



Trematoda

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Medical Parasitology II

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Lecture 4

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***Schistosoma* sp.**

Trematoda

They are leaf-shaped unsegmented, flat and broad helminths (hence the name fluke, from the Anglo-Saxon word *floe* meaning flatfish). The name trematode comes from their having large prominent suckers with a hole in the middle (Greek *trema*: hole, *eidos*: appearance)

Classification Based on Habitat

Based on habitat, trematodes can be classified as (Table):

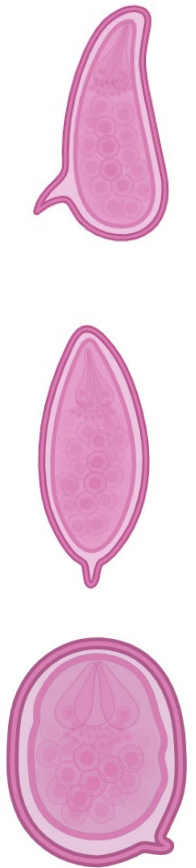
- Blood flukes
- Liver flukes
- Intestinal flukes
- Lung flukes.

Schistosoma sp.

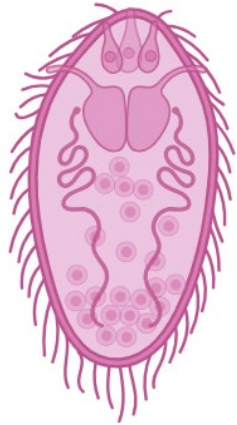
- Blood flukes, cylindrical in shape (not flat like other flukes).
- Dioecious (male and female are separate).
- Eggs with characteristic spines (species-specific).
- Life cycle involves snails (intermediate host) and humans (definitive host).
- Infection by skin penetration of cercariae in water.
- Adults live in blood vessels (mesenteric veins or bladder plexus).
- Cause schistosomiasis due to tissue reaction to eggs.

Schistosoma sp stages

Egg



Miracidia



Cercariae



Adult



Schistosoma Haematobium

Epidemiology

This vesical blood fluke, formerly known as *bilharzia haematobium*, has been endemic in the Nile valley in Egypt

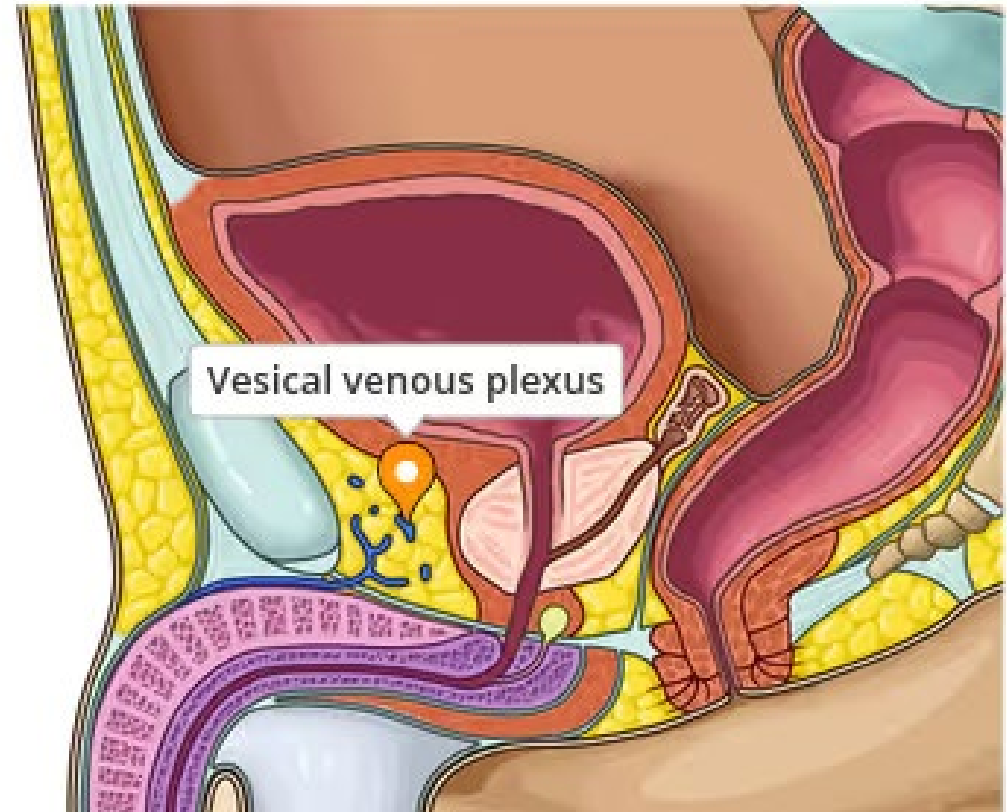
Although maximally entrenched in the Nile valley, *S. haematobium* is also endemic in most parts of Africa and in West Asia.

About 200 million persons are at a risk of infection and 90 million are infected by *S. haematobium* globally.



Habitat

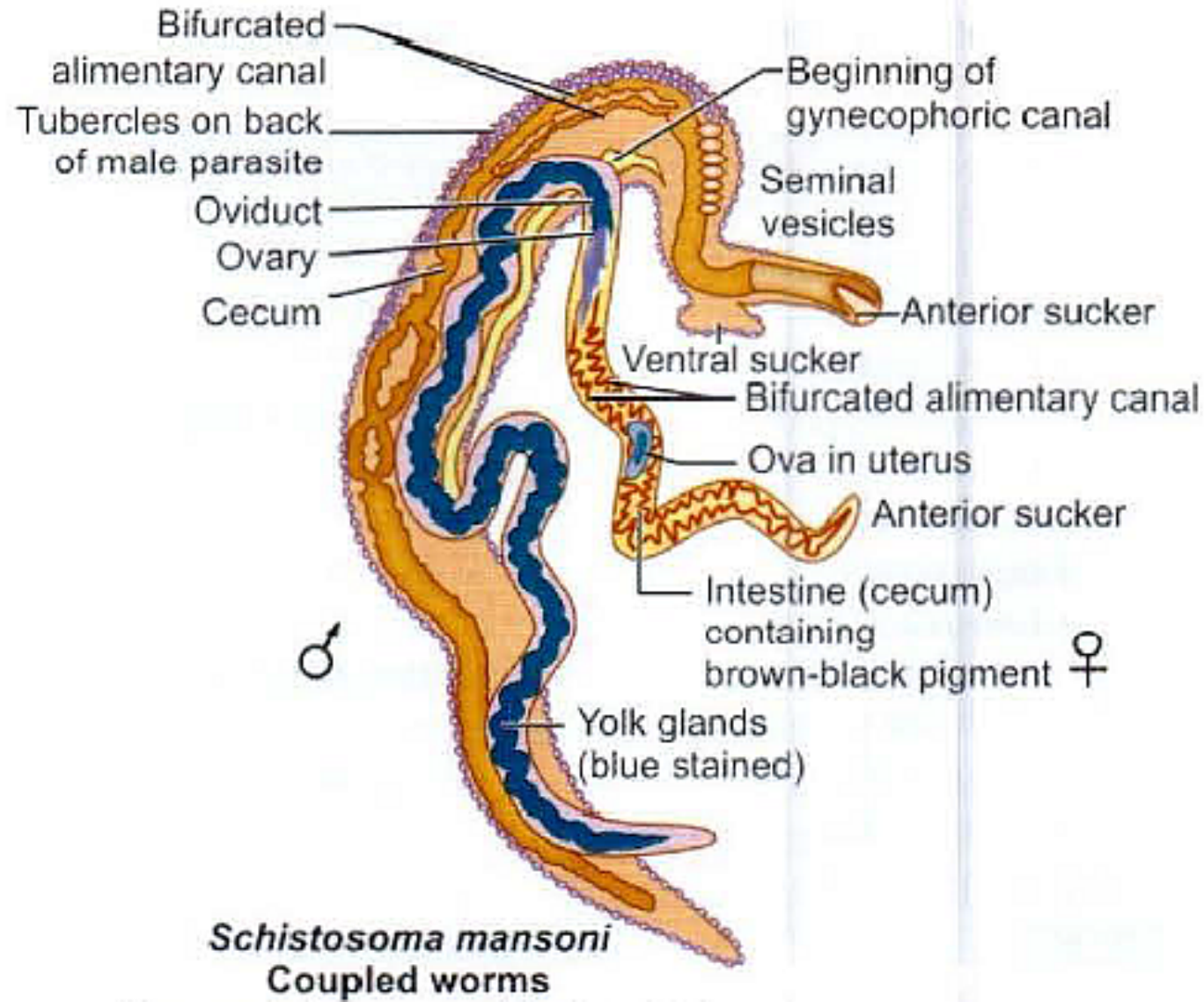
The adult worms live in the vesical and pelvic plexuses of veins.



Morphology

Adult worm

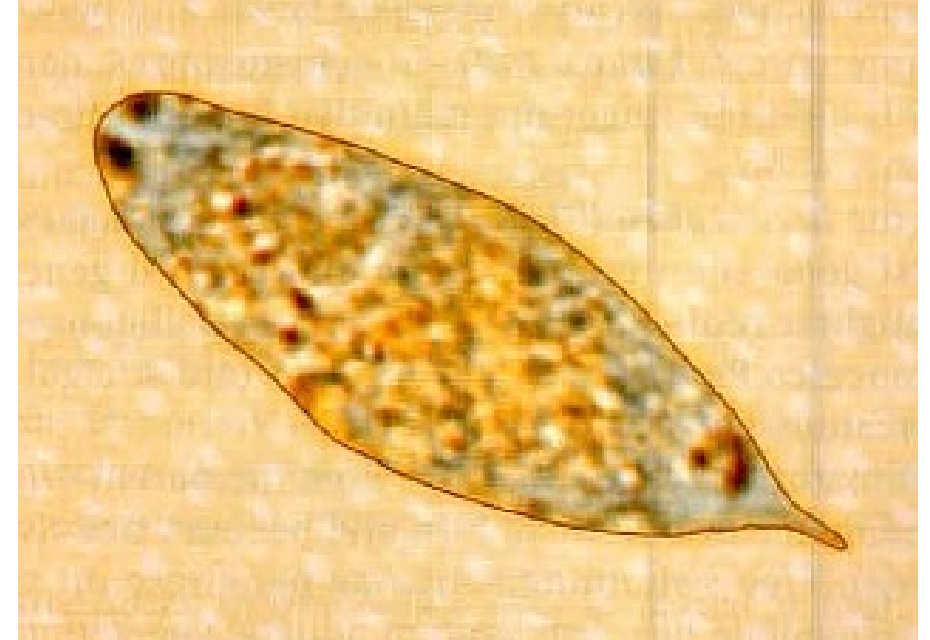
- The male is 15 mm long by 0.9 mm thick and covered by a thick tuberculate tegument.
- It has two muscular suckers: (1) the oral sucker being small and (2) the ventral sucker is large and prominent. Beginning immediately behind the ventral sucker and extending to the caudal end is the gynecophoric canal, in which the female worm is held (Fig. 3).
- the adult female is long and slender (20 mm by 0.25 mm).
- the gravid worm contains 20-30 eggs in its uterus at one time and may pass up to 300 eggs a day.



Morphology

Egg:

The eggs are elongated, brownish yellow (about $150\text{ }\mu\text{m}$ by $50\text{ }\mu\text{m}$) and nonoperculated. the eggs have characteristic terminal spine at one pole.



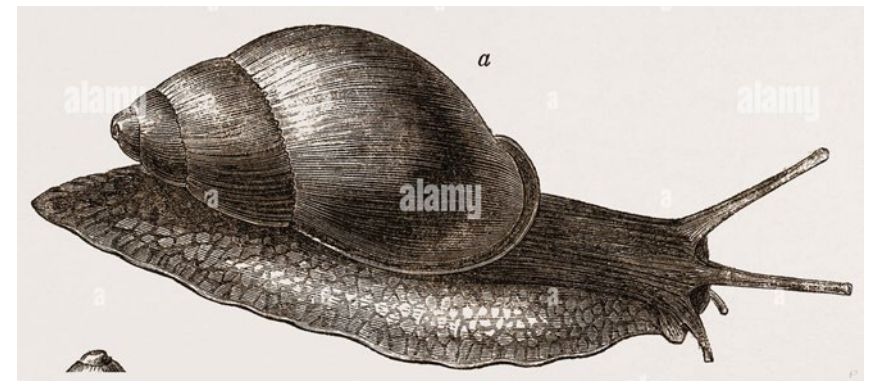
Life cycle

S. haematobium passes its life cycle in two hosts:

1. Definitive host: Humans are the only natural definitive hosts. o animal reservoir is known.

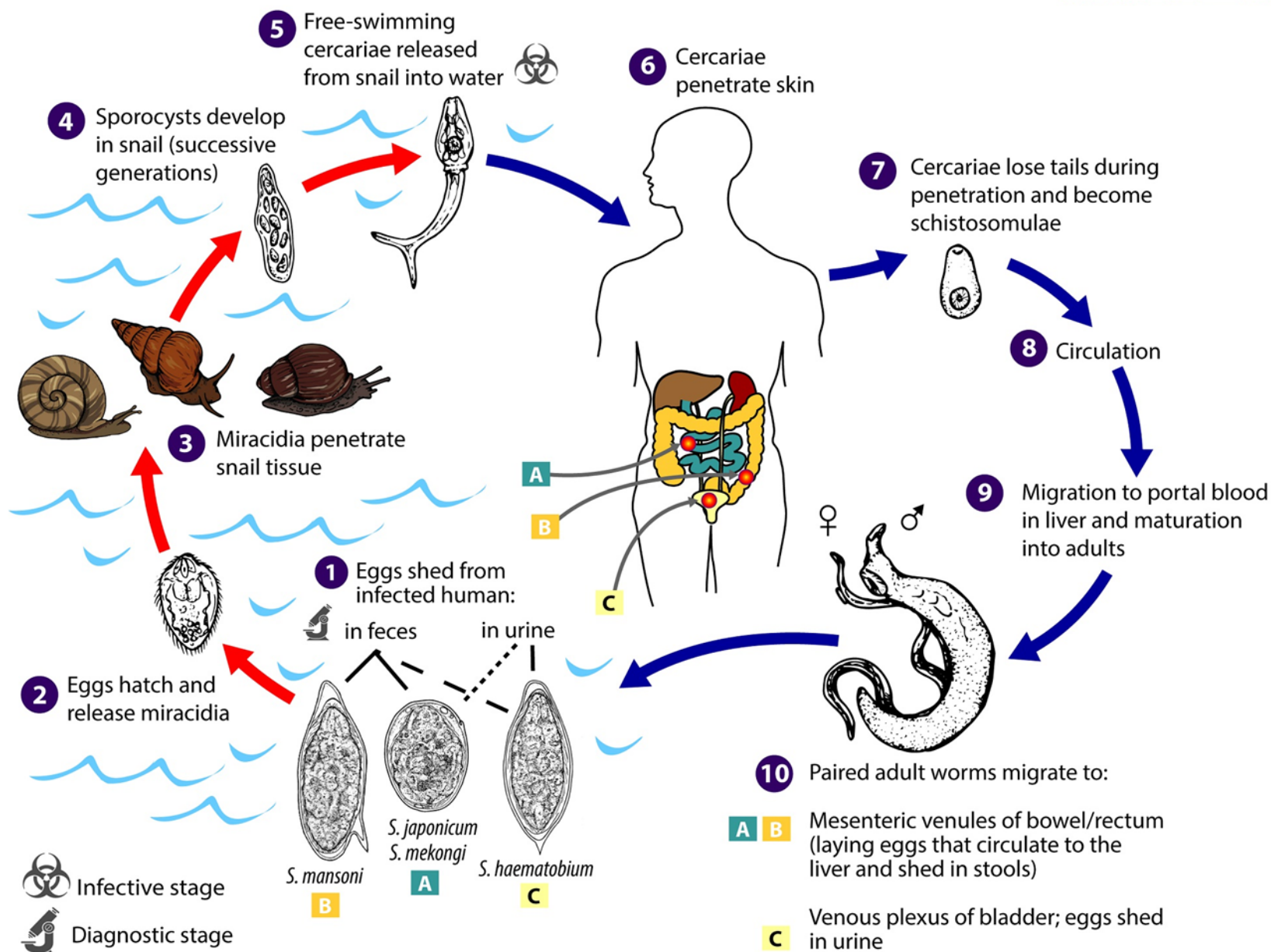


2. Intermediate host: Freshwater snails (snail of the genus *Bulinus*).



Life Cycle

- Eggs with terminal spine are passed in urine.
- In freshwater, eggs hatch → release miracidia.
- Miracidia infect *Bulinus* snails (intermediate host).
- Develop into sporocysts → cercariae.
- Cercariae leave snail and swim freely.
- Infect humans by penetrating skin in contaminated water.
- Transform into schistosomula → migrate via blood to venous plexus of the bladder.
- Mature into adult worms (male & female) in bladder veins → produce eggs, continuing the cycle.



Sign and Symptoms

Acute Schistosomiasis:

- Skin penetration causes itching and rash (swimmer's itch), more severe with nonhuman species.
- Schistosomule migration may lead to allergic or toxic reactions, cough, and mild fever.

Sign and Symptoms

Chronic Schistosomiasis:

- Eggs in bladder cause painless hematuria, dysuria, and proteinuria, especially in children.
- Bladder mucosa becomes inflamed, ulcerated, thickened with granulomas (sandy patches), fibrosis, and calcification.
- Complications include hydronephrosis, hydroureter, bladder carcinoma, chronic cystitis, and stone formation.
- Other organs that may be affected include lungs, CNS, skin, and genital organs.

Laboratory diagnosis

1. Urine microscopy:

The eggs with characteristic terminal spines can be demonstrated by microscopic examination

2. Histopathology:

Schistosome infection may also be diagnosed by demonstrating its eggs in bladder mucosa! biopsy and rectal biopsy

3. Serology:

Detection of antigen or antibody by ELISA

4. Ultrasonography (USG):

is also useful in diagnosing *S. haematobium* infection. USG may show hydroureter and hydronephrosis

Treatment

Treatment

- **Praziquantel** (40-60 mg per kg in divided doses in a single day) is the drug of choice.
- **Metriphonate** is the alternative drug of choice in schistosomiasis due to *S. haematobium* (7.5 mg/kg weekly for 3 weeks).



Schistosoma Mansoni

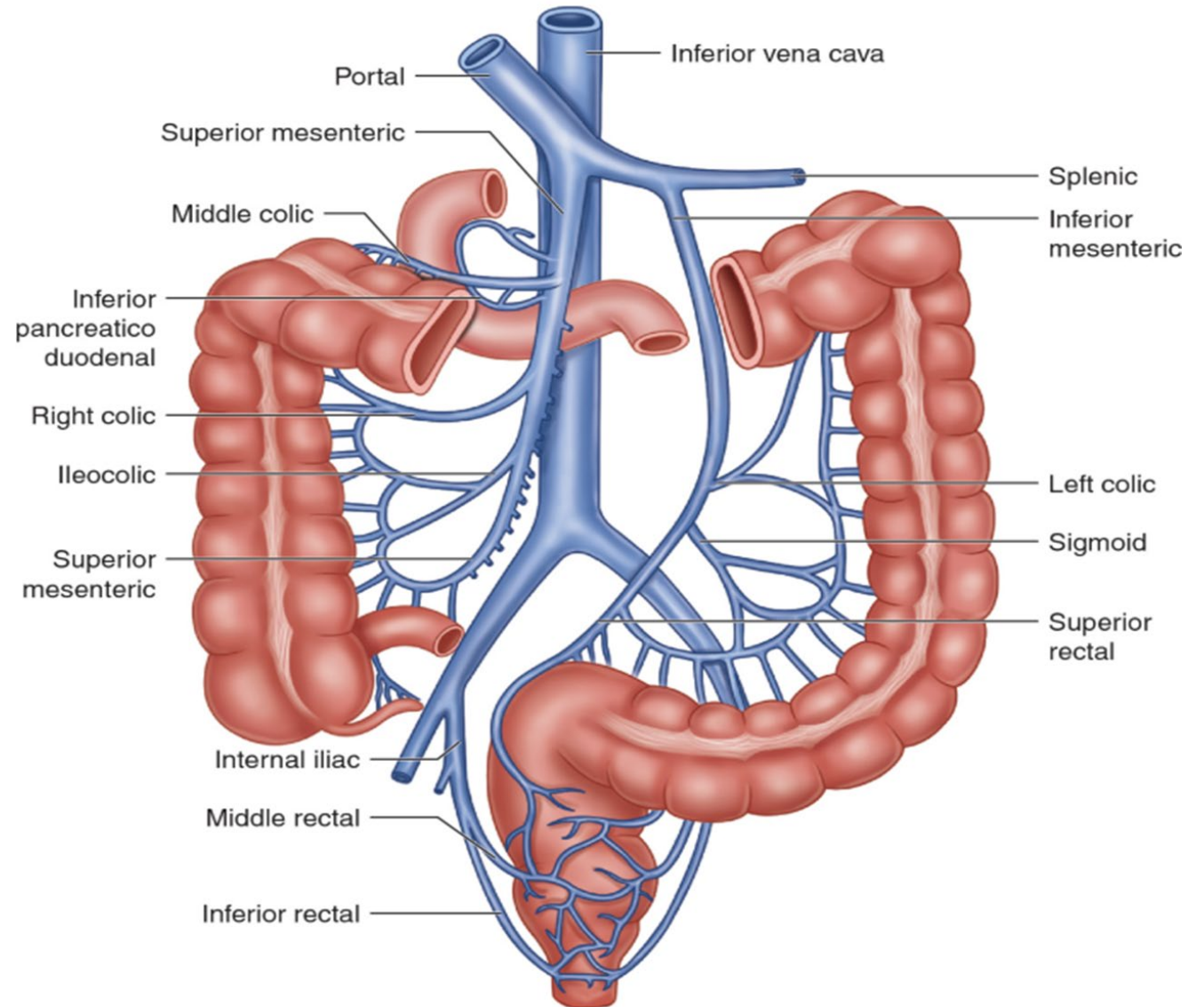
Epidemiology

It is widely distributed in Africa, South America and the Caribbean islands.



Habitat

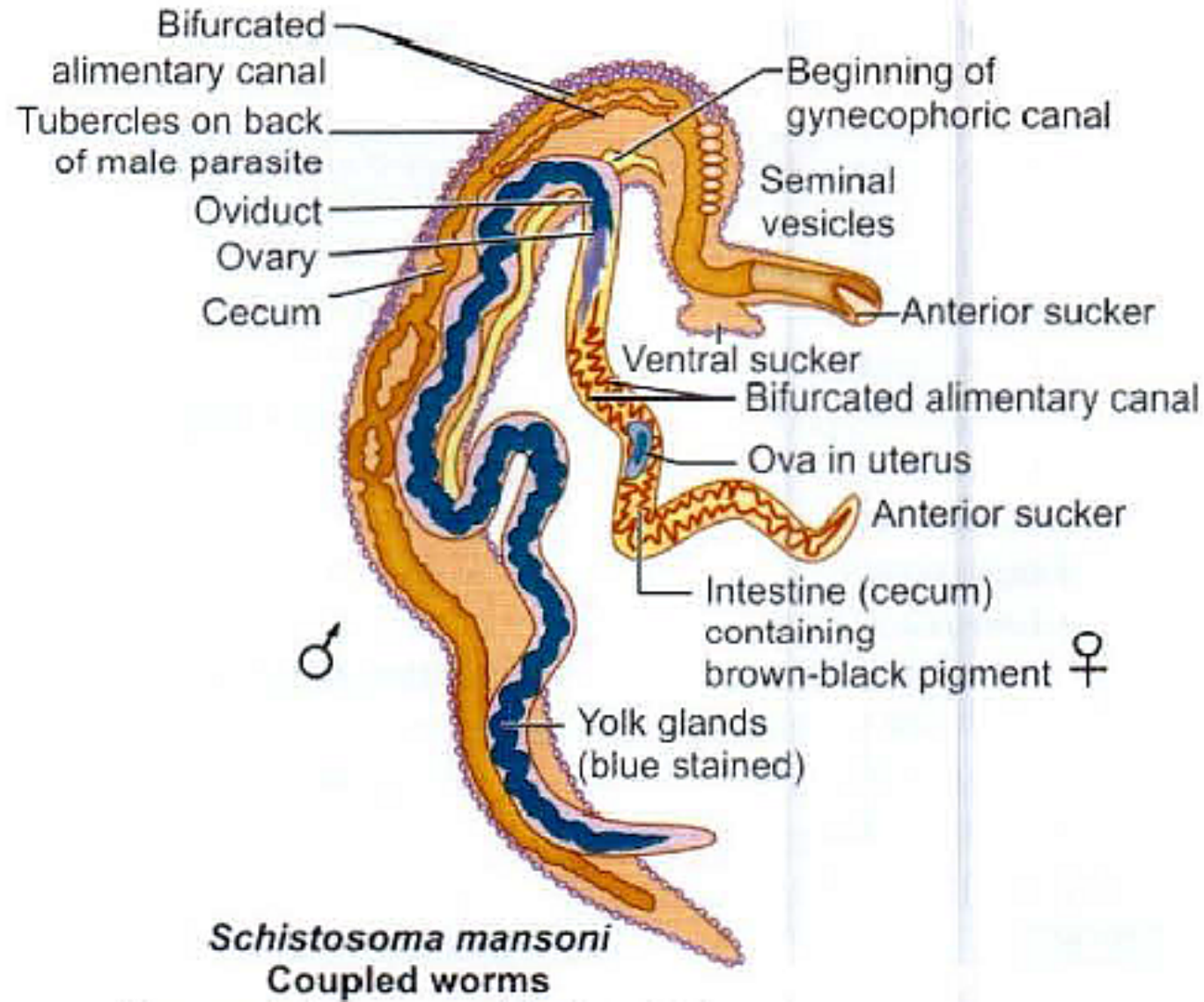
Adult worm lives in the inferior mesenteric vein.



Morphology

Adult worm

- *S. mansoni* resembles *S. haematobium* in morphology and life cycle, except:
- The adult worms are smaller and their integuments studded with prominent coarse tubercles.
- The prepatent period (the interval between cercarial penetration and the beginning of egg laying) is 4-5 weeks.



Morphology

Egg:

The egg has a characteristic lateral spine, more near to the rounded posterior end. The eggs are nonoperculated and yellowish brown.



Life cycle

Definitive host: Humans are the only natural definitive hosts, though in endemic areas monkeys and baboons have also been found infected.

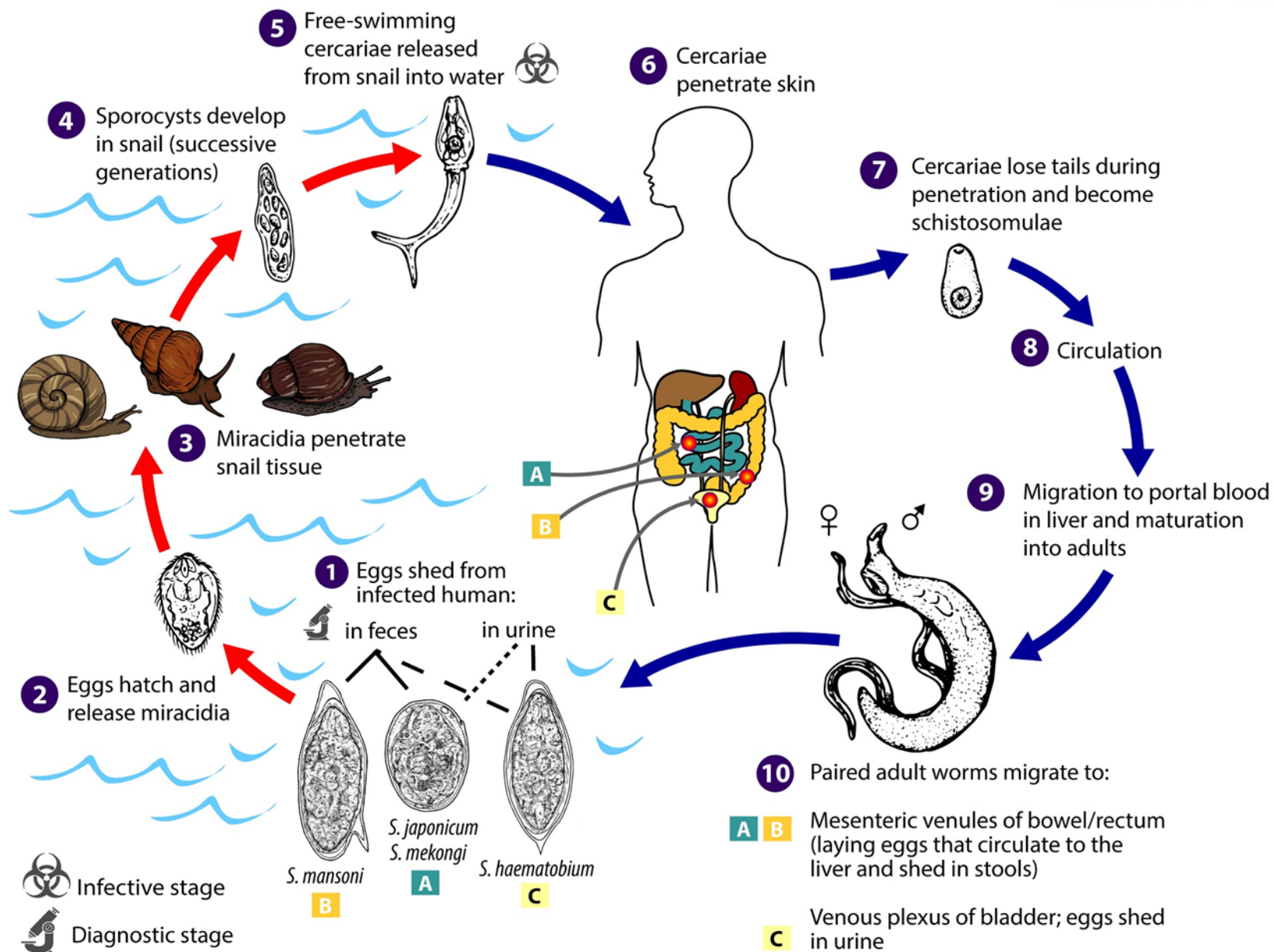


Intermediate host: freshwater snails of the genus *Biomphalaria*.



Life Cycle

- Eggs with lateral spine are passed in feces.
- In freshwater, eggs hatch releasing miracidia.
- Miracidia infect Biomphalaria snails (intermediate host). Develop into sporocysts → cercariae.
- Cercariae leave the snail and swim freely.
- Infection occurs when cercariae penetrate human skin in contaminated water.
- They transform into schistosomula, migrate via circulation to mesenteric veins of large intestine.
- Mature into adult worms, produce eggs, and cycle repeats.



Sign and Symptoms

Cercarial dermatitis:

Following skin penetration by cercariae: A pruritic rash called as cercarial dermatitis or swimmers itch may develop locally. It is a self-limiting disease.

Sign and Symptoms

Katayama Fever (Acute Schistosomiasis):

- Occurs 4–8 weeks after infection when egg laying begins.
- Due to immune complex reaction from high worm load.
- Symptoms: fever, rash, joint pain, hepatosplenomegaly, lymphadenopathy, eosinophilia.

Intestinal Schistosomiasis (Chronic):

- Egg deposition in intestine causes abdominal pain and bloody diarrhea.
- Leads to microabscesses, granulomas, fibrosis, mainly in colon and rectum.
- Eggs carried to liver cause hepatosplenomegaly, periportal fibrosis, and portal hypertension.
- Complications: gastrointestinal bleeding.

Laboratory diagnosis

1. Stool microscopy:

Eggs with lateral spines may be demonstrated microscopically in stools.

2. Rectal biopsy:

Proctoscopic biopsy of rectal mucosa may reveal eggs when examined as fresh squash preparation between two slides.

3. Serology:

Detection of antigen or antibody by ELISA

4. Imaging:

Ultrasonography is useful to detect hepatosplenomegaly and periportal fibrosis.

Treatment

- **Praziquantel** (single oral dose 40 mg/kg) is the drug of choice.
- **Oxamniquine** (single oral dose 15 mg/ kg) is also effective. It damages the tegument of male worm and thereby, makes the worm more susceptible to the action of the immune



Schistosoma Japonicum

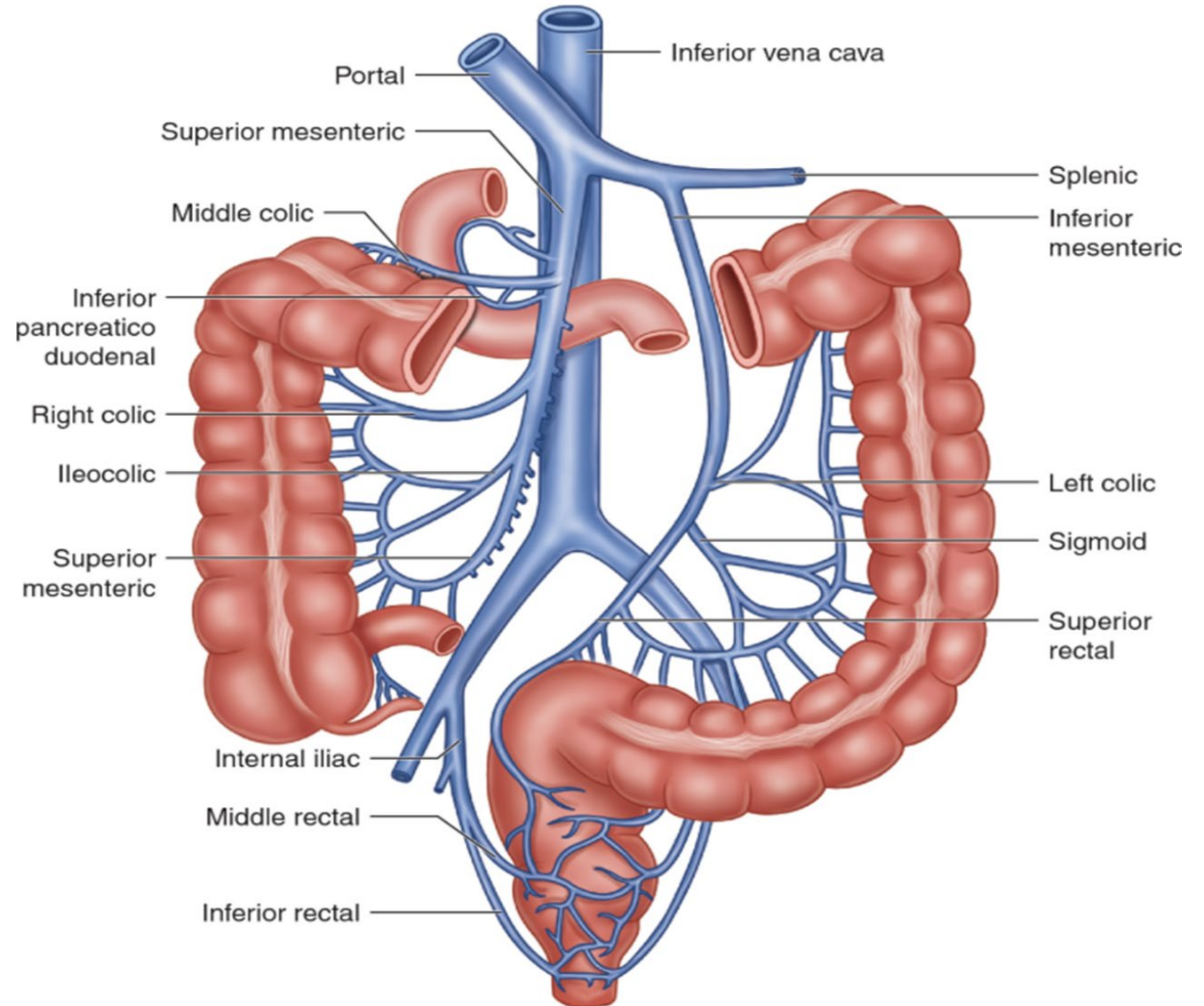
Epidemiology

S. japonicum is found in the Far East, Japan, China, Taiwan, Philippines and Sulawesi.



Habitat

The adult worms are seen typically in the venules of the superior mesenteric vein draining the ileocecal region.

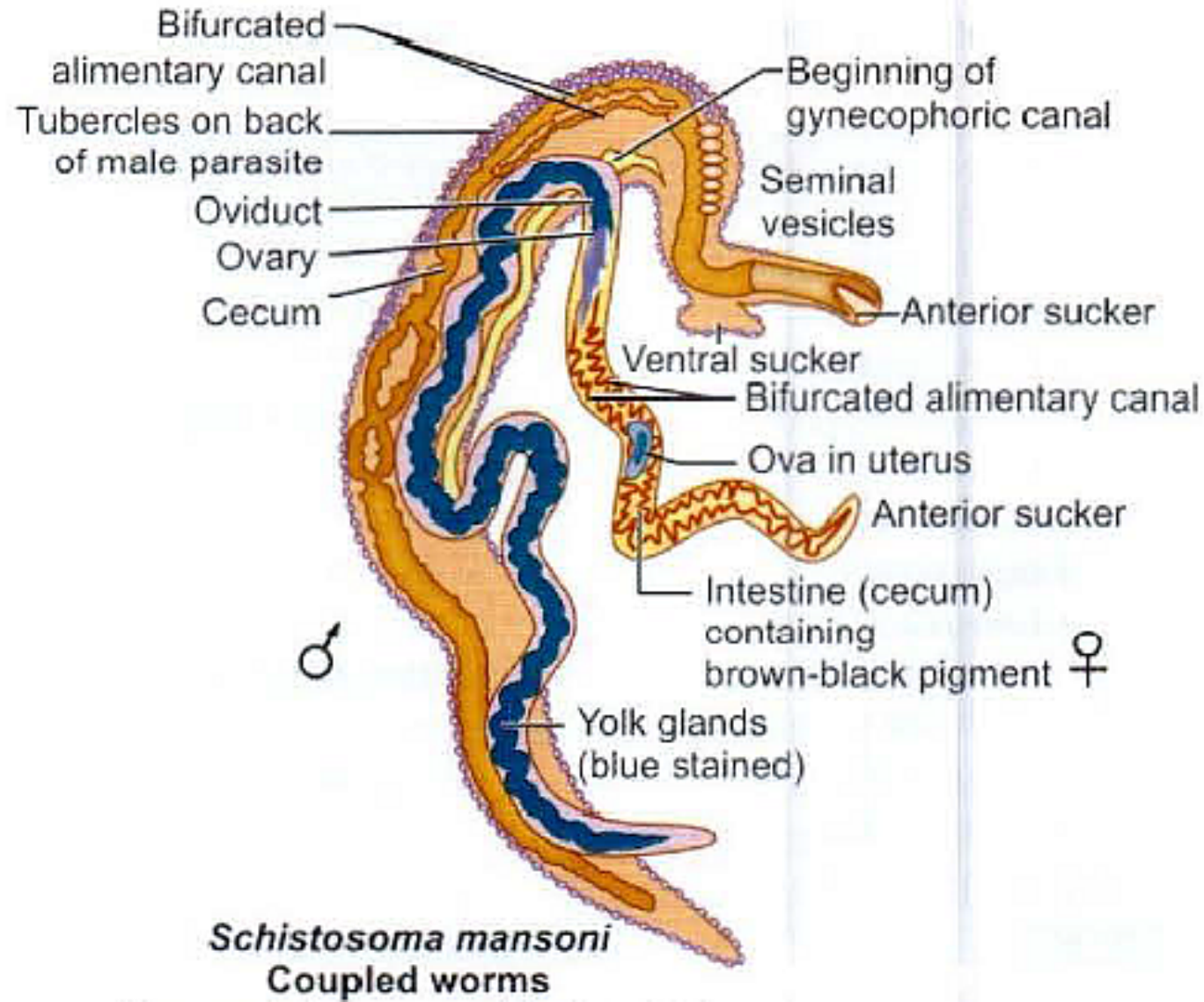


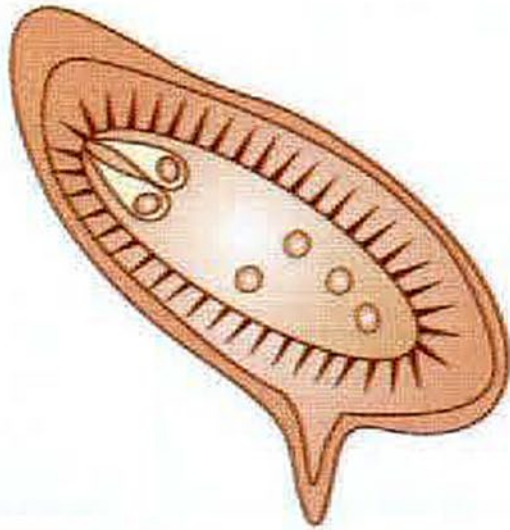
Morphology

Adult worm

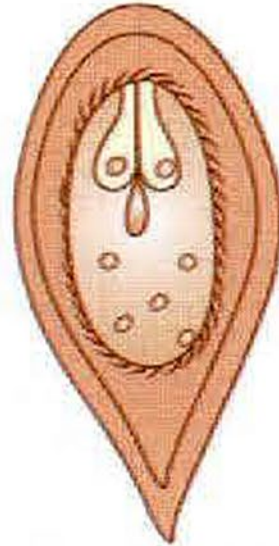
- Morphologically, they are similar to the schistosomes described earlier
- The prepatent period is 4-5 weeks.
- The eggs are smaller and more spherical than those of *S. haematobium* and *S. mansoni*. The egg has no spine but shows a lateral small rudimentary knob.



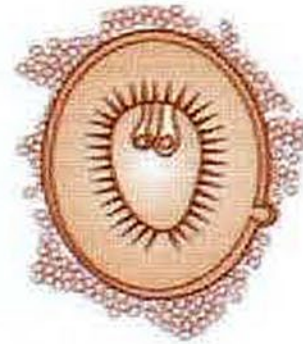




S. mansoni
Ova with a lateral spine
(obtained from stool)



S. haematobium
Ova with a terminal spine
(obtained from urine)



S. japonicum
Ova with a lateral knob
(obtained from stool)
Note: The characteristic surround
of tissue particles

Morphology

Egg:

The egg has a characteristic lateral spine, more near to the rounded posterior end. The eggs are nonoperculated and yellowish brown.



Life cycle

Definitive host: Man is the definitive host but in endemic

areas, natural infection occurs widely in several domestic

animals and rodents, which act as reservoirs of infection.

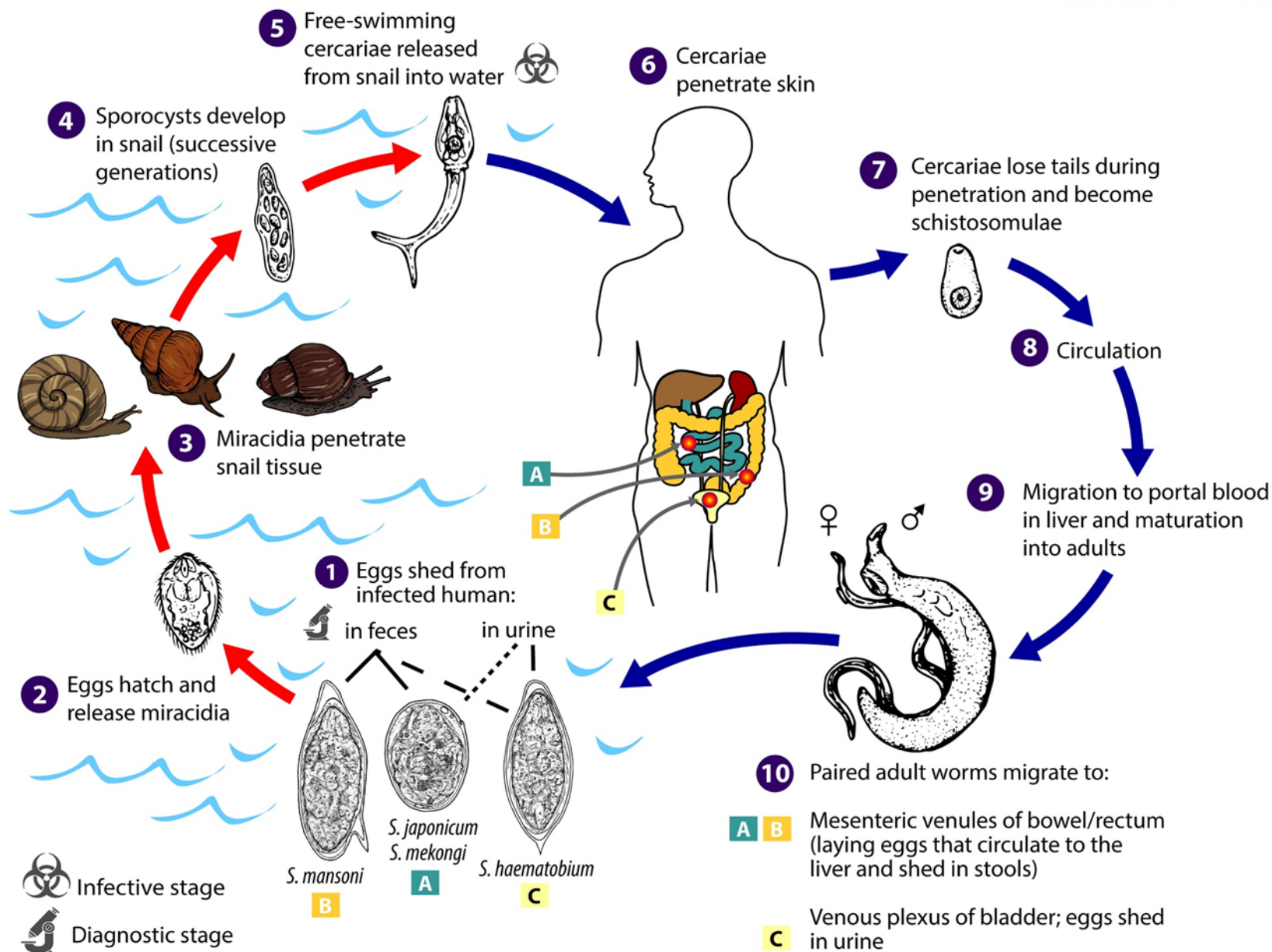
Intermediate host: Amphibian snails of the genus

Oncomelania.



Life Cycle

- Eggs with small lateral knob are passed in feces.
- In freshwater, eggs hatch releasing miracidia.
- Miracidia infect *Oncomelania* snails (intermediate host). Develop into sporocysts → cercariae.
- Cercariae leave the snail and swim freely.
- Humans get infected when cercariae penetrate the skin in contaminated water.
- They become schistosomula, migrate via circulation to mesenteric veins of small intestine.
- Mature into adult worms, produce eggs, and the cycle continues.



Sign and Symptoms

Causes Oriental schistosomiasis (Katayama disease). More severe than *S. mansoni* due to higher egg production. Acute phase: Katayama fever (similar to *S. mansoni*).

Chronic phase: intestinal hyperplasia, hepatosplenomegaly, periportal fibrosis (clay pipe-stem), portal hypertension → esophageal varices, GI bleeding. Intestinal symptoms: abdominal pain, bloody diarrhea, anemia.

Complications: CNS involvement (epileptic seizures, spinal lesions) and lung disease (cor pulmonale) in some cases.

Laboratory diagnosis

1. Stool microscopy:

Eggs with lateral spines may be demonstrated microscopically in stools.

2. Rectal biopsy:

Proctoscopic biopsy of rectal mucosa may reveal eggs when examined as fresh squash preparation between two slides.

3. Serology:

Detection of antigen or antibody by ELISA

4. Imaging:

Ultrasonography is useful to detect hepatosplenomegaly and periportal fibrosis.

Treatment

S. japonicum infection is more resistant to treatment than other schistosomiasis. A prolonged course of intravenous tartar emetic gives good results. Praziquantel is the drug of choice.



Fasciola hepatica

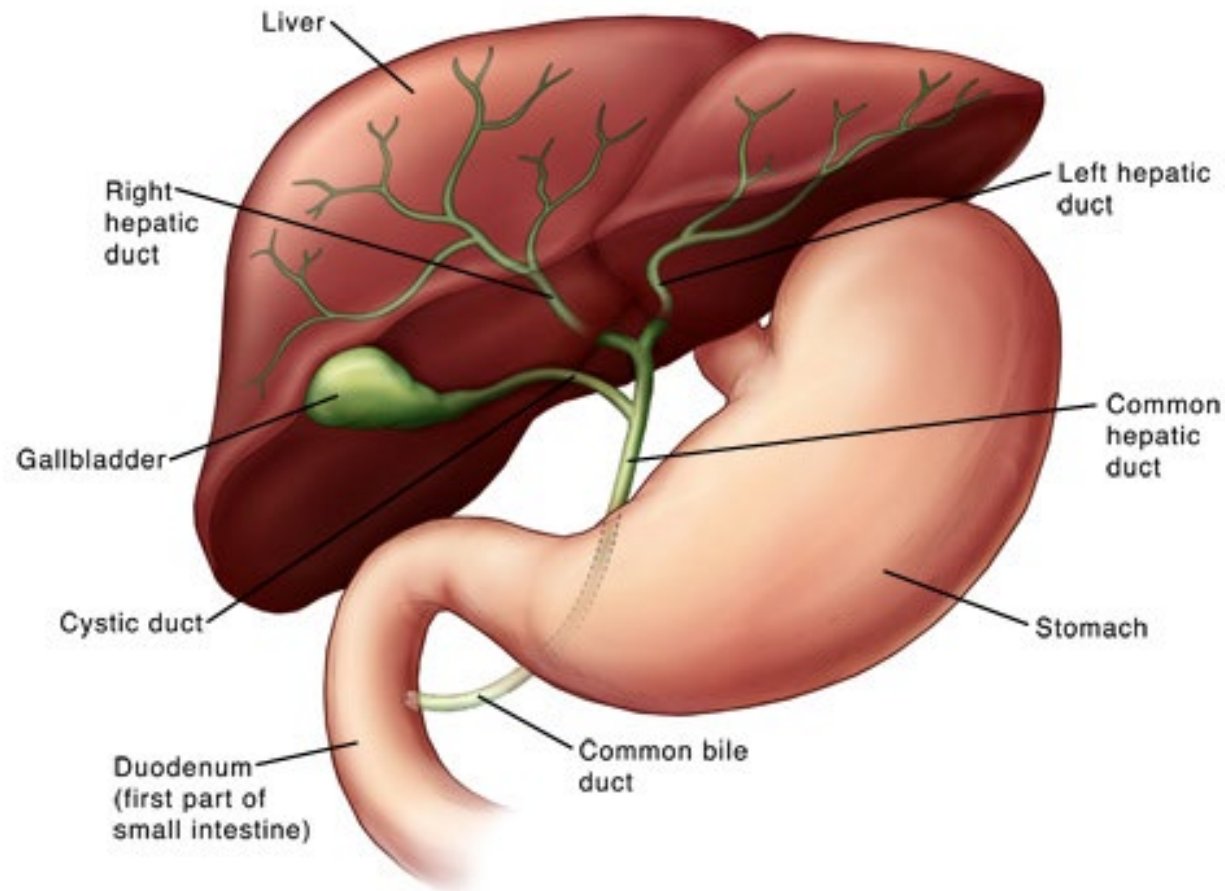
Geographical distribution

- It is the largest and most common liver fluke found in man, however its primary host is the sheep and to a less extent, cattle.
- It causes the economically important disease, "liver rot"; in sheep.
- It is worldwide in distribution, being found mainly in sheep-rearing areas.



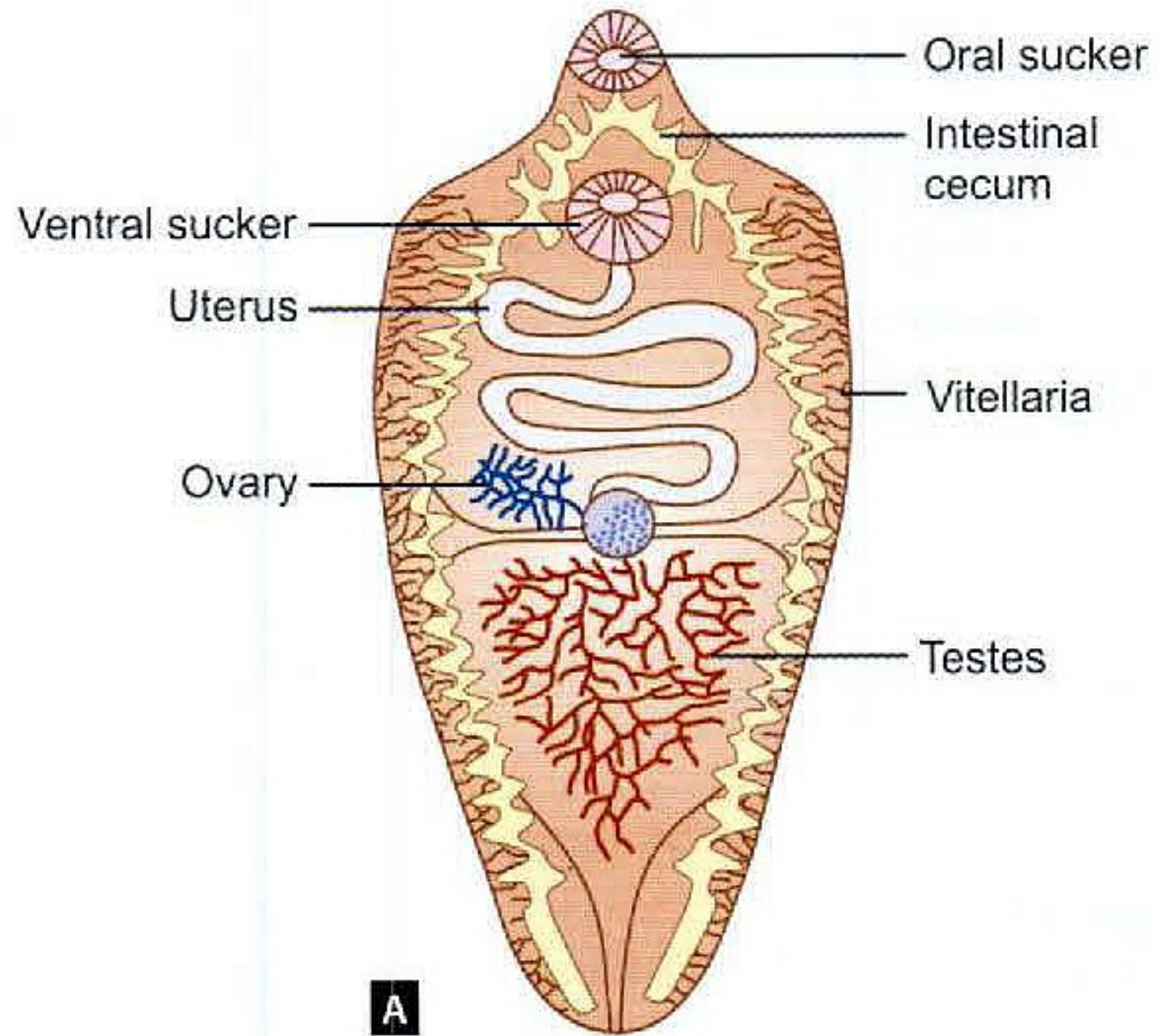
Habitat

Adult worm lives in the bile duct of the definitive host



Morphology

1. Shape: broad and leaf-like with head lobe.
2. Size: 2-3cm in length by 0.8-1.3 in breadth.
3. Oral and ventral sucker: oral sucker is smaller than ventral sucker
4. They are hermaphrodite.
5. Female reproductive system consist of single multilobed ovary, a short oviduct leads from the ovary to ootype, two vitteline gland, and coiled tube uterus.
6. Male reproductive system consist of two testes, vas efferent, vas deferens.
7. The intestinal tract has well developed pharynx, short esophagus and two branched intestinal caeca which are extending from the anterior end to the posterior end of the body.



Morphology

Egg:

The eggs are large, ovoid, operculated, bile-stained and about 140 μm by 80 μm in size

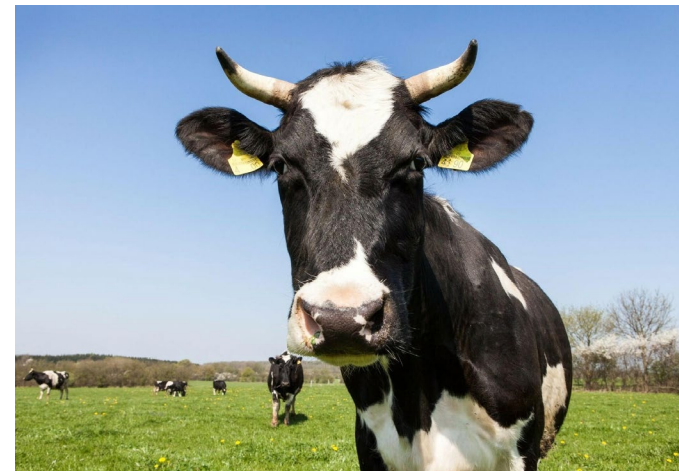


Hosts

- Intermediate host: freshwater snail (*Lymnaea* sp.)

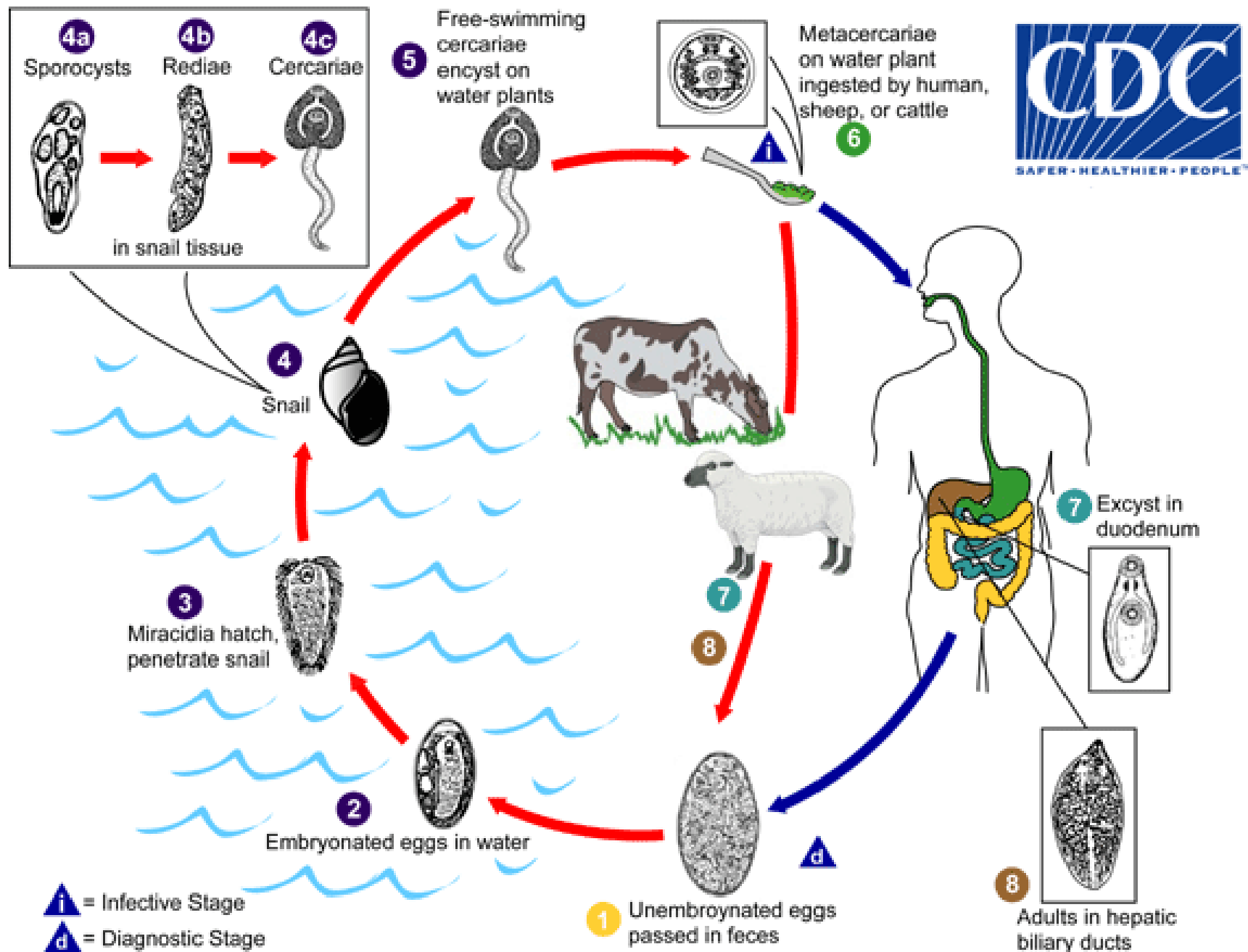


- Definitive host: sheep, cattle, goat and human



Life Cycle

- Eggs are passed in feces of infected herbivores or humans. In water, eggs hatch and release miracidia.
- Miracidia infect *Lymnaea* snails (intermediate host).
- Inside the snail they develop into sporocyst, redia, and cercariae.
- Cercariae leave the snail and encyst on aquatic plants as metacercariae (infective stage).
- Humans or animals become infected by eating contaminated water plants such as watercress.
- Metacercariae excyst in the duodenum, penetrate the intestinal wall, migrate through the peritoneum, and reach the liver.
- Adult flukes live in bile ducts and produce eggs, completing the cycle.



Pathogenesis

F. hepatica is larger, causing greater mechanical and parenchymal liver damage. Since humans are not its primary host, it produces a stronger inflammatory response, and in some cases larvae may migrate through the liver and diaphragm into the lungs.

Acute phase

In the acute phase during the migration of the larva, patients present with fever, right upper quadrant pain, eosinophilia, and tender hepatomegaly.

Pathogenesis

Chronic Phase

In the chronic phase, fascioliasis may cause biliary obstruction, cirrhosis, jaundice, gallstones, and anemia, but it is not linked to liver cancer.

Eating raw infected sheep liver can cause halzoun, where worms attach to the throat mucosa, leading to congestion, dysphagia, dyspnea, and rarely asphyxia. This condition, also seen with pentastome larvae, is common in Lebanon and parts of the Middle East and North Africa.

Diagnosis

- **Stool microscopy:** Demonstration of eggs in feces or aspirated bile from duodenum is the best method of diagnosis. Eggs of *E. hepatica* and *F. buski* are indistinguishable
- **Serodiagnosis:** Serological tests such as ELISA, is helpful in lightly infected individuals for detection of specific antibodies. ELISA becomes positive within 2 weeks of infection and is negative after treatment. In chronic fascioliasis, *Fasciola* antigen may be detected in stool.

Treatment

Oral triclabendazole (10 mg/kg once) is the treatment of choice.

Alternative drug is **bithionol** (30-50 mg for 10- 15 days).

Prednisolone at a dose of 10-20 mg/ kg is used to control toxemia.

Prevention

Fascioliasis can be prevented by:

- Health education.
- Control of snails.
- Proper disposal of human, sheep, and cattle feces.
- Proper disinfection of watercresses and other water vegetations before consumption.