the liver.

2) Other ectopic foci

- Amebiasis of the skin (Amebiasis cutis) usually develops as a perianal extension of acute amebic colitis or on the abdominal wall due to rupture of an intestinal or hepatic lesion.
- Amebiasis cutis may also occur as a venereal infection of homosexual persons.
- Amoebic brain abscess is rare and usually spreads from either hepatic or pulmonary lesions via blood.
- Other rare ectopic sites include the spleen, adrenals, kidneys, ureters, urinary bladder, and urethra.

❖ Diagnosis

- **History and clinical picture**
- **Laboratory diagnosis**

a) Direct diagnostic methods

1. Stool examination:

- Wet mount preparations from stool are positive in only 10% of cases. But it remains the gold standard to detect *E. histolytica* trophozoites which have phagocytosed red blood cells. In wet smear trophozoites can be seen moving by pseudopodia.
- Cysts in stool are strongly suggestive of amoebiasis because its presence does not differentiate between pathogenic and non-pathogenic (*E. dispar*).
- For morphological identification, a permanent stained smear with trichrome or iron hematoxylin may be done.
- Charcot-Leyden crystals (microscopic crystals formed of eosinophilic protein) may be found in association with *E. histolytica* in the stool of chronic patients.

2. Sigmoidoscopy specimens

At least six areas of the mucosa should be sampled and stained with permanent stains.

3. **Liver abscess aspirate**

- Definitive diagnosis of liver abscess can be reached by isolation of trophozoites from liver aspirate material.
- Amebae trophozoites are found only in the abscess wall not in the necrotic center.

b) Indirect (serodiagnosis & Antigen detection)

- Serological tests to detect anti-amoebic antibody, include immunofluorescent antibody tests (IFAT), indirect haemagglutination assays (IHAs), radioimmunoassay (RIA), and enzyme-linked immunosorbent assays (ELISAs).
- ELISAs are the most sensitive and specific in patients with amoebic liver abscesses.

- The results of serological tests may be negative in acute disease and should be repeated in 5–7 days.

c) Stool Antigen detection.

d) Molecular diagnosis.

e) Radiological investigations e.g., ultrasonography and CT for diagnosis of complications, liver abscess, and ameboma.

❖ **Treatment**

a) <u>Medical treatment</u>

- There are two classes of drugs used in the treatment of amoebic infections.

1) Luminal amoebicides (diloxanide furoate and iodoquinol):

- They act on organisms in the intestinal lumen and are not effective against tissue stages.

2) Tissue amoebicides (metronidazole, tinidazole, nitazoxanide, dehydroemetine and chloroquine):

- They are effective in the treatment of invasive amoebiasis but less effective in the treatment of lumen stages.
- Metronidazole or tinidazole are the **drugs of choice** for amoebic colitis and liver abscess. But it should be followed by a luminal agent such as diloxanide furoate to eradicate the luminal stages.

Drug treatment of amoebiasis		
Disease	**Drugs**	**Doses**
Asymptomatic Intestinal Carrier	diloxanide furoate	500 mg 3 times a day ×10 days
Intestinal amoebiasis	Metronidazole followed by diloxanide furoate. Or tinidazole followed by diloxanide furoate.	750–800 mg 3 times a day ×10 days 500 mg 3 times a day ×10 days 2 g/day ×2–3 days 500 mg 3 times a day ×10 days
Amoebic Liver Abscess	Metronidazole followed by diloxanide furoate. Or tinidazole followed by diloxanide furoate. Or dehydroemetine followed diloxanide furoate	The same dose as intestinal amoebiasis The same dose as intestinal amoebiasis 1–1.5 mg/kg daily (max. 90 mg/day) IV ×5 days 3 times a day ×10 days

b) <u>Surgical treatment</u>

- Surgery for patients with rupture of the abscess, or when an abscess that needs drainage cannot be reached percutaneously for anatomical reasons.
- Surgical treatment of complications such as intestinal perforation and appendicitis.

❖ **Prevention and Control**

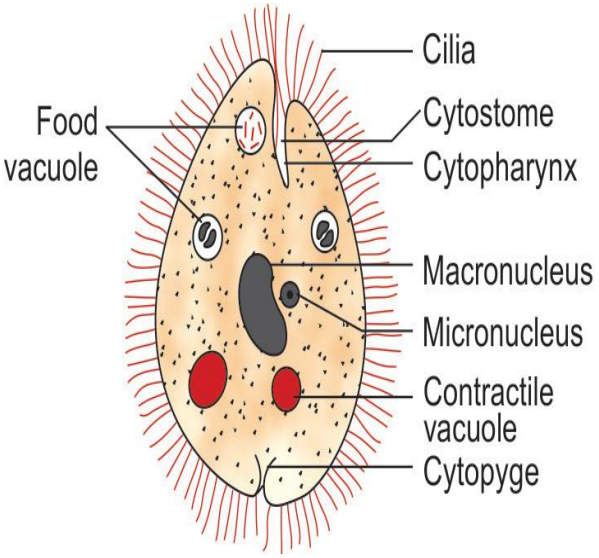
- Cysts remain viable and infective for several days in feces and may survive in soil for 8 days at 34–38°C, and for 1 month at 10°C.
- Cysts remain infective in fresh water, sea water, sewage, and wet soil.
- Cysts survive for up to 45 minutes in fecal material lodged under the fingernails.

Therefore, the following measures may be applied:

1. Sanitary water supply, adequate disposal of feces, food safety and health education to prevent fecal–oral transmission.
2. Early detection and treatment of patients and food handlers.
3. Using iodine, 5–10% acetic acid, and heating at a temperature above 68°C to kill *E. histolytica* cysts.
4. The quantity of chlorine normally used in water purification doesn't kill the cysts; therefore, chlorination alone cannot prevent epidemics originating from fecal contamination of water.
5. In places where purification of water supplies is inadequate, boiling for 10 minutes will kill all cysts.
6. Proper washing hands and cutting nails short.
7. Fly control.

Balantidium coli

- **Geographical Distribution:** worldwide. Because pigs are the primary reservoir (63% to 91% of pigs harbor *B. coli*), human infections occur more frequently in areas where pigs are raised, and sanitation is inadequate.
- **Habitat:** large intestine (caecum, colon).
- **Definitive Host:** Man.
- **Reservoir host:** Pigs, less common monkeys.
- **Infective stage:** Mature cyst.
- **Diagnostic stage:** cyst and trophozoite.
- **Morphology:**



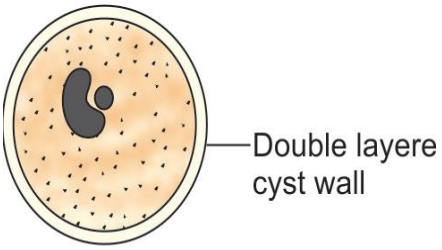
➢ **Trophozoite:**

It is the active form of the parasite, which multiplies in the intestinal lumen, tissue,and organs of the host, and therefore considered the pathogenic form.

- **Size:** 60 × 45 µm (largest protozoon of man).
- **Shape:** ovoid, tapers at the anterior end with boring motility. It is covered with a layer of cilia (organ of locomotion). It has a small cytostome (primitive mouth) which leads to cytopharynx that extends to 1/3 of the body length.
- **Nuclei:** It has two nuclei, Kidney-shaped macronucleus (appear as a hyaline mass, especially in unstained preparations) and small spherical micronucleus (often not readily visible, even in stained preparations).
- **Cytoplasm:** granular cytoplasm, contains two contractile vacuoles. food vacuoles, as well as ingested microbes (bacteria).

➢ **Cyst**: It is the resistant, and the infectious form.

- **Size:** 52 to 55 µm.
- **Shape:** Subspherical to oval, Double cyst wall with row of cilia visible in between cyst wall layers.
- **Nuclei:** two; the macronucleus and micronucleus,
- **Cytoplasm:** granular cytoplasm contains one or two contractile vacuoles. The other structures are not usually apparent.



❖ **Life cycle:**

Mode of infection:

1. **<u>Human infections can occur through:</u>**

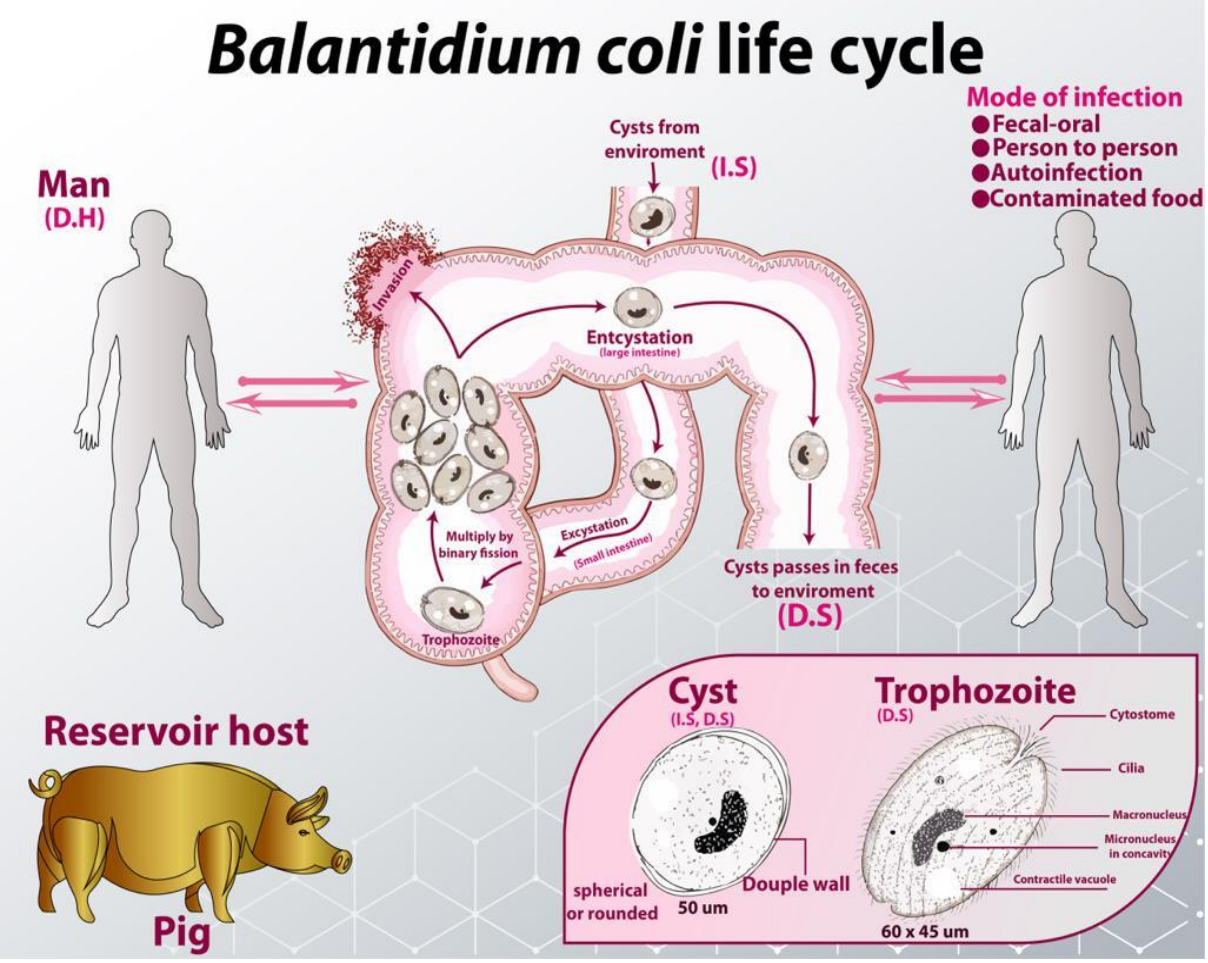
a. <u>fecal-oral route; through contaminated food or drink with pigs' feces.</u>

b. <u>Person to-person spread, as in food handlers.</u>

c. <u>Autoinfection.</u>

d. <u>House flies may carry cysts to exposed food.</u>

2. Excystation occurs in the small intestine, the resulting trophozoites take up residence and feed primarily in the cecal region and terminal ileum, as well as in the lumen, mucosa, and submucosa of the large intestine.
3. Multiplication of the trophozoite occurs by transverse binary fission, from which two young trophozoites emerge.
4. Part of the trophozoites invade the wall of the colon and multiply, causing ulcerative pathology in the colon wall. The remaining part returns to the lumen.
5. Trophozoites are delicate and not able to survive in the outside environment, so encystation occurs in the lumen. The resulting cysts mature and pass out in stool ready for transmission into a new host.



❖ **Clinical picture:**

Disease: Balantidiasis.

Pathogenesis:

B. coli disease is like amebiasis as the organisms secrete proteolytic and cytotoxic substances that mediate tissue invasion and intestinal ulceration. Infection is severe in immunocompromised patients.

Clinical manifestations:

1. Light infections are asymptomatic despite that, they can still transmit the infection.

2. Symptomatic disease may be mild or severe. Mild infection is usually in the form of mild abdominal discomforts.
3. Severe infection is characterized by abdominal pain and tenderness, tenesmus, nausea, anorexia, and watery stools with blood and pus. Ulceration of the intestinal mucosa and secondary bacterial invasion into the eroded intestinal mucosa can occur.
4. Chronic infections may develop in the form of a tender colon, anemia, cachexia, and occasional diarrhea, alternating with constipation.
5. Extraintestinal invasion of other organs is extremely rare in balantidiasis.

❖ **Complications:**

a. Haemorrhage.
b. Secondary bacterial infection.
c. Appendicitis.
d. Intestinal perforation and peritonitis.

❖ **Diagnosis:**

- **History and Clinical picture**
- **Laboratory:**

a- Stool examination: for detection of *B. coli* cysts and trophozoites.

b- Colonoscopy or sigmoidoscopy to obtain a biopsy specimen from the large intestine, which may be an evidence for the presence of trophozoites.

❖ **Treatment:**

1. **Tetracycline:** adults: 500 mg orally four times daily for 10 days.
   Children ≥ 8 years old: 40 mg/kg/day (max. 2 grams) orally in 4 doses for 10 days.

   Note:

   -Tetracyclines are contraindicated in pregnancy and in children < 8 years old.

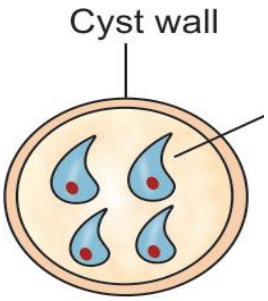
2. **Metronidazole:** adults: 500-750 mg orally three times daily for 5 days. Children: 35-50 mg/kg/day orally in three doses for 5 days.
3. **Iodoquinol** 650 mg orally 3 times a day for 20 days. Iodoquinol is considered an alternative to tetracycline in the treatment of *Balantidium coli* infections.

❖ **Prevention and control:**

1. As in amoebiasis
2. Care in handling pigs in pig farms and slaughterhouses.

Cryptosporidium parvum

- **Geographical distribution:** worldwide.
- **Definitive host:** man.
- **Reservoir host:** rats, guinea pigs, pigs, horses, etc.
- **Habitat:** small and large intestine.
- **Morphology**



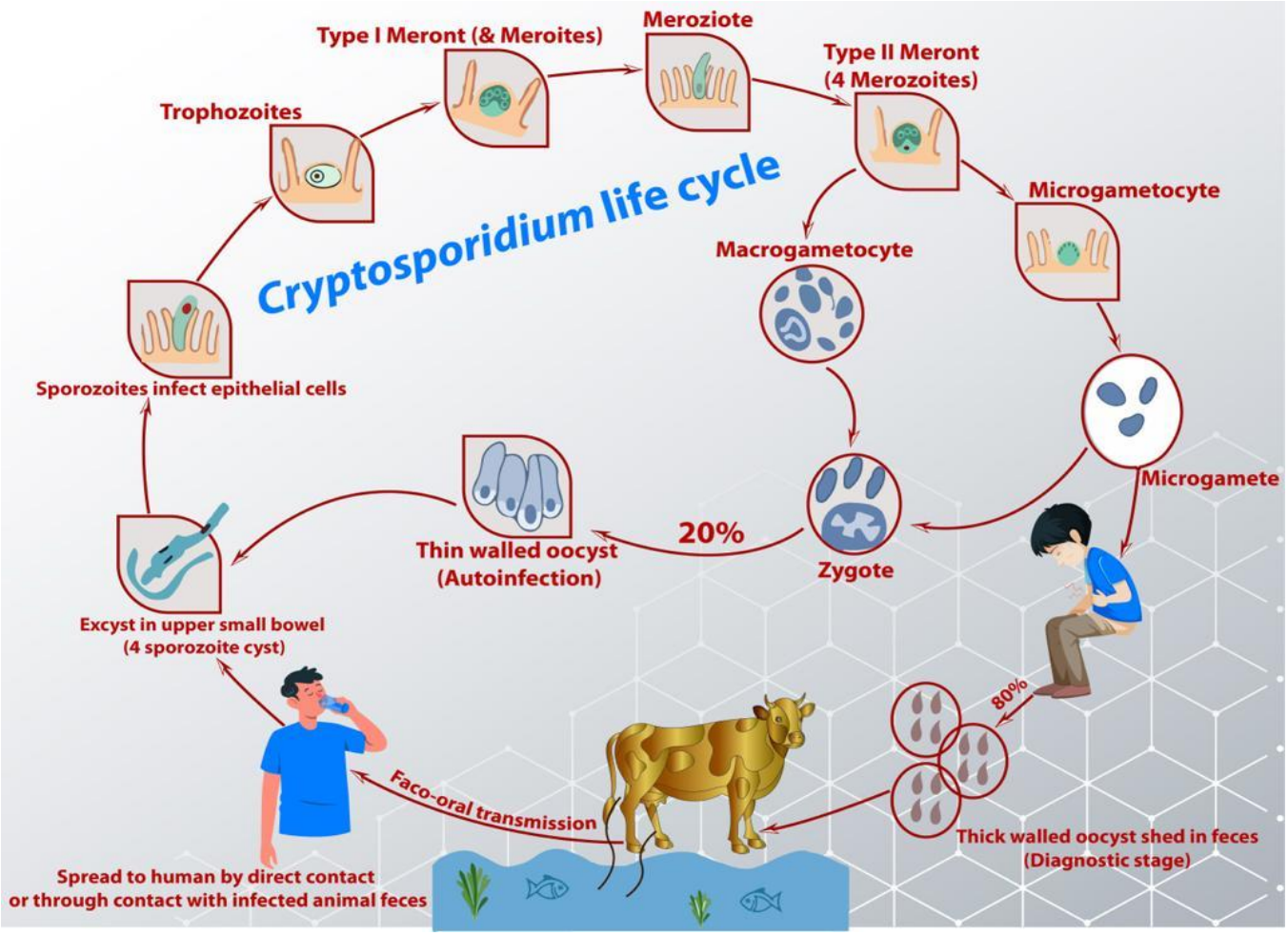
➢ **Oocyst (the infective and the diagnostic stage).**

- **Size:** 4- 6 µm.
- **Shape:** Rounded surrounded by a cyst wall and bears four sporozoites.
- Each sporozoite is crescentic shaped with a pointed anterior end, blunt posterior end and a nucleus located posteriorly.
- The oocysts are acid fast in nature but don't stain by iodine.
- They are extremely resistant to routine chlorination, heat, and other disinfectants.

❖ **Life Cycle**

1. *Cryptosporidium parvum* completes its life cycle (both sexual and asexual stages) in single host (man or other animals).
2. Sporulated oocyst is the infective form of the parasite. **Man acquires infection by ingestion of food and water contaminated with feces containing oocysts.**
3. In the small intestine, the wall of the oocyst gets dissolved and four slender sporozoites are released from each oocyst.
4. Sporozoites invade the brush border epithelium of the small intestine and lie inside a parasitophorous vacuole near the microvilli surface, within which all the stages of development take place.

5. The sporozoites subsequently differentiate into trophozoites which then undergo asexual multiplication (schizogony) to produce type I meronts.
6. Each type I meront undergoes schizogony to release eight merozoites, which then again invade the adjacent enterocytes and undergo repeated schizogony to produce type II meronts. Four merozoites are released by the schizogony of each type II meront.
7. The merozoites undergo gametogony and transform into sexual forms (microgamont and macrogamont). Each microgamont releases 16 microgametes while only one macrogamete is produced from each macrogamont.
8. Fertilization takes place between microgamete and macrogamete to produce the zygote. Subsequently, about 80% of zygotes transform into highly resistant double-layered thick-walled oocysts, and the remaining 20% transform into single-layered thin-walled oocysts.



❖ **Clinical features**

Cryptosporidium is an intestinal coccidian parasite affecting various animals and men. It causes self-limiting acute diarrhea in immunocompetent healthy individuals, whereas it is an opportunistic pathogen in immunocompromised patients (including HIV-infected patients), causing chronic persistent life-threatening diarrhea.

Cryptosporidiosis in Immunocompromised Hosts

- Autoinfection by thin-walled oocyst is key factor for the chronic diarrhea which maintains the infection.
- It produces chronic, persistent remarkably profuse diarrhea (1–25 L/day), leading to significant fluid and electrolyte loss (resembling cholera and diarrhea).
- Severe weight loss, and abdominal pain may be seen.
- Involvement of sites other than small intestine like pharynx, stomach, large intestine, and respiratory tract is quite common in HIV positive patients.
- Involvement of the biliary tract can cause papillary stenosis, sclerosing cholangitis, or cholecystitis and can manifest as mid epigastric or right upper quadrant pain.

❖ **Diagnosis:**

- **History and clinical picture.**
- **Laboratory Diagnosis**

1. Stool Examination:

- Three consecutive stool samples should be collected.
- Acid fast staining: The oocysts of *C. parvum* are acid fast to 1% sulfuric acid or acid alcohol and appear as round, 4–6 μm red color oocyst against blue background. The sensitivity of acid-fast staining is low, and it requires a minimum concentration of more than 50,000oocysts/mL of stool.

Commonly used modified acid-fast staining methods are:

−Kinyoun's method (cold acid fast staining).

−Rapid safranin methylene blue method.

−Carbol fuchsin negative staining method.

2. **Sputum, bronchial wash, duodenal or jejunal aspirate** can be also examined.
3. **Immunological diagnosis:**

a. Antigen detection from Stool

- ELISA has been developed to detect *C. parvum* specific coproantigen from stool, shows a sensitivity ranging from 66% to 100% with excellent specificity.
- Immunochromatographic test (ICT) is also available for simultaneous detection of antigens of *C. parvum, Giardia* and *E. histolytica.*

b. Antibody detection

- ELISA is used to detect *C. parvum*-specific antibodies (IgM and IgG) in a patient's serum for seroepidemiological purposes.
- Indirect fluorescent antibody test is also available to detect *C. parvum*-specific antibodies against oocyst antigens.

4. **Molecular diagnosis**

❖ **Treatment**

1. **Mild cases** are self-limited, and require fluid replacement like ORS, with a lactose-free glutamine-supplemented diet.
2. **Severe cases:**

a. **Nitazoxanide** is given to adults (500 mg twice daily for 3 days).

b. **Paromomycin** can be given as an alternate.

c. **Macrolide antibiotics** including spiramycin, azithromycin, and clarithromycin have some activity against *Cryptosporidium* species.

❖ **Prevention and Control**

1. Proper hand washing.
2. Use of submicron water filters.
3. Improve personal hygiene.

Isospora belli
(*Cystoisospora belli)*

- **Geographical Distribution:** It is more common in tropical and subtropical countries.
- **Habitat:** epithelial cells of small intestine.
- **Definitive host:** man
- **Intermediate host:** no intermediate host.
- **Diagnostic stage:** immature oocyst.
- **Infective stage:** mature oocyst.

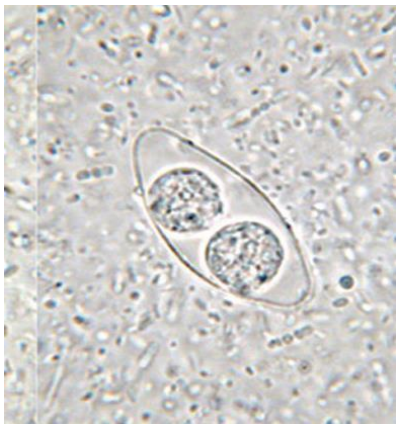
❖ **Morphology**

➢ **Oocysts:**

Size: 20–30 × 11– 19 mm.

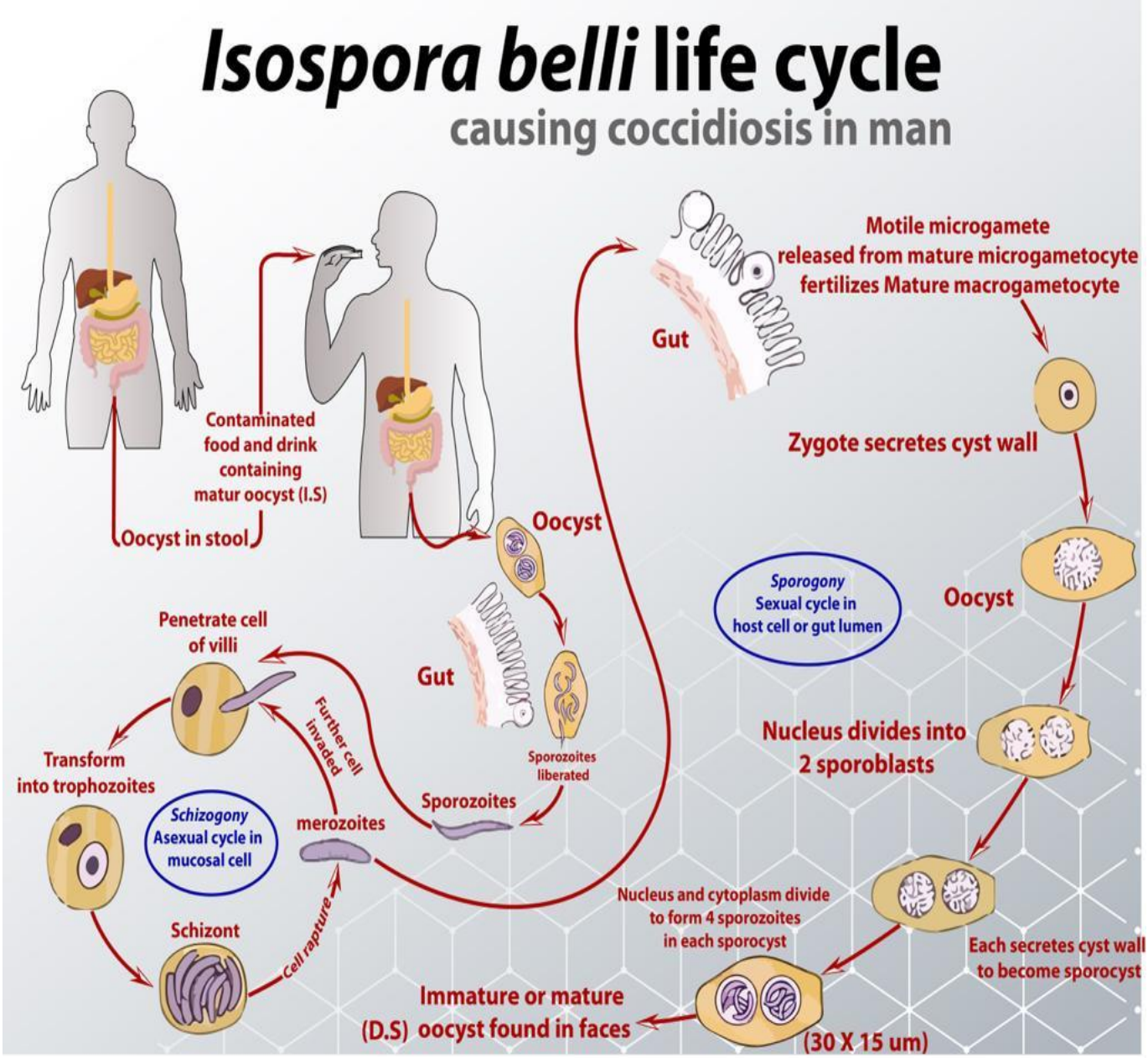
Shape: oval and surrounded by a smooth thin cyst wall.

Mature oocyst, contain two sporocysts. Each of them contains 4 sporozoites.



❖ **Life Cycle**

1. *Isospora belli* completes its entire life cycle only in one host.
2. **Humans get infection by ingestion of food or water contaminated with the mature oocysts.**
3. In the small intestine, the mature oocyst ruptures releasing 8 sporozoites which invade the intestinal epithelial cells.
4. In the intestinal epithelium, the sporozoites are transformed into trophozoites, which multiply asexually by schizogony to produce merozoites.
5. The merozoites invade adjacent enterocytes to repeat the asexual cycle.
6. Some of the trophozoites develop by sexual cycle (gametogony) in the cytoplasm of intestinal epithelium and transform into microgametocytes and macrogametocytes.
7. When fertilization occurs, a zygote is formed that secretes a cyst wall and then develops into an immature oocyst.
8. Unsporulated oocysts are excreted in faeces of infected person.
9. The excreted immature oocysts mature in the soil.



❖ **Clinical picture**

***Isospora belli* is one of the opportunistic parasites.**

1. Immune competent individuals are usually asymptomatic to this parasite's infection. But clinical symptoms such as mild self-limiting diarrhea, abdominal discomfort, and low-grade fever for approximately one week has been observed in some individuals.

2. Manifestations are more severe in immunocompromised patients. They can experience extreme diarrhea (watery without blood or pus) that can lead to weakness, anorexia, and weight loss. Other symptoms include abdominal pain, cramps, loss of appetite, nausea, vomiting, and fever, that can last from weeks to months

❖ **Diagnosis**

- **History and clinical picture**
- **Laboratory Diagnosis**

I. Microscopic examination

1. Stool examination

- The high fat content, fatty acid crystals or Charcot-Leyden crystals in stool may provide indirect evidence for presence of Isospora.
- Stool concentration techniques may be needed when wet mount preparation is negative.
- Many staining techniques can be used as modified Ziehl Neelsen stain or Kinyoun acid fast. (*Isospora* oocyst appears as large Pink-colored acid fast spheres more than 25 µm).

II. Molecular diagnosis using PCR of the stool sample.

III. Duodenal Aspirates (enterotest)

Examination of duodenal aspirate or enterotest can be performed when repeatedly negative stool examinations detect Isospora oocyst.

IV. Intestinal biopsy

❖ **Treatment**

1. In immunocompetent persons infection with Isospora is self-limiting, and no treatment is required.

2. In immunosuppressed patients with diarrhea, patients are treated orally with **co-trimoxazole** (160 mg trimethoprim/800 mg sulfamethoxazole /day for 10 days).
3. **Pyrimethamine** in a dose of 50-75 mg/day is given for patients with intolerance to sulfonamides.

   Relapses are common in people with AIDS and are treated with maintenance therapy with co-trimoxazole one tablet thrice a week.

❖ **Prevention and Control:**

1. Proper sewage disposal.
2. Proper personal hygiene.
3. Safe water supply (boiling or filtration of drinking water).
4. Proper washing of fruits and vegetables with clean water.
5. Health education.

Sarcocystis

A coccidian protozoa needs **two hosts**, the **definitive host** where the parasites multiply sexually in the intestine producing **oocysts**, and the **intermediate host** where the parasites multiply asexually producing muscle cysts **(sarcocysts)**.

Human infection includes:-

1- Intestinal sarcocystosis. **2- Muscular sarcocystosis.**

Intestinal Sarcocystosis

- Geographical distribution: Cosmopolitan.
- Causative organism:

1) ***Sarcocystis bovihominis:*** **D.H:** Man **&** **I.H:** Cattle.
2) ***Sarcocystis suihominis*:** **D.H:** Man **&** **I.H:** Pigs.

- Habitat: Intestinal mucosa of man.
- Morphological characters: Oocyst (disporocytic tetrazoic) in the stool.
- **Life cycle:**

➢ **Sexual cycle (gametogony and sporogony):** Occurs in **the small intestine of man (D.H)** due to **ingestion of sarcocysts** (containing bradyzoites) in improperly cooked meat of cattle or pigs. In the intestine, the cyst excysts and the bradyzoites invade the intestinal mucosa where they multiply sexually by gametogony and sporogony→ **oocyst** that sporulates→ **disporocystic tetrazoic** oocyst that passes with faeces. The wall of the oocyst is thin, so sporocysts are also detected in the faeces.

- **Asexual cycle (schizogony):** Occurs **in muscles of cattle or pigs (I.H)** due to **ingestion of oocysts or sporocysts** (released from oocysts). The sporozoites multiply asexually by schizogony in the vascular endothelial cells → the cell ruptures → the released merozoites invade the muscle fibers and develop into sarcocysts which are the infective stage to man.

❖ **Clinical pictures:** Infection in man is usually asymptomatic, and sometimes results in mild self-limited diarrhea and colic.

❖ **Diagnosis:** Stool examination for oocysts or sporocysts by using concentration methods.

❖ **Treatment:** Symptomatic treatment is required. No specific treatment for intestinal sarcocystosis.

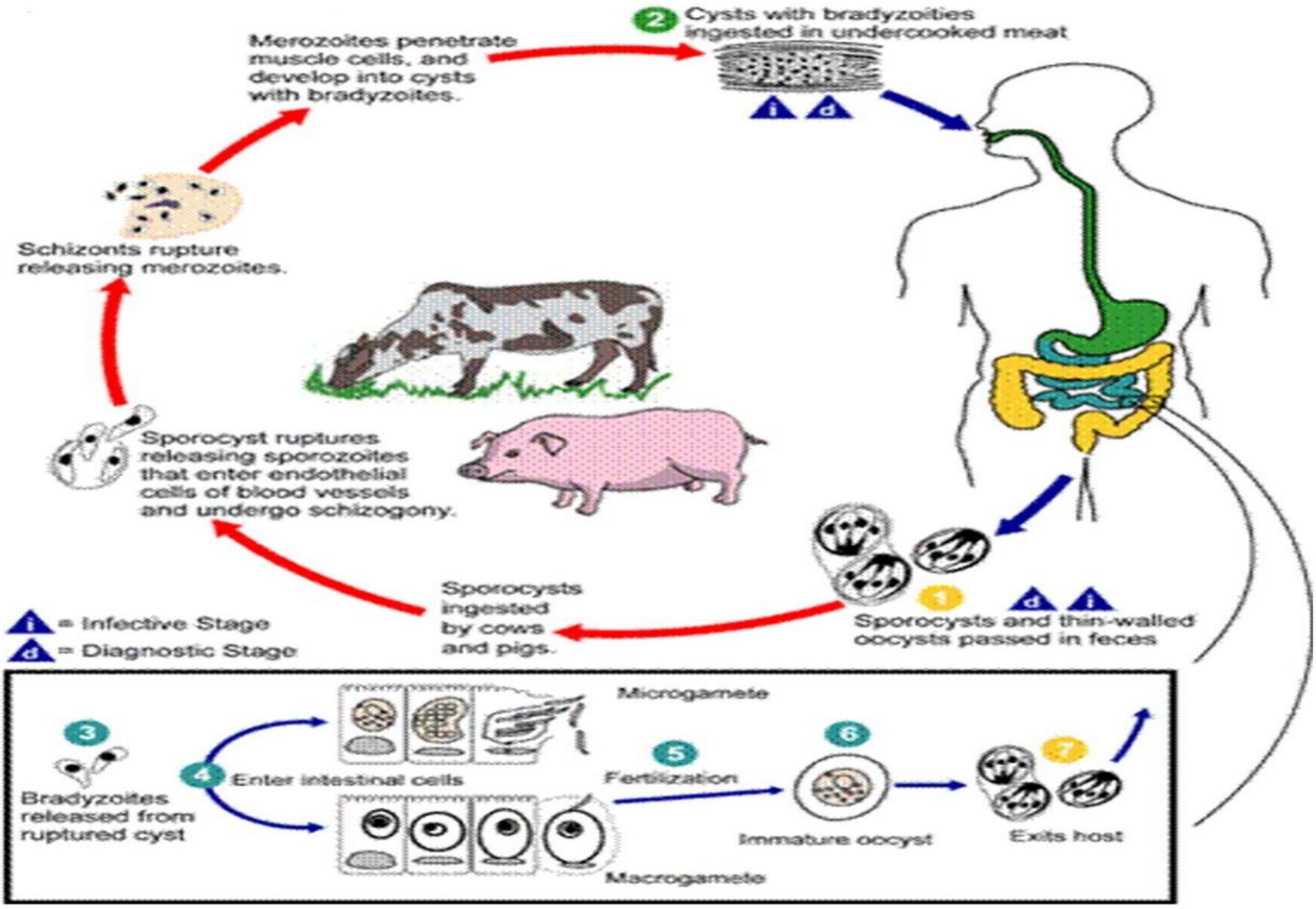
❖ **Prevention and Control: -**

1) Treatment of patient.
2) Proper cooking of meat (beef- pork) or deep freezing (-20).
3) Proper sewage disposal to prevent infection of cattl

Muscular sarcocystosis

- **Geographical distribution:** Cosmopolitan.
- **Causative organism**: ***Sarcocystis lendermanni***
- **D.H:** Dogs, cats, foxes and wolfs.
- **I.H:** Man.
- **Habitat:** Muscles.
- **Morphological characters:** Sarcocysts in muscles.
- **Life cycle:**

- **Sexual cycle (gametogony and sporogony):** Occurs **in the small intestine of carnivores (D.H.)** due to **ingestion of sarcocysts** (containing bradyzoites) with meat. The bradyzoites in the cyst invade the intestinal mucosa and multiply by gametogony and sporogony→end by the formation of an **oocyst** that passes with feces **(infective stage to man)**.
- **Asexual cycle (schizogony):** Occurs **in muscles of man (I.H)** due to **ingestion of oocysts or sporocysts** (released from oocysts). The sporozoites multiply asexually by schizogony in the vascular endothelial cells → the cell rupture → the released merozoites invade the muscle fibers and develop into **sarcocysts**.



❖ **Clinical pictures:** Usually asymptomatic.

❖ **Diagnosis: -**

1) Muscle biopsy.

2) Serological tests.

❖ **Treatment:** No specific treatment for muscular sarcocystosis. Corticosteroids may be required.

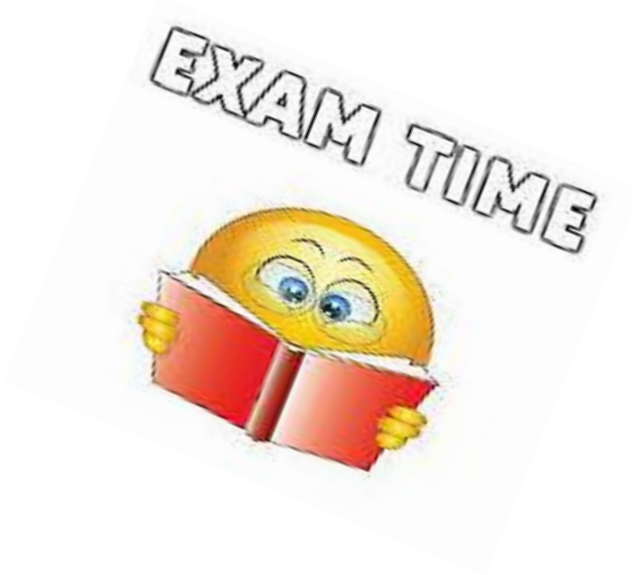
❖ **Prevention and Control:**

1) Carnivores should be excluded from houses.

2) Uncooked meat should never feed to carnivores.

3) Protect food and drink from contamination with animal stool.

4) Fly control



1. **Which of the following parasites can be transmitted by flies?**

a. Ancylostoma duodenale.

b. Capillaria philippinensis.

c. Entamoeba histolytica.

d. Fasciola gigantica.

e. *Strongyloides stercoralis*

2. **A 45-year-old male patient attended the clinic of internal medicine complaining of abdominal colic and diarrhea. His stool examination revealed the shown figure. Which of the following organisms will be your diagnosis?**

a. Balantidium coli.

b. Entamoeba histolytica.

c. Enterobius vermicularis.

d. Cryptospordium.

e. Trichuris trichiura.

3- **Which one of the following complications can occur with the shown parasite?**

a. Hematemesis.

b. Intestinal obstruction

c. Liver abscess.

d. Perianal itching.

e. Pernicious anemia.

Laboratory diagnosis of parasitic infection

Diagnosis of parasitic infections could be reached through:

I- History.

II- Clinical picture.

III- Laboratory investigations:

1- Direct methods (by the naked eye or by microscopy):

- To detect the whole parasite, a part of the parasite, an egg or larva.

2- Indirect methods: serological and molecular methods.

I) Microscopy

Advantages of microscopy:

- Important diagnostic tool, especially in low-income communities.
- Cheap.
- Simple.

Limitations of microscopy:

- Low sensitivity.
- Requires long-timed preparations of the specimens.
- Requires examination of multiple specimens.
- Impossible to diagnose pre-patent parasitic infection.
- It cannot distinguish species within many parasite genera e.g., *Cryptosporidium*.

❖ Stool examination

<u>Precautions for propre stool examination:</u>

- Avoid contaminating the specimens with water or urine by collecting feces in clean, wide-mouth containers with a tight-fitting cover.
- Three stool specimens should be submitted every other day.
- For the recovery of motile trophozoites, fresh specimens are required.
- Liquid specimens should be inspected within 30 min of passage.

Contents

- Semi-formed specimens may contain protozoan trophozoites and cysts and should be examined within 1 h of passage.
- Formed specimens can be examined at any time within 24 h after passage.
- Fecal samples should be placed in fixatives e.g., Formalin (5% or 10%), Schaudinn's fluid or Polyvinyl alcohol (PVA).

A) Macroscopic examination of the stool:

Stool specimens are examined by naked eye for the presence of scolices and proglottids of cestodes or adult worms. It also includes comments on

1. Smell.
2. Ph.
3. Consistency
4. Contents: like mucous, tissues or occult blood.

B) Microscopic Examination (Ova and Parasite Examination):

Consists of three separate techniques:

a. Direct wet smear.
b. Concentration methods.
c. Permanent stained smear.

a. Steps of direct wet smear:

1. Put 1 drop of iodine on the right side of the slide and 1 drop of 0.85% NaCl on the left.
2. Emulsify 2 mg of the sample in the saline and iodine preparations.
3. Place a coverslip on each suspension and examine under the microscope.

b. Concentration of fecal samples (Sedimentation and Flotation):

- Fecal concentration is a routine procedure as it allows the detection of small numbers of organisms which could be missed by using a direct wet smear.

- There are two types of concentration procedures, sedimentation, and flotation.

Contents

(1) Concentration with Sedimentation:

Formalin-ethyl acetate sedimentation concentration:

- It facilitates the recovery of protozoa, eggs, and larvae. However, the preparation has more debris than is found in the flotation method.
- The specimen may be fresh or formalinized stool.

Steps:

1. Put 4 gm of fresh stool in 10 ml of 10% formalin.
2. Mix thoroughly and leave the mixture for a minimum of 30 min for fixation.
3. Strain through wet gauze into a conical 15-ml centrifuge tube.
4. Add 10% formalin in the tube, and centrifuge for 10 min at 500 X *g*.
5. Discard the supernatant, and re-suspend the sediment in formalin, and centrifuge for 10 min at 500 X *g*.
6. Put 5 ml of ethyl acetate, close the tube, and shake it vigorously for at least 30 seconds.
7. After 15-30 seconds, Centrifuge for 10 min at 500X *g*. 4 layers will be formed.

- Sediment in the bottom of the tube.
- Formalin layer.
- Layer of fecal debris.
- Layer of ethyl acetate at the top.

8. Free the plug of debris and discard all the supernatant fluid.
9. Mix the sediment and take a drop into a slide and examine with the 10X objective and then with the 40X objective lens of the microscope.

(2) Concentration with flotation (Zinc sulfate flotation):

- The use of a liquid with a high specific gravity enables the separation of protozoan cysts, coccidian oocysts, particular helminth eggs and larvae. Parasites are found in the surface film.
- However, some helminth eggs (operculated eggs and Ascaris eggs), do not concentrate well in the flotation approach.
- This technique produces cleaner preparation than does the sedimentation procedure.

Contents

- The surface film and the sediment must be inspected to ensure the discovery of all potential organisms.

Steps:

1. Place 4 gm of fresh feces into a round-bottomed tube containing 10 ml of 5 or 10% formalin.
2. Thoroughly mix the formalin and feces. Allow the mixture to stand for at least 30 minutes for fixation. Stir the stool-formalin mixture if the sample is already fixed in 10% or 5% formalin.
3. Strain through wet gauze into a 15-ml centrifuge tube to give 0.5 to 1 ml of sediment.
4. Add 0.85% NaCl and centrifuge for 10 minutes at 500 X g.
5. Re-suspend the sediment at the bottom of the tube in 1-2 ml of zinc sulfate after discarding the supernatant fluid.
6. Fill the tube with zinc sulfate until the tube is nearly full.
7. Centrifuge for 2 min at 500 X *g*. Then, two layers will result: a small amount of sediment in the bottom of the tube, and a layer of zinc sulfate.
8. Use a Pasteur pipette to take 1-2 drops from the surface layer, place them on a microscope slide, and then inspect without removing the tube from the centrifuge.
9. The surface film contains the protozoan cysts, and the sediment contains some operculated and/or heavy eggs.

c. Permanent staining of stool samples:

Permanent stain is recommended for every stool sample.

There are several staining techniques available:

1) Trichrome stain:

- It is a rapid and simple procedure which produces uniformly well stained smears of the intestinal protozoa, human cells, or yeast cells.
- *Cryptosporidium* and *Cyclospora* oocysts are not recognized on a trichrome-stained smear.

2) Specialized Stains for Coccidia (*Cryptosporidium*, Isospora, and Cyclospora Species): Modified Ziehl-Neelsen acid-fast stain:

- Besides stool samples, other types of clinical specimens, such as duodenal fluid, bile, and pulmonary specimens may be stained by this method.
- The oocysts of *Cryptosporidium* stain pink to red to deep purple. The background stains blue.

F) Culture of Larval-Stage of Nematodes

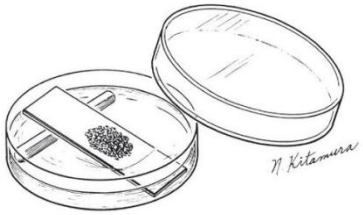
Culture of fresh feces for larvae is useful to:

a. Detect light infections with hookworm, *S. stercoralis*, and *Trichostrongylus*.
b. Distinguish between S. *stercoralis* and hookworm based on rhabditiform larval morphology.
c. Permit larval growth to reach the filariform stage so that further differentiation can occur easily.

1) Harada-Mori Filter Paper Strip Culture

a) Put 0.5 to 1 gm of **fresh stool** sample in the middle of a short strip of filter paper.
b) Place the filter paper strip into a 15-ml conical centrifuge tube that is already filled with 3 to 4 ml of distilled water. The water should be about half an inch below the fecal area.
c) Keep the tube in an upright position in a rack at 25 to 28 °C. Frequently add distilled water to keep the level at its initial value.
d) Leave the tube for 10 days, check it every day by taking a few drops of fluid out of the bottom and Prepare a smear on a glass slide, cover the slide with a coverslip, and examine under the microscope.

2) Filter Paper/Slant Culture Technique (Petri Dish)



a. Cut a strip of filter paper in half, spread 2 gm of fresh feces on the center of the strip.
b. Put the strip on a glass slide. By supporting the slide on a glass rod, position the slide inclined at one end of the petri dish.

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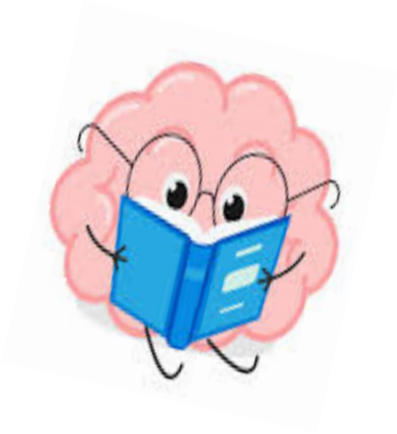
c. Fill the petri dish with water so that at least the bottom quarter of the slide is immersed. Keep the dish covered at a temperature of 25 to 28 °C. Add water as necessary to keep the initial level.
d. Examine daily (for 10 days) by withdrawing a small amount of fluid and placing it on a glass slide. Cover with a coverslip and examine under a microscope.

3) Baermann's Technique

a. A funnel device is used.
b. A fresh fecal sample is put in a wire mesh having multiple layers of gauze and in contact with tap water.
c. The active larvae will migrate through the layers into the water and settle to the bottom of the funnel.
d. From there, the larvae can be collected and examined.

Contents



Please, visit the following website to answer a formative exam on GIT module:

https://docs.google.com/forms/d/1DV88EkUYeMs4fFSfkbpB3oxK5Qv0yfiV1uijlprWYlY/edit

References

- **Esteban JG, Muñoz-Antoli C, Toledo R and R. Ash L. (2014):** Digenetic Trematodes, chapter: Diagnosis of Human Trematode Infections, pp 293:327. Access provided by Egyptian Knowledge Bank. https://link.springer.com/chapter/10.1007/978-1-4939-0915-5_9

- **Mahmud R, Lian Lim YA and Amir A. (2014):** Medical Parasitology A Textbook, (eBook). Access provided by Egyptian Knowledge Bank. https://link.springer.com/book/10.1007/978-3-319-68795-7

- **Shaughnessy M, Frosch A, and Stauffer W. (2020):** Refugee Health Care, An Essential Medical Guide. 2nd ed. Parasitic Infections Chapter, pp 75- 96. Access provided by Egyptian Knowledge Bank. https://link.springer.com/chapter/10.1007/978-3-030-47668-7_6?fromPaywallRec=false

- **Garcia LS (2007):** Diagnostic medical parasitology, 5th ed., 2007, ASM press, American Society for Microbiology, Washington, D.C. https://archive.org/details/diagnosticmedica0000garc/page/n1229/mode/2up

- **Farrar J, Hotez PJ, Junghanss T, Kang G, Lalloo, D and White NJ. (2014):** Manson's Tropical Diseases: Twenty- third ed., Saunders, an imprint of Elsevier Inc. http://doi.org/10.1016/C2010-0-66223-7

- **Murray PR, Rosenthal KS and Pfaller MA. (2016):** Medical microbiology: 8th ed., 2016, an imprint of Elsevier Inc. https://books.google.com.eg/books?id=jqeCgAAQBAJ&hl=ar&source=gbs_navlinks_s.

• **John DT and Petri WA (2006):** Medical parasitology: 9th ed., Saunders, an imprint of Elsevier Inc. https://shop.elsevier.com/books/markell-and-voges-medical-parasitology/martin/978-0-7216-4793-7

• **Satoskar AR, Simon GL, Hotez PJ and Tsuji M. (2009):** Medical parasitology: Landes Bioscience, Austin, Taxas, USA. https://doi.org/10.1201/9781498713672

• **Garcia LS. (2021):** Practical guide to diagnostic parasitology: 3rd ed., ASM press, Washington, DC, American Society for Microbiology. https://openlibrary.org/books/OL30178847M/Practical_Guide_to_Diagnostic_Parasitology_3rd_Edition_EBook(ePDF).

• **Gillespie S and Pearson RD (2001):** Principles and Practice of Clinical Parasitology: John Wiley & Sons Ltd, Baffins Lane, Chichester, West Sussex PO19 1UD, England. https://doi.org/10.1002/0470842504.