Helminths:
Trematoda - non-segmented flat worms

The schistosomes:
- *Schistosoma mansoni*
- *Schistosoma haematobium*
- *Schistosoma japonicum*
- *Schistosoma mekongi*

Aquatic freshwater snails are the intermediate hosts for all schistosome species

*Schistosoma mansoni*

*Schistosoma japonicum*
Adult male and female *Schistosoma mansoni*

One Effective Evolutionary Strategy for Survival: Camouflage

Locked in life’s embrace

Possible application

Cross section of a pair of adult schistosomes in situ in a mesenteric venule
Embryonated egg of *Schistosoma mansoni*

![Image of Embryonated egg of Schistosoma mansoni](image1)

- Lateral spine
- Miracidium (larva)
- Terminal spine

* The adult female requires tumor necrosis factor for maximum egg production

Embryonated egg of *Schistosoma japonicum*

![Image of Embryonated egg of Schistosoma japonicum](image2)

- Inapparent spine
- Miracidium

Embryonated egg of *Schistosoma mekongi*

![Image of Embryonated egg of Schistosoma mekongi](image3)

- Terminal spine
- Miracidium

Embryonated egg of *Schistosoma haematobium*

![Image of Embryonated egg of Schistosoma haematobium](image4)

- Terminal spine
- Miracidium

Biomphalaria glabrata, a common intermediate snail host for *Schistosoma mansoni*
Miracidium of *Schistosoma mansoni* caught in the act of hatching

Miracidium of *Schistosoma mansoni* caught in the act of hatching

SEM of a cercaria of *Schistosoma mansoni*, the infective stage for humans

Oncomelania nosophora, a common snail intermediate host for *Schistosoma japonicum* in China

**Pathogenesis:**

1. Miracidium inside egg in small intestine releases proteases, dissolves tissues, induces bleeding and diarrhea.
2. Eggs (50% of those produced) wash back into liver, lodge in pre-sinusoidal capillaries, eventually block flow of blood.
4. Portal hypertension leads to induction of embryonic circulatory paths, eggs then by-pass liver. Toxic brain syndrome may ensue.
5. Adults avoid immune detection by:
   a. camouflage strategy, incorporating host serum proteins on tegumental surface.
   b. synthesizing β-2 microglobulin-like molecule on tegumental surface.
Clinical Disease:

Acute Phase
1. "Katayama Fever"
2. Paralysis
3. CNS involvement

Chronic Phase
1. GI bleeding and diarrhea
2. Portal hypertension due to blockage of pre-sinusoidal capillaries
3. Esophageal varices
4. Ascites
5. Rupture of varices, bleeding, death
6. Cor pulmonale, right side heart failure, death
7. Toxic brain syndrome

Diagnosis:
1. Microscopic examination of feces, urine, rectal "snip" for eggs.
2. Capture ELISA for detecting circulating antigens (experimental).
3. Serological tests (e.g., ELISA): indirect measure of exposure, not active disease.
Summary of Schistosome Egg Morphology

<table>
<thead>
<tr>
<th>Schistosoma mansoni</th>
<th>Schistosoma japonicum</th>
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<td>75 μm</td>
<td>30 μm</td>
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Schistosoma haematobium

Biopsy of rectal tissue revealing eggs of *Schistosoma mansoni*.

Drug of Choice: Praziquantel

Mode of Action: Interferes with Ca²⁺ ion channels, leads to disrupted tegument. This drug is more effective if the patient has already developed antibodies against tegumental antigens.

Histological section of bladder with pseudopolyp due to chronic infection with *Schistosoma haematobium*.
Pathogenesis:

Eggs lodge in bladder wall, induce cellular changes associated with granuloma formation.

Clinical Disease:

1. Squamous cell epithelioma
2. Calcification of dome of bladder due to accumulation of dead eggs
3. Hydronephrosis

Medical Ecology:
Transmission is encouraged by:

1. Dam building, irrigation projects (e.g., 3 Gorges Dam, China).
2. Reservoir hosts (primates, oxen).
3. Indiscriminate dispersal of feces and urine into environment.
Prevention and Control:
1. Sanitary disposal of feces

Prevention and Control (cont’d)
2. Public health education.
3. Snail control.
5. Vaccine development for reservoir hosts (e.g., water buffalo).