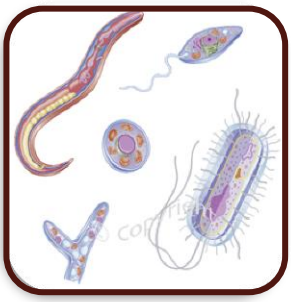


Parasitology

3rd Stage

Dr. Abdulmohsin

Trematodes

Liver (hepatic) flukes

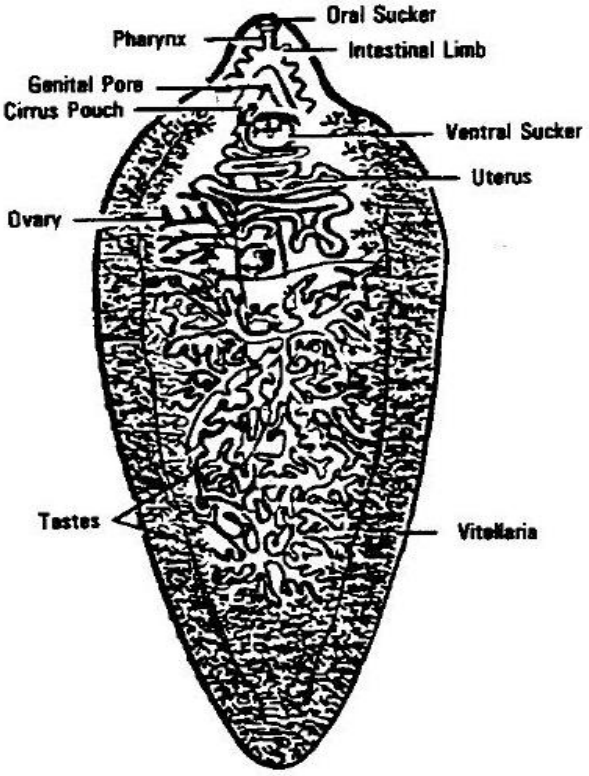
1. *Fasciola hepatica*
2. *F. gigantica*
3. *Clonorchis sinensis*
4. *Opisthorchis felineus*
5. *Dicrocoelium dendriticum*

Fasciola hepatica

(common sheep liver fluke)

Fascioliasis hepatica:

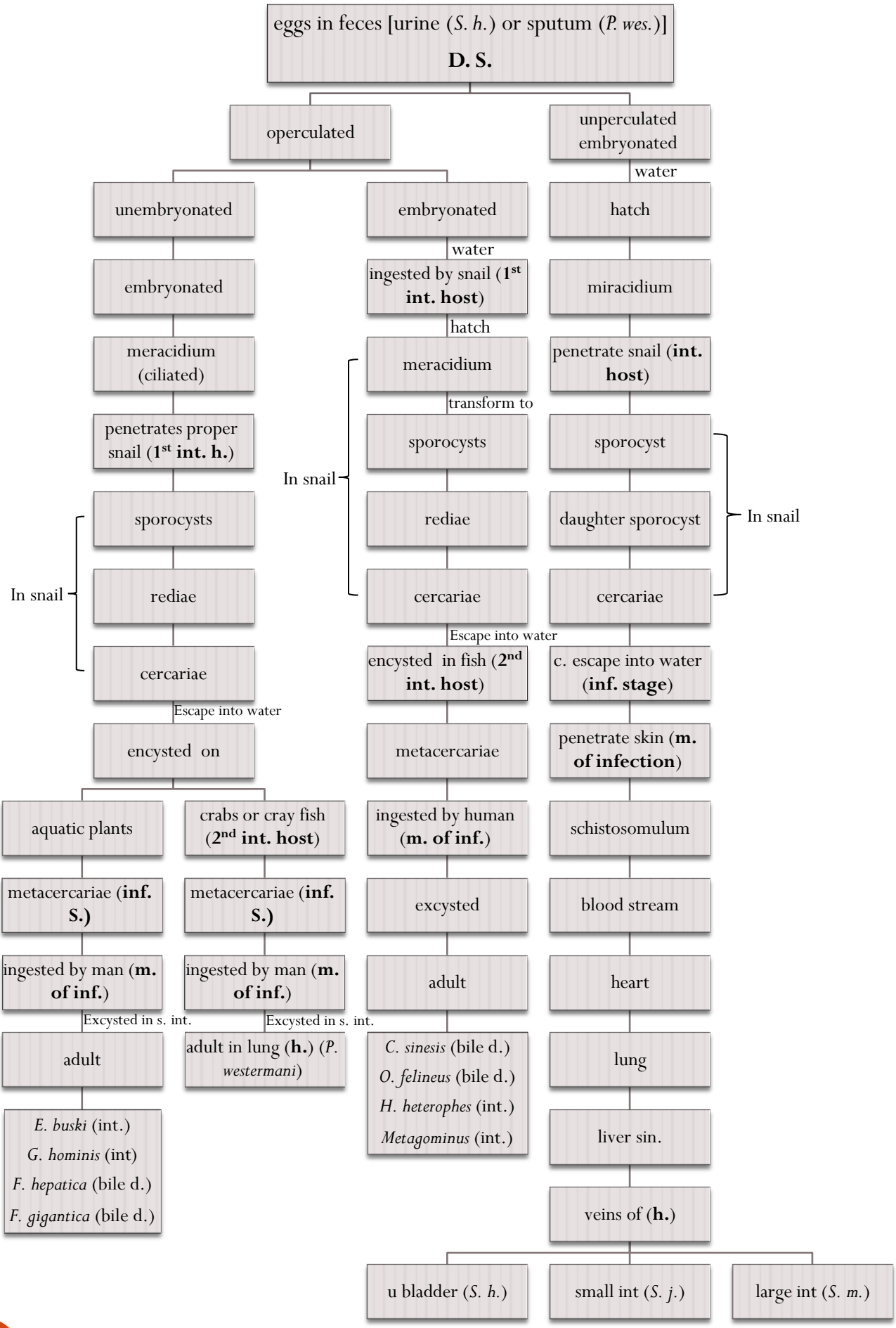
- First recorded in 1379.
- 1758, Linnaeus gave the worm its name.
- Pallas, 1760 was the first to find it in human.
- Size: 30 x 13 mm. ..... Large flukes.
- Leaf shaped, oral sucker end on a con-shaped projection at anterior end.
- Acetabulum (ventral sucker): close to the O- sucker.
- Teguement (surface coat): covered with scale like spines.
- Extensive branching of intestinal ceca, testes, vetilline follicles.
- Ovary also branched (dentritic).
- Uterus: short and coiling.
- Adult: live in bile passage of liver, (mammal, especially ruminants).



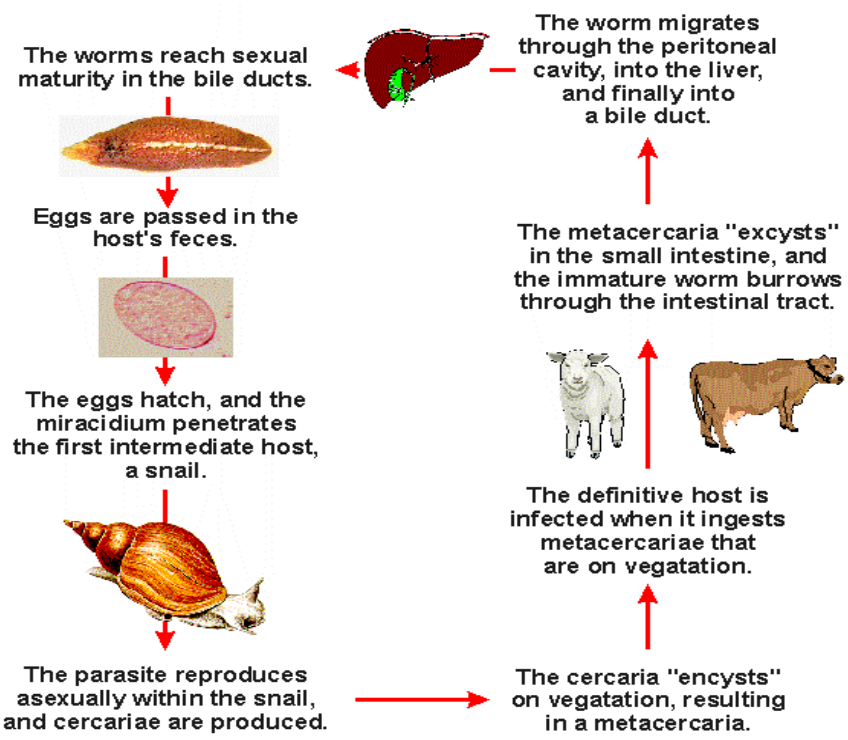
By: Mohammed Salim

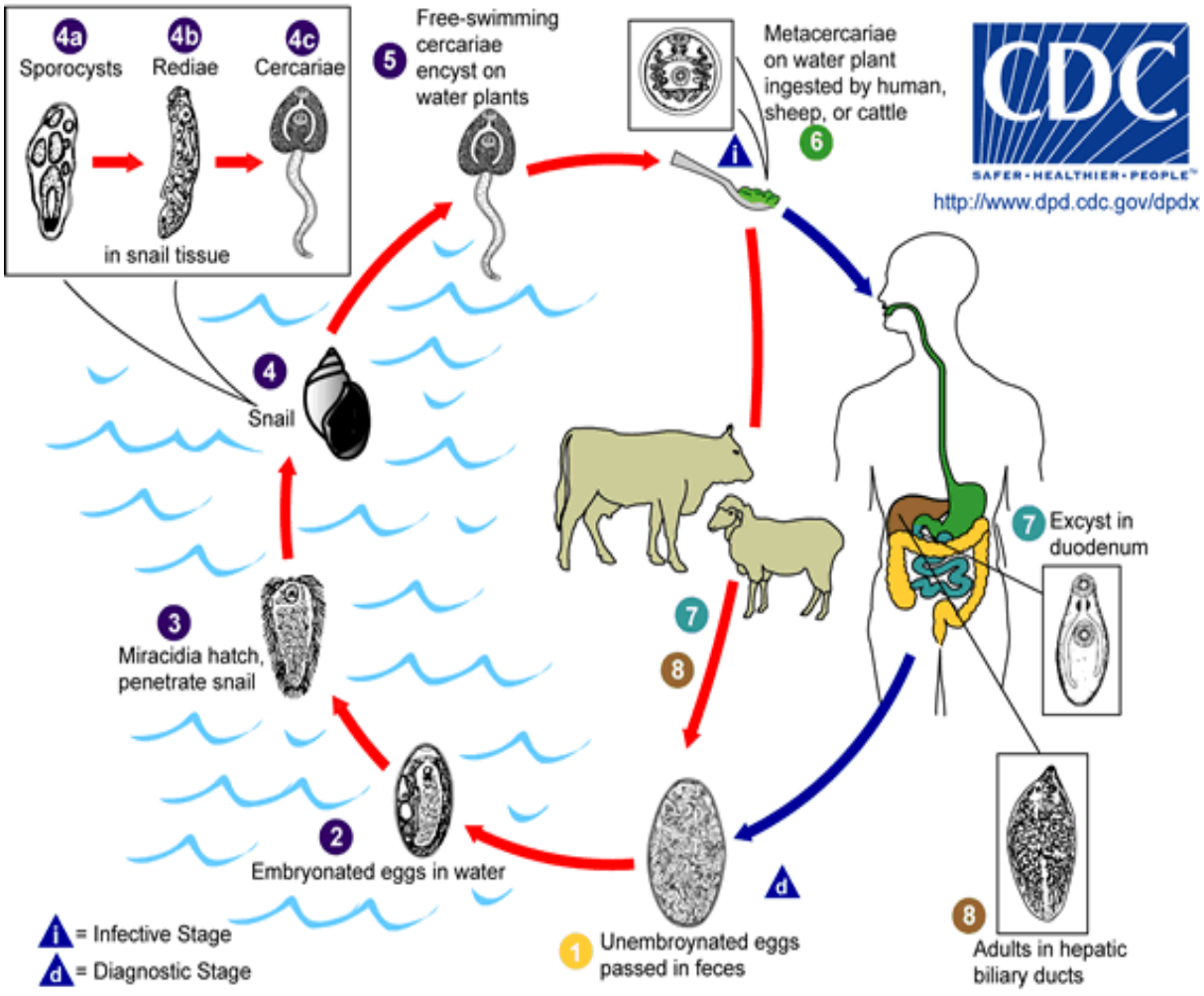
- Adult fed on lining of biliary ducts.

Trematodes (flukes)



Life cycle


The worms reach sexual maturity in the bile ducts.
Eggs are passed in the host's feces.
The eggs hatch, and the miracidium penetrates the first intermediate host, a snail.
The parasite reproduces asexually within the snail, and cercariae are produced.
The cercaria "encysts" on vegatation, resulting in a metacercaria.
The definitive host is infected when it ingests metacercariae that are on vegatation.
The metacercaria "excysts" in the small intestine, and the immature worm burrows through the intestinal tract.
The worm migrates through the peritoneal cavity, into the liver, and finally into a bile duct.


4a Sporocysts
4b Rediae
4c Cercariae
in snail tissue
5 Free-swimming cercariae encyst on water plants
Metacercariae on water plant ingested by human, sheep, or cattle
6
i
CDC
SAFER·HEALTHIER·PEOPLE™
http://www.dpd.cdc.gov/dpdx
4
Snail
3 Miracidia hatch, penetrate snail
2 Embryonated eggs in water
7 Excyst in duodenum
7
8
d
1 Unembroynated eggs passed in feces
8 Adults in hepatic biliary ducts
i = Infective Stage
d = Diagnostic Stage

Eggs are passed out of the liver with bile into intestine to be voided with feces.

develop in water into miracidium → (9-10 days) Hatch → miracidium swim (24 hours) → suitable snail (Lymnaeidae) → in the snail, mother sporocyst → 1st gen. rediae → daughter rediae → cercariae. (begin emerging 5-7 weeks) → attach to any object → deposit their tail and produce thick, transparent cyst around itself or encysted free in water → metacercaria → mammals eat mctacercaria encysted on vegetation or in water. → excysted in small intestine, penetrate the wall → peritoneal cavity → creep over the viscera → (5 days) → contracting the capsule of liver → burrow in liver parenchyma (2 months) → bile duct → egg (8-12 weeks post infection).

N.B.:

1. glycochonic acid elicited emergence of metacercaria from the cyst.
2. Man infected by eating water cress containing metacercaria.

Pathogenesis

Amount of damage depend on worm burden.

Acute phase

(Migrating phase in liver parenchyma) (8 weeks):

1. Traumatic damage
2. Eosinophelic inflammation
3. Linear hemorrhagic track along the path of migrating juvenile fluke.
4. Liver swollen.
5. Hyperplasia of bile duct (toxic product of larvae)

The disease known as liver rot due to extensive damage of liver.

Chronic phase

Occur beyond 12 weeks. (worm in the bile duct).

1. Mechanical irritation and metabolic by-product and edema → hyperplasia of biliary epithelium and duct fibrosis (pipe steam fibrosis) → partial or total biliary obstruction.
2. Anemia: Due to
    - blood sticking by worm.
    - or from hemorrhage from mucosa of bile duct.
    - Proline from the worm.
3. hypoproteinemia edema .
4. Gall bladder may be infected (same pathological changes).
5. In heavy infection: wall of bile duct eroded completely → worms reenter liver parenchyma → abscesses.
6. Migrating juveniles produce ulcers in ectopic location such as eye, brain, skin and lung.

Symptoms and signs

- ✓ Acute phase
  fever, epigastric pain, right upper quadrant pain, urticaria, leukocytosis, eosinophilia, mild and moderate anemia, ↑ level of IgG, IgM, IgE.
- ✓ Chronic stage:
  12 weeks post infection (associated with biliary obstruction & cholangitis)
  Acute epigastric pain, biliary colic, fever, pruritus, jaundice, hepatomegaly, eosinophilia, macrocytic anemia, cholecystitis, cholclithiasis, diarrhea.

Laboratory Diagnosis

- Microscopic identification of eggs is useful in the chronic (adult) stage.
- Eggs can be recovered in the stools or in material obtained by duodenal or biliary drainage. They are morphologically indistinguishable from those of *Fasciolopsis buski*.
- False fascioliasis (pseudofascioliasis) refers to the presence of eggs in the stool resulting not from an actual infection but from recent ingestion of infected livers containing eggs. This situation (with its potential for misdiagnosis) can be avoided by having the patient follow a liver-free diet several days before a repeat stool examination.
- Antibody detection tests are useful especially in the early invasive stages, when the eggs are not yet apparent in the stools, or in ectopic fascioliasis.

Epidemiology

- ☒ Is cosmopolitan dis. (worldwide in distribution).
- ☒ d. host: sheep, cattle, goat, many other ruminants, rabbit , human, swine, equids. (herbivores)
- ☒ snail vector (I. host): Lymnaeidea-Lymmnaea spp.
- ☒ infect man where sheep, cattle, goat, are raise and there are lymnaeid snail.
- ☒ It is found where there are a close association of live stokes, humans and snail.

Treatment

Bithionol.

Parasitic pharyngitis

Temporary condition due to attachment of adult *fasciola hepatica* on pharyngeal mucosa as a result of eating fresh infected liver (containing *fasciola hepatica*) leading to congestion accompanied by difficulties in breathing and asphyxiation.

False fascioliasis

Appearance of eggs of *fasciola hepatica* in stool of man following ingestion of infected liver of sheep, goats, or cattle, row or cooked. The egg disappear within few days.

Control

1. molluscicide to kill the snails
2. draining of pasture lands.
3. treatment of inflected animals.
4. avoidance eating of water cress and water plants.

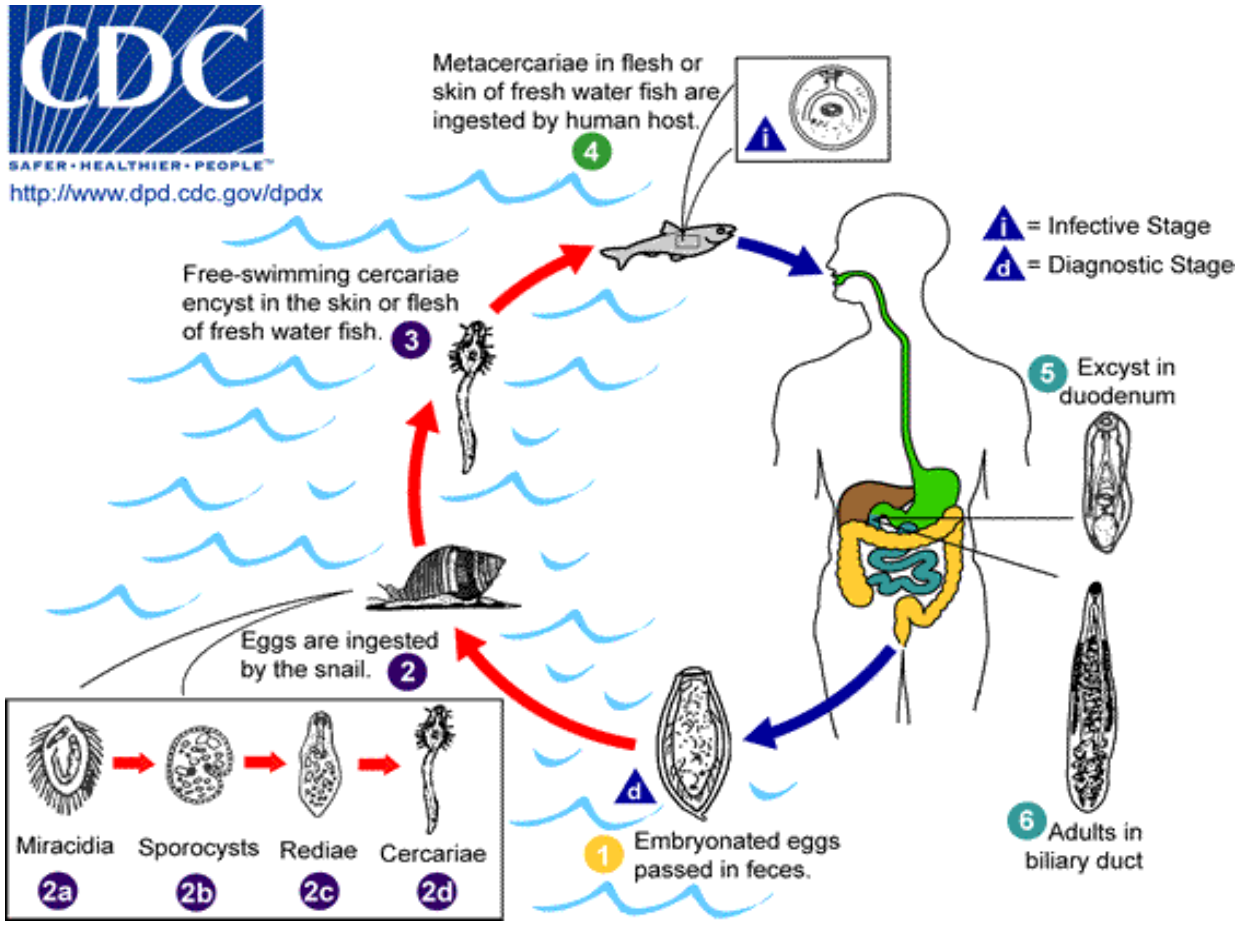
Clonorchis sinensis

Clonorichiasis

Causal Agent

The trematode *Clonorchis sinensis* (Chinese or oriental liver fluke).

Life cycle



Clinical Features

Most pathologic manifestations result from inflammation and intermittent obstruction of the biliary ducts. In the acute phase, abdominal pain, nausea, diarrhea, and eosinophilia can occur. In long-standing infections, cholangitis, cholelithiasis, pancreatitis, and cholangiocarcinoma can develop, which may be fatal.

Laboratory Diagnosis

Microscopic demonstration of eggs in the stool or in duodenal aspirate is the most practical diagnostic method.

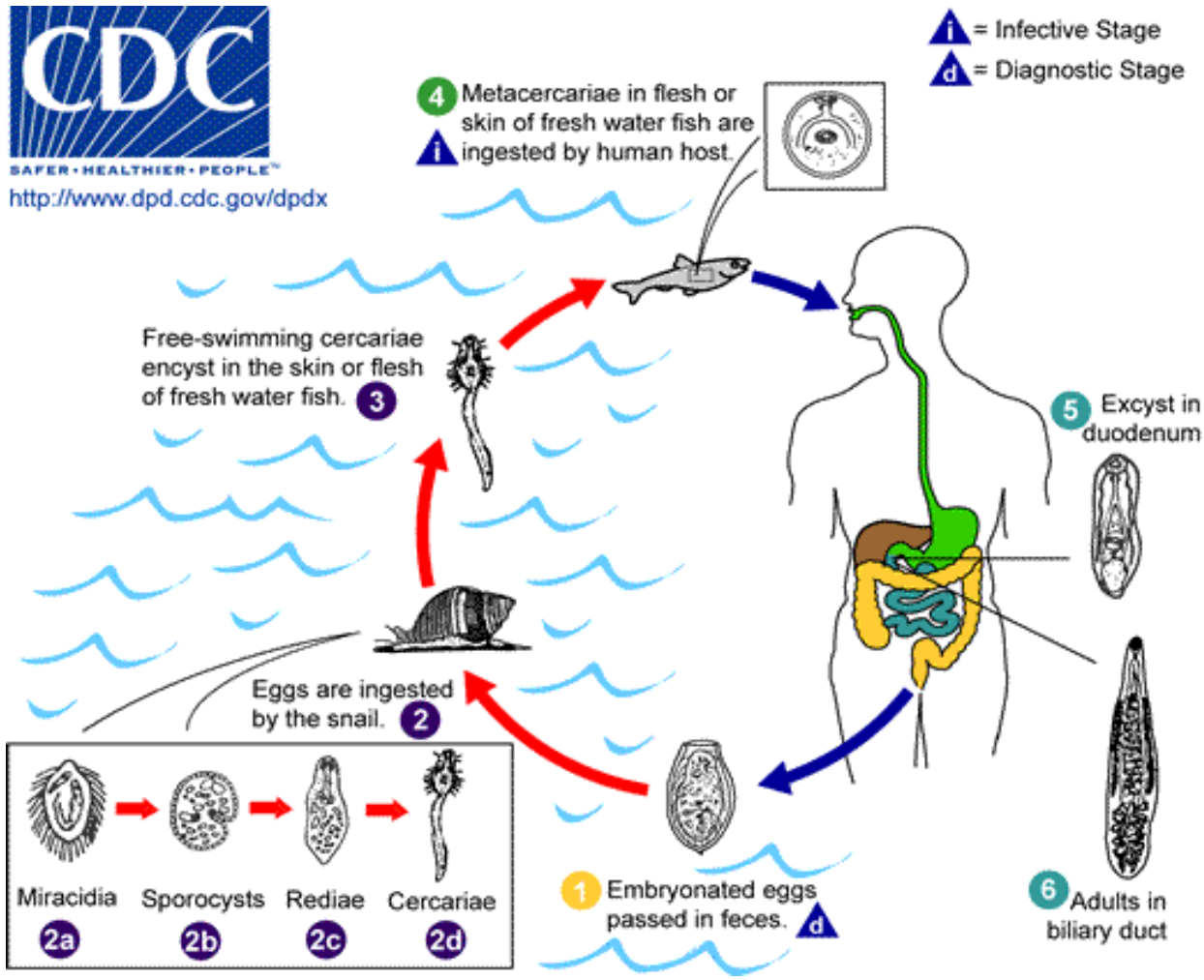
Opisthorchis felineus

(Opisthoochiasis)

Causal Agent

Trematodes (flukes) *Opisthorchis viverrini* (Southeast Asian liver fluke) and *O. felineus* (cat liver fluke).

Life cycle



Clinical Features

Most infections are asymptomatic. In mild cases, manifestations include abdominal pain, diarrhea or constipation. With infections of longer duration, the symptoms can be more severe, and hepatomegaly and malnutrition may be present. In rare cases, cholangitis, cholecystitis, and chlolangiocarcinoma may develop.

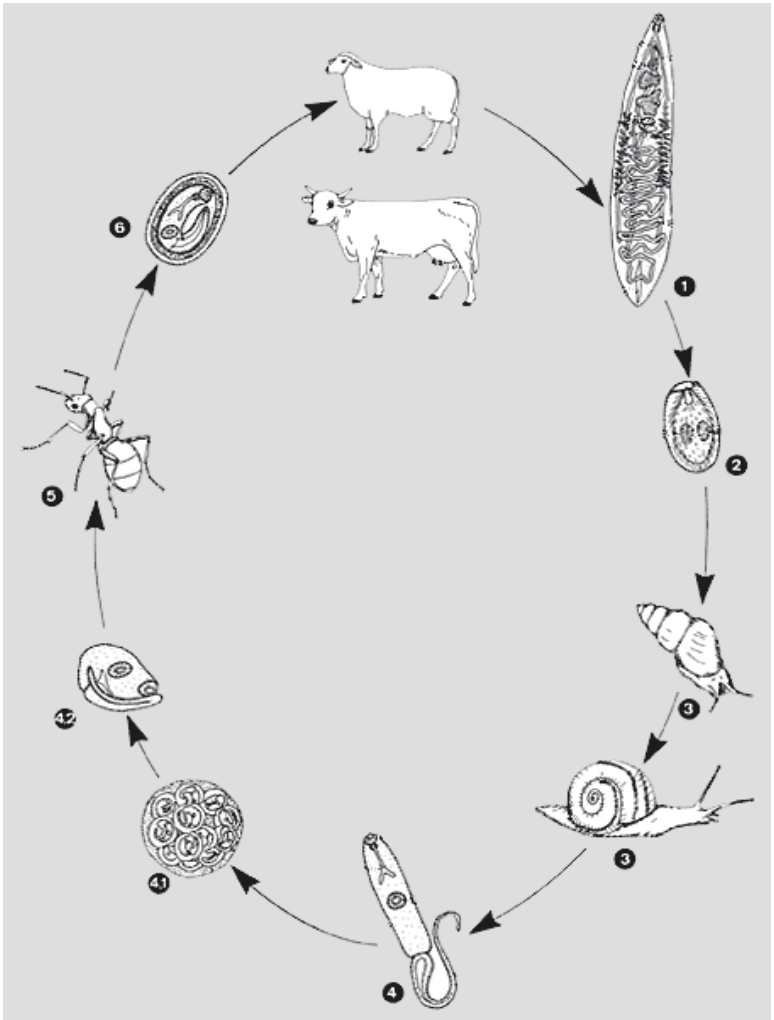
Laboratory Diagnosis

Diagnosis is based on microscopic identification of eggs in stool specimens.

Dicrocoelium dendriticum

Dicrocoelium dendriticum is a bile duct fluke of ruminants such as sheep, goats and deer as well as pigs

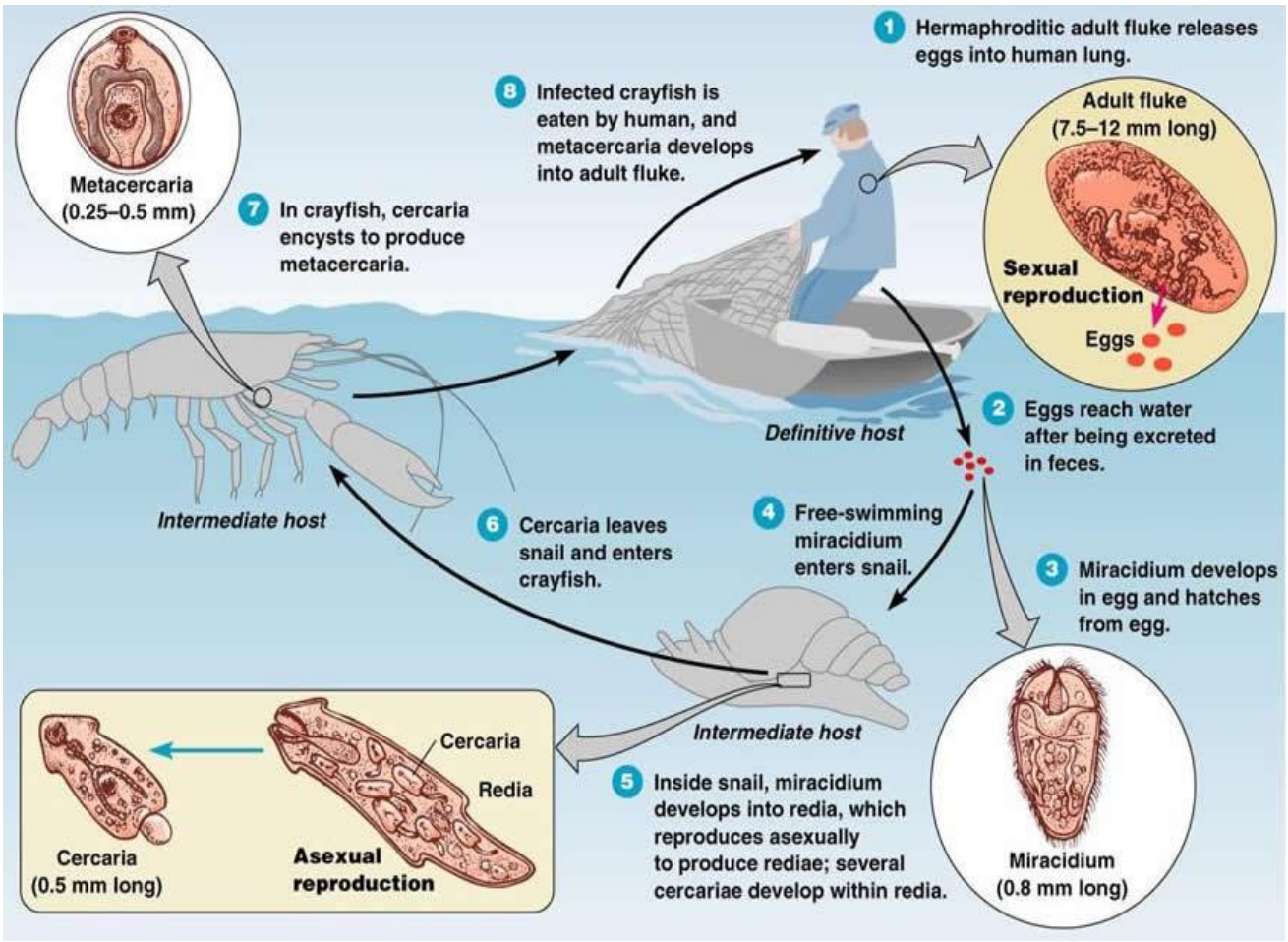
Life cycle

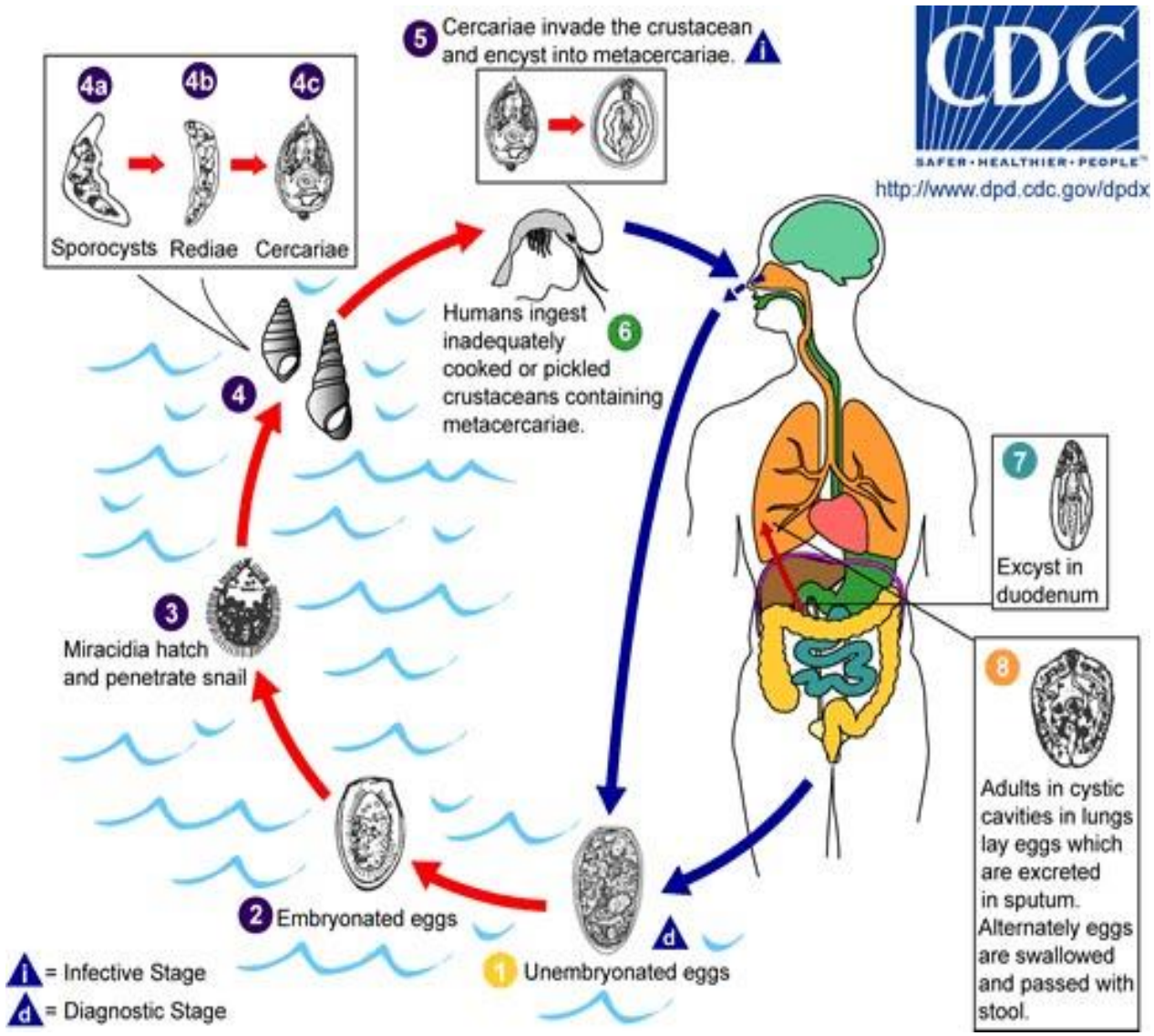


Pulmonary flukes

Paragonimus westermani (lung fluke)

- D. host: dog, man, cat.
- India to far-east .
- Adult: live in fibrous cap. in lung .
- mode of inf.: Ingestion of crabs or Cray—fish containing metacercaria, then metacercaria penetrate intestinal wall, to peritoneal cavity → burrow through diaphragm → lungs → adult → eggs in bronchial secretion →
  - coughing up with sputum
  - Swallowed → feces





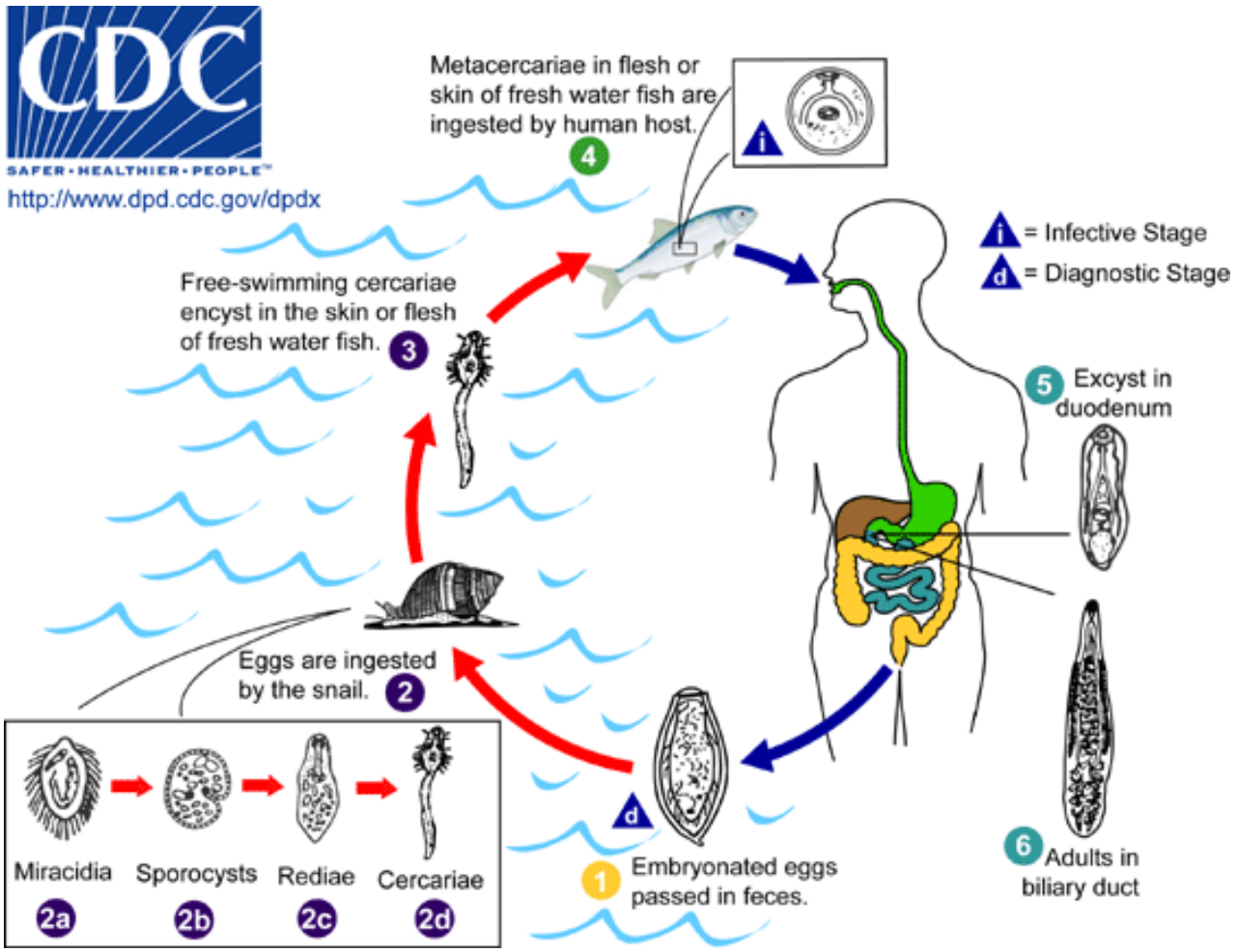
Pathogenesis

due to

- ☒ T. reaction.
- ☒ Neutrophilic + eosinophilic infiltration → formation of fibrous capsule + cyst with channel to bronchiole. This capsule contain adult, eggs, and purulent fluids which lead to paroxysmal coughing + hemorrhage + blood in sputum (rusty or blood tinged sputum)

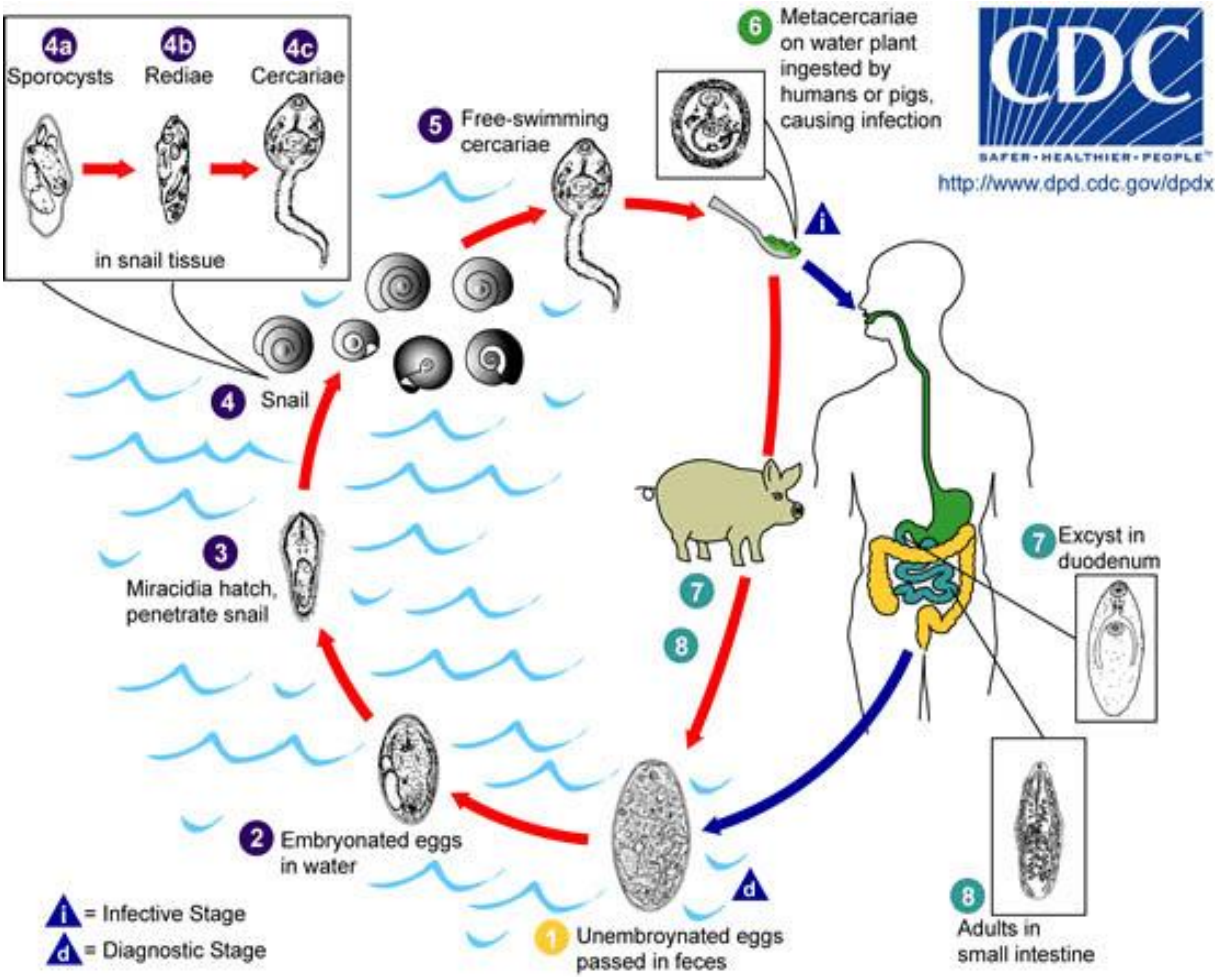
fever, dyspnea, malaise, anorexia, chest pain, bronchitis, haemoptysis and epilepsy .

Intestinal flukes



1. *Fasciolopsis buski* (giant intestinal flukes)

- mode of inf.: as *F. hepatica* ingestion of aquatic plant.
- In far east only .
- D. Host: pig, dog, rabbits, man


4a Sporocysts
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in snail tissue
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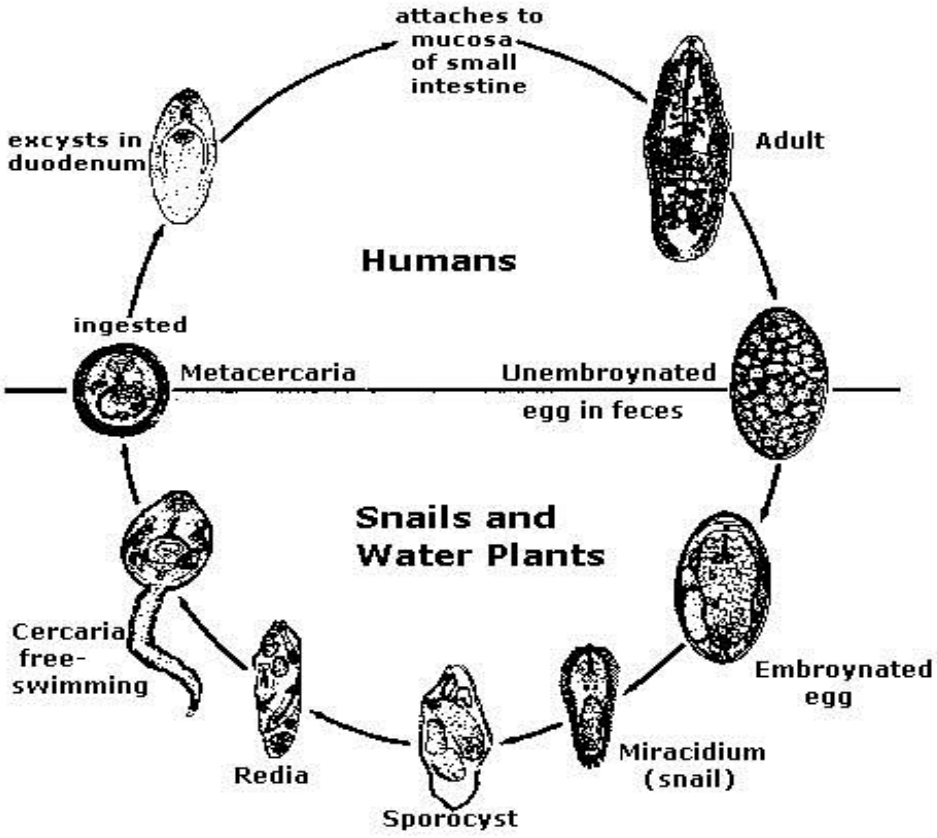

attaches to mucosa of small intestine
excysts in duodenum
Adult
Humans
ingested
Metacercaria
Unembroynated egg in feces
Snails and Water Plants
Cercaria free-swimming
Redia
Sporocyst
Miracidium (snail)
Embroynated egg

Pathogenesis and symptoms

Local inflammation + ulcer + hemorrhage + hyper secretion of mucous

In heavy inf.: generalized edema (due to toxic byproducts), ascitis, diarrhea, abdominal pain, hvpereosinophilia.

2. *Gastrodiscoides hominis*

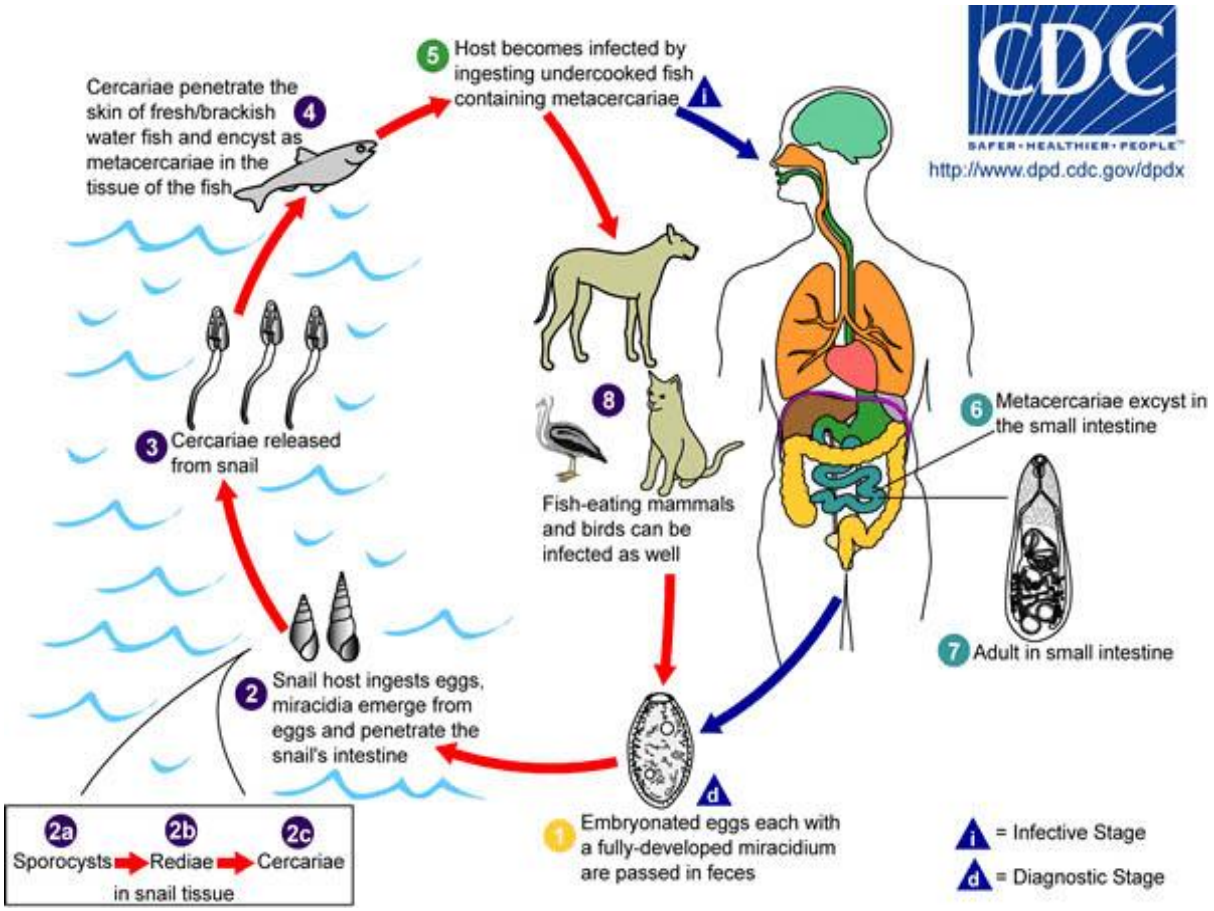
D. host: man, pig, deer (as *F. buski*).

3. *Heterophyes heterophyes*: heterophyiasis

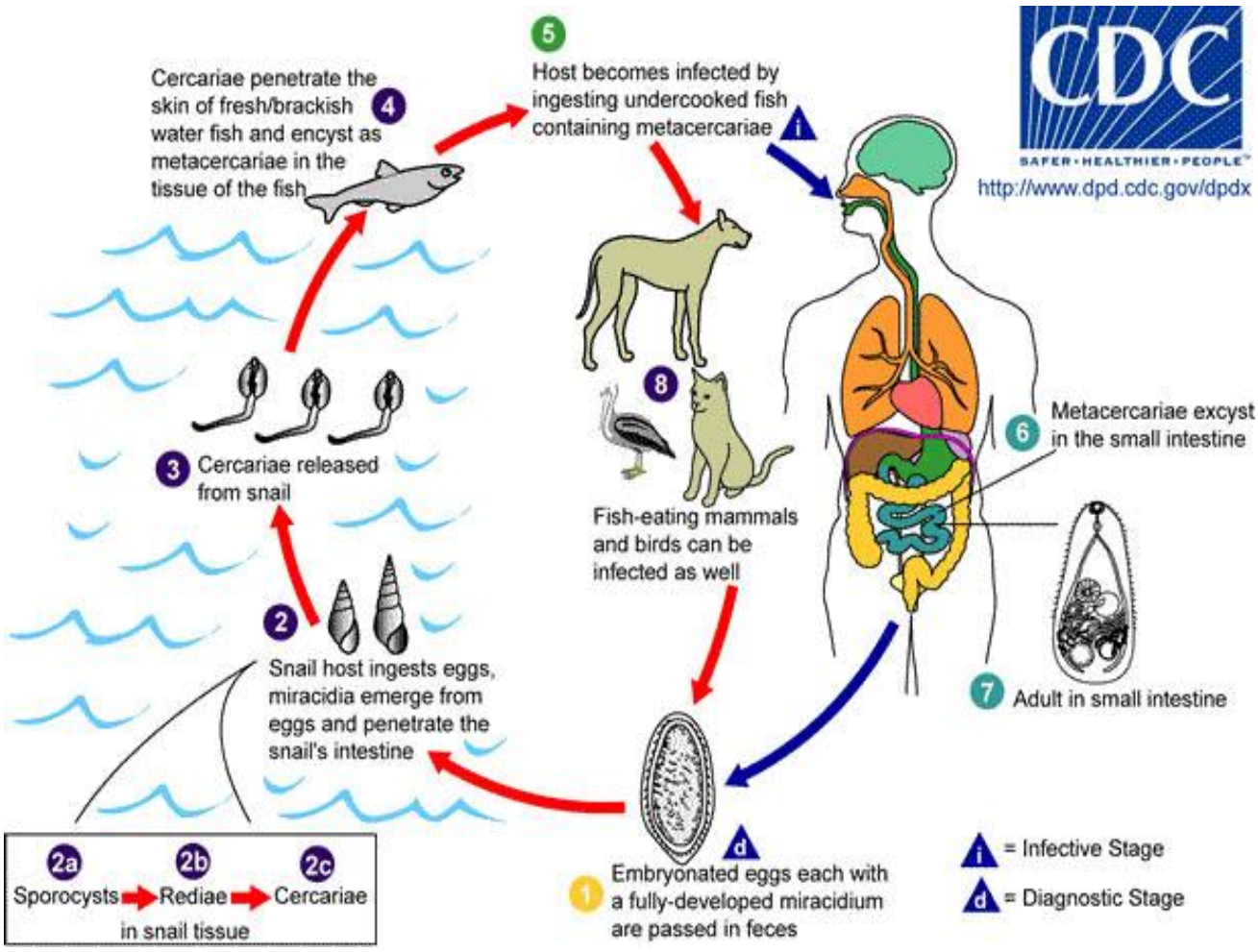
- ✓ D. host: dog, cat, fish eating birds.
- ✓ Mode of infection: eating uncooked, raw, salted, dried fish containing metacercaria
- ✓ found in middle & far east.

Pathogencsis

Heavy ulceration of intestinal wall, abdominal pain, mucous diarrhea



metagonimiasis (like *Heterophyes heterophye*s).



Blood fukes

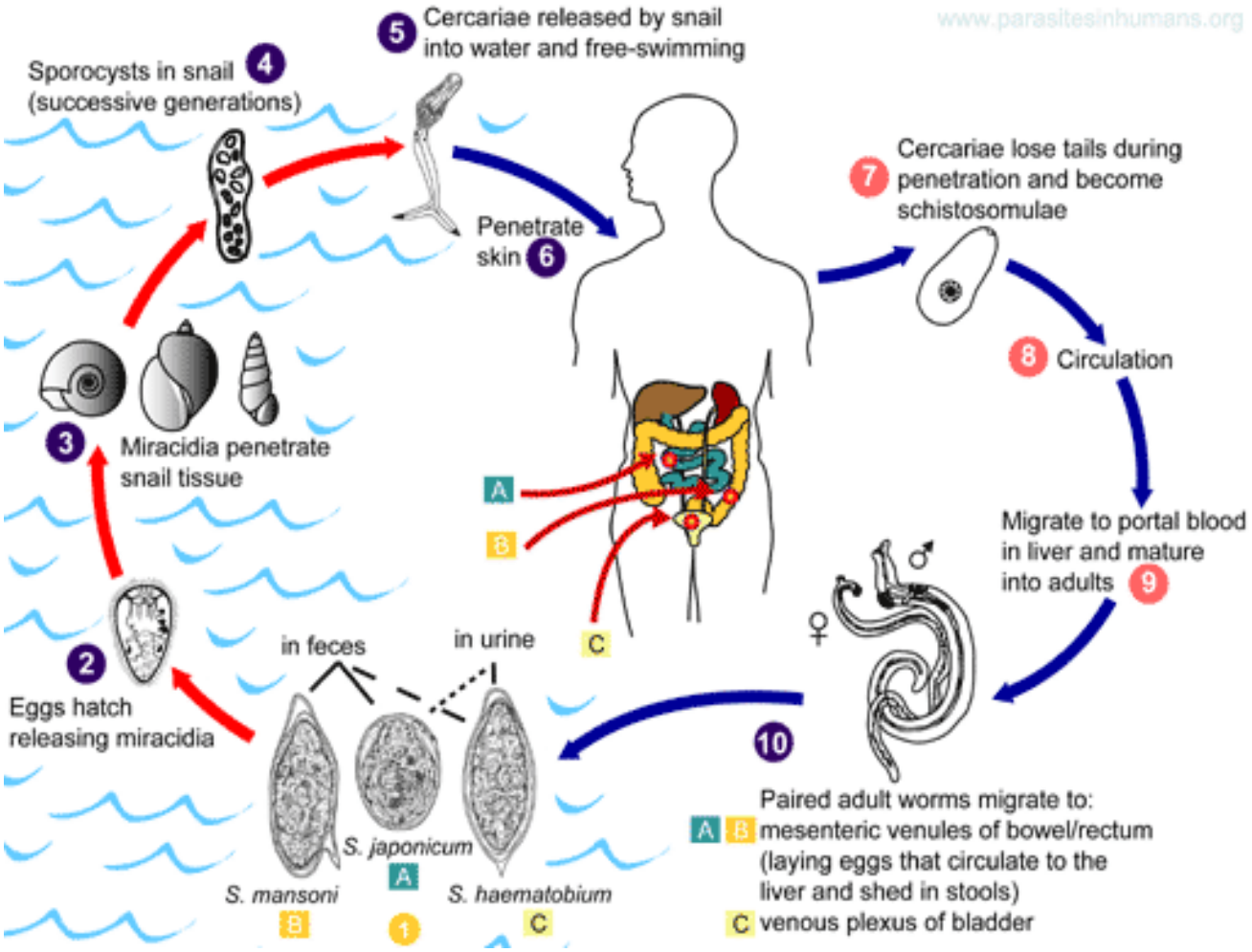
Schistosoma spp.

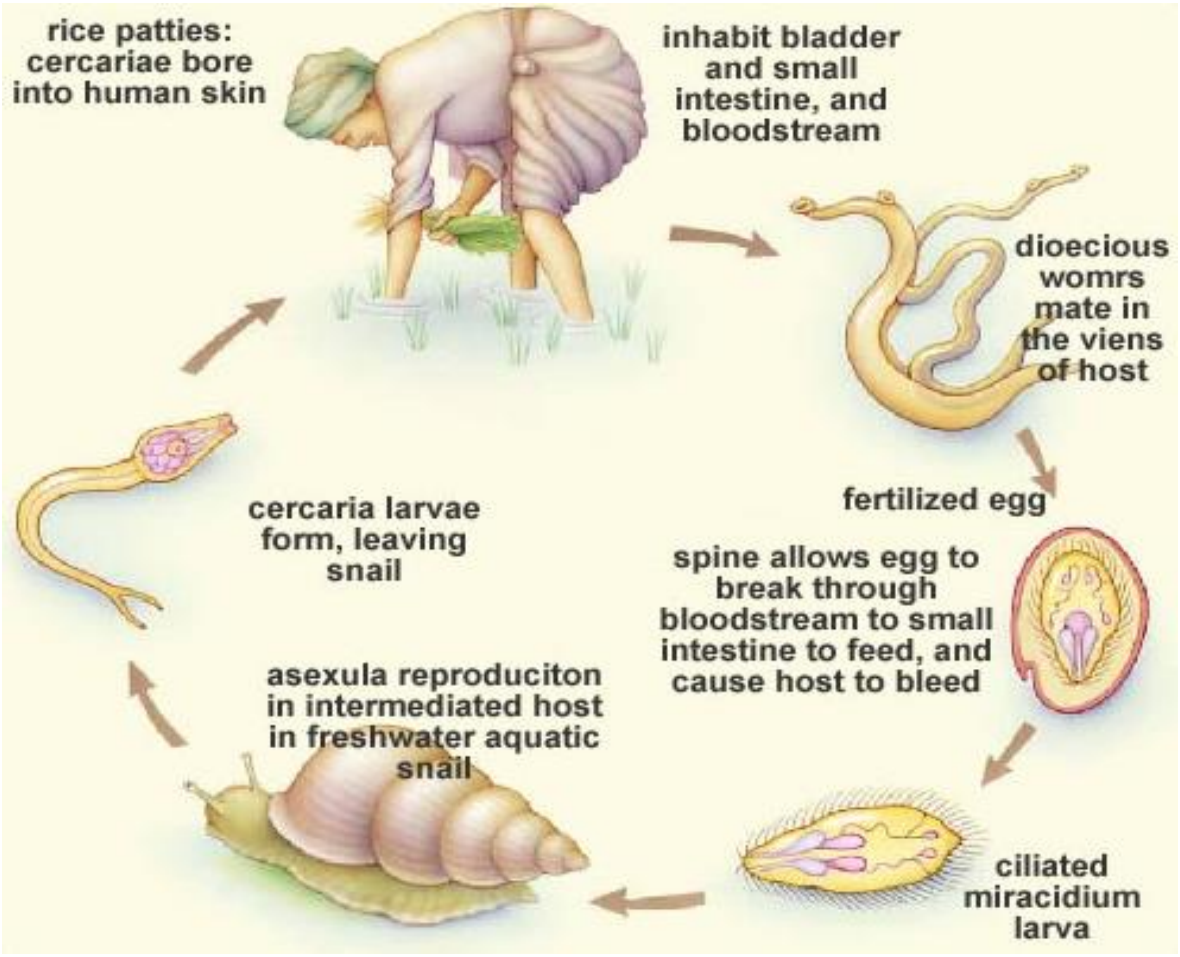
1. *Schistosoma haematobium*: urinary schistosomiasis, vesical schistosomiasis, vesical blood fluke, yrinary bilharziasis
   Endemic in Iraq
2. *S. mansoni*: manson's blood fluke, manson's intestinal schistosomiasis, bilharziasis
3. *S. japonicum*: oriented blood fluke, oriental schistosomiasis.
4. *S. mekongi*: Mekong schistosomiasis.
5. *S. intercalatum*

Life cycle

Female deposit their eggs in venules & pass through the wall and sloughed into lumen of bladder or the intestine and evacuated in feces or urine.

The egg work their way through the tissue to the lumen of organs which is facilitated by miracidium (the embryo of blood flukes) enzyme that release through egg shell.

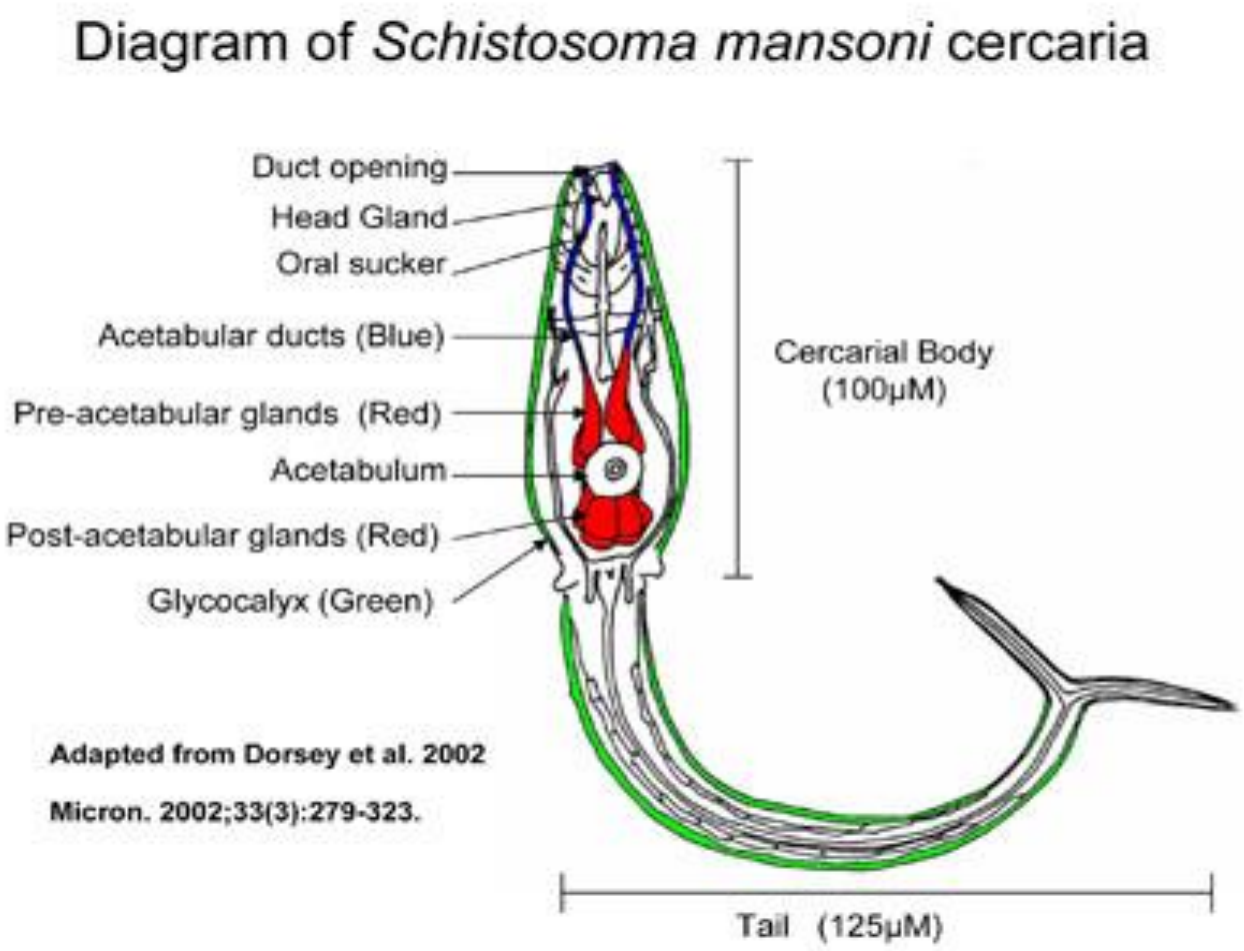




Differences between *schistosoma* and other trematodes

	schistosoma	other trematodes
Sex	Separated (doiecious)	Hermaphrodite
Egg	Non operculated	Usually operculated
Ceca	Reunite at posterior part of the body	Remain separated
Redia	Not present	Present
Cercaria tail	bifurcated	Simple tail
Mode of infection	Penetration of skin	Ingestion
Body	Cylindrical	Flattened
Intermediate host	One	Two
Infective stage	cercaria	Meta cercaria

	S. haematobuim	*S. mansoni*	*S. japanicum*
Geographic distribution	Africa to India	Africa, South America	Far East
Main habitat	Venule of urinary bladder	Venule of colon, rectal, portal redicals	Venule of intestine, portal venous system
Male			
Size	10-18 mm by 1 mm	10-12 mm by 1 mm	10-20 mm by 0.4 mm
Cuticle	Tubercular	Markedly tubercular	Smooth
Reunion of int. ceca in the body	In the middle	In anterior half	In posterior fourth
Testis	4-5, large	8-9, small	6-8, medium
Female			
Size	20 mm	12-16 mm	12-20 mm
Position of ovary	Posterior half	Anterior half	In the middle
Ova	Has a terminal spine	Has lateral spine	Has lateral tubercle
Ova found in	Urine, rarely feces	Feces, rarely in urine	In feces only
No. of ova in uterus	20-30/uterus	1-4/uterus	50 or more/uterus
Reservoir host	Human	Human, non-human primate	Dog, cat, cattle, water buffalo, pig
Intermediate host	*Bulinnus* spp.	*Bimophalaria* spp.	*Oncomelania* spp.



Pathogenesis

Pathogenic changes consist of:

1. Intoxication and sensitization due to absorption of worm's metabolite → hypereisinophylia, systemic sensitization.
2. Tissue damage due to egg extrusion.
3. Reaction to eggs that remain in the tissue (main pathogenic effect).

Severity of the disease depend on No. of worms + egg:

- *S. japonium* → 3000 eggs/worm/day (More sever damage).
- *S. mansoni* → 100-300 eggs/worm/day
- *S. haematobium* → 20-300 eggs/worm/day

☒ Schistosomula (immature worm) evoke a host immune response (IgE, eosinophils) → ↑ resistance to re-infection.
☒ Adult less antigenic [Incorporate host Ag (RBC) → evode host's immune response].
☒ Egg deposit in fine Venules →

1. Pass through the wall of tile organ → lumen → outside the body (few damage) .
2. Carried to other organs (by blood)
3. Retained in the tissue (more damage)

☒ Miracidium, (in egg) → Secrete soluble Ag →

1. evoke minute abscesses → facilitate egg passage.
2. invoke (stimulate) host immune response →
    a. Granuloma (cellular infiltration) formation
    b. Pseudotubercle (macro granuloma):
        - The Cellular infiltrate (Lymphocytes, eosinophils, macrophages, fibroblast)
        - Granuloma aggregation in the intestine and/or urinary bladder wall is the major cause of pathological changes.
3. The wall of bladder and/or intestine become →
    - Inflamed
    - Thickened
    - Fibrotic

    ] Mechanical obstruction

fibrosis of bladder neck → obstruction →

- hydroureter
- hydronephrosis with bacteruria
- pyelonephritis
- renal failure

some lesion calcified → giving sandy appearance to the wall,

some lesion (granuloma) break through into intestine or bladder lumen →

- discharge their content
- ulceration

- thickening of mucosa
- Epi. Hyperplasia
- Ulceration

} polyps in mucosa of intestine or bladder (under malignant change)

In chronic inf.

S. m + *S. j.* → eggs carried to and filtered into liver (portal triad of liver) → granuloma + fibrosis of periportal T. (symmer's pipe-stem fibrosis: hard and white fibrosis) .

Periportal fibrosis lead to

1. hepatomegaly.
2. blockage of portal blood flow.
3. increase portal hypertension .
4. Ascites.
5. congestion of spleen and enlarge.
6. Development of collateral circu. (e.g. esophageal varices) so eggs → lung → pulm. Involvement (less common in *S. m.* and *S. j.*).

In ch. *S. h.* inf.:

- eggs → Heart → lung → liver (rare)
- in lung involvement →
  - pulm. Fibrosis
  - hypertention
  - rt. Side heart future.
- egg trapped in other tissue (brain, spinal cord, spleen, pancreas, myocardium) → inflammatory reaction and focal lesion .
- ch. Bacteremia. (mainly *salmonella*).
- circulating immune complex deposited in kidney → nephrosclerosis + kidney failure.

Signs and symptoms

Incubation period and main lesion

- ✓ *S. j.* … 4-5 wks. … small int.
- ✓ *S. m.* … 6-7 wks. … large int.
- ✓ *S. h.* … 10-12 wks. … urinary bladder

in prepatent period… common in all *schistosoma* spp.

- ✓ skin penetration by cercaria → few except in subsequent inf. will, *S. j.* → cercarial dermatitis (for 36/hrs.)
- ✓ fever with rigor and night sweat.
- ✓ generalized pain in back and limbs.
- ✓ diarrhea, eosinophilia, leukocytosis, uriticaria
- ✓ abdominal pain .
- ✓ enlarged lender liver, acute hepatitis and splenomegaly

in early egg deposition stage in *S. j.* and *S. m.*

- ✓ diarrhea or dysentery, loss of appetite and wt.
- ✓ fever, appendicitis, ascites, epigastric pain.
- ✓ enlarge tender liver and enlarge spleen.
- ✓ anemia, leukopenia, and decrease eosinophilia .

in ch. stage in *S. j.* and *S. m.* inf.

- ✓ liver gradually decrease,
- ✓ splenomegaly, ascites, dyspnea , emaciation.
- ✓ increase anemia and skin pallar.
- ✓ Mesentery and omentum frequently become thickened and separate the abdomen into upper and lower portion
- ✓ Pulm. involvement (rare in *S. j.* and *S. m.*) → hypertention, rt. Side heart failure, fatigue, cough, hemoptysis and dyspnea.

Signs and symptoms of *S. h.* during egg deposition stage

- ✓ Painless terminal hematuria with pus cells and necrotic t. debris (first evidence of infection) later on become painful
- ✓ Frequent micturition or anuria due to urethral stricture.
- ✓ marked cont. of bladder due to extensive fibrosis of the wall.
- ✓ fibrosis of the bladder neck → obst. of urine flow lead to:
    1. hydrourete
    2. hydronephrosis with bacteremia
    3. renal failure .
    4. pyelonephritis
- ✓ fever, pain of hack, liver tenderness.
- ✓ bladder colic (cardinal symptom)
- ✓ if rectal involvement → tenesmus with dysentery.
- ✓ If pulm. Involvement (more common in *S. h.* than in other spp.), fatigue, cough, hemoptysis, palpitation and dyspnea.

Note

S. j. is the most pathogenic and resistance to treatment while *S. h.* is the less pathogenic and most responsive lo treatment.

Epidemiology

- *S. j.*, *S. m.* and *S. h.* … from the table
- *S. mekongi* … Indochina, infect man, dog, rodent, egg in stool, pathogenesis similar to *S. m.*
- *S. interculatum* … infect man only, in Africa, egg in stool, Bulinn's is snail intermediate host

Treatment of schistomiasis: Pranziquantel

Diagnosis

1. Recovery of egg in urine, or stool exam (direct, concentration)
2. keto thick smear (*S. j.*, *S. m.*)
3. nucleopore filtration technique: *S. h.* (method of concenation of egg in uine).
4. reagent strips (detect hematuria and proteinuria) (*S. h.*).
5. rectal biopsy (the biopsy crushed between two glasses).
6. serological test (ELISA).

Control

1. Treatment
2. Mass treatment
3. Sanitary disposal of human excreta
4. Avoidance contact with cercaria infected water
5. water resource developing program
6. snail control by e.g.:
    a. molluscicide (niclosamide)
    b. removal of water vegetation
    c. by snail eating fish or birds
    d. by using competitive snail

Schistosome dermatitis (swimmer's itch)

dermatitis result from human exposure to cercaria of non-human *Schistosomes* (blood flukes of ducks, geese, birds, cattle, rodents, dogs, water buffalo).

Pathogenesis and symptoms

1. netting sensation with erythematous muscular rash.
2. local or generalized urticaria for few hrs.
3. 10-15 hrs., the macule change to papule or vesicle.
4. itching become intense .
5. few days, the reaction gradually decrease.

Note

- ☒ *S. m.* → all nearly derived from human source and non-human primate, and rodent can carry the inf.
- ☒ *S. h.* → man only
- ☒ *S. j.* → Several animals and man infected.