HUMAN BLOOD FLUKES:

Three major species:
Schistosoma haematobium
Schistosoma mansoni
Schistosoma japonicum

Minor species:
Mekongi
Malayensis
Intercalatum

FEATURES OF HUMAN SCHISTOSOMES

They develop in the portal venous system and adult flukes (depending on species) live in the vein of the intestine or bladder.

Sexes are separate
Unlike most trematodes, they are not flattened and leaflike.
They are long and worm-like
Humans are the only definitive host
Transmission is by contact with water containing the infective form of parasite (cercariae)

Schistosoma mansoni eggs
These eggs are large (length 114 to 180 µm) and have a characteristic shape, with a prominent lateral spine near the posterior end. The anterior end is tapered and slightly curved. When the eggs are excreted, they contain a mature miracidium.

S. haematobium egg: In this species, the eggs are large and have a prominent terminal spine at the posterior end. Length 112 to 174 µm.

S. japonicum egg: In this species, the eggs are large and have a prominent terminal spine. Length 112 to 174 µm.

Schistosoma japonicum eggs: These eggs are smaller (68 to 100 µm by 45 to 80 µm) than those of the other species.

Transmission

- Pollution of fresh water with excreta containing Schistosome eggs
- Presence of the snail host
- Snail from genus Biomphalaria
- Human contact with water infested with cercaria

http://www.cdfound.to.it/H TML/sch1.htm
Main intermediate snail hosts that transmit human schistosomiasis

- S. mansoni (Biomphalaria)
  - Africa
    - B. pfeifferi
    - B. alexandrina
    - B. suavis
  - America
    - B. glabrata
    - B. straminea
    - B. tenagophila

- S. intercalatum (Bulinus)
- S. hematobium (Bulinus)
- S. japonicum (Oncomelania)
- S. mekongi (Tricula)

- M. catalinensis
  - Snail intermediate host

Schistosome miracidium
- Eggs hatch releasing miracidia
  - 200 μm length and 40 μm diameter
  - Swims at 2 mm/sec by beating of the cilia
  - Remain infective for 8-12 h
  - Infects the snail
  - They accumulate around the snail or in a drop of snail-conditioned water

Cercaria in the water
- First escape into the hemolymph and then through the snail’s integument
- Swim into the surrounding water to find their definitive host
- Swims by alternating side-to-side rhythmic contractions
- It is composed of a body 125 μm long by 25 μm in diameter to which a 200 μm long tail is attached

Intramolluscan stages
- The miracidium penetrates the snail
  - It sheds the epithelium and remodels its surface (2 h approx.)
  - A new surface layer appears around the newly formed sporocyst
- A new syncytial tegument is formed
- Primary sporocyst: hollow, fluid filled germinal sac
- Daughter sporocysts in less than a week (35-600)
- Cercaria by 3-4 weeks after infection (1500/day for 18 days)

Morphology of Adult Schistosoma

Female worms: thin and long – resides in the gynecophoral canal of male
  - reproductive organs are arranged linearly

Both sexes have oral sucker and acetabulum.
Intestine (cecum) divides, then fuses near middle of the body.

Male worms: shorter and stouter than females.
  - males have
    - ventral longitudinal groove in which the female resides
  - several testes are located behind the acetabulum
Differences of Adult Schistosoma

Adults of the 3 species differ in:
1.
2.
3.
4.

However, there is overlap of many characteristics.
We will not be responsible for identification to species.

Differences of Schistosoma Eggs

Specific diagnosis is determined by ID of eggs

*S. mansoni* - elliptical egg with ______________________________________

*S. haematobium* - elliptical egg ______________________________________

*S. japonicum* - round to oval egg with __________________________________

Differences of Adult Schistosoma

Pathogenesis of Schistosomiasis

Eggs that do not leave the body are swept to the pre-sinusoidal capillaries of the liver and are trapped there (or in the bladder wall).

The immune system responds and walls off the eggs with a granuloma, the egg dies.

Continuous stimulation of the immune system leads to regulation or fibrosis (less or more morbidity).

Periportal fibrosis leads to portal hypertension.

Portal hypertension leads to collateral esophageal varices, blood circulation, and/or mortality (Sm; Sj).

Continuous aggravation in the bladder wall leads to carcinoma of the bladder (Sh).

Schistosome life cycle

Eggs passed in feces or urine

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Clinical symptoms

- Schistosomiasis is an immunologic disease.
- Symptoms are rarely seen except in heavily infected individuals.
  - Skin penetration-local dermatitis: within 24 h
  - Migration of the schistosomula: chills, fever, sweating, cough, diarrhea, leukocytosis
  - Acute phase (Katayama diseases by S. japonicum)
    - fever, chills, headache, anorexia, urticaria, and diffuse megaly, lymphadenopathy and diffuse vasculitis lesions
    - 2-3 weeks after the infection and usually lasts 1-2 months (typhoid fever!
  - Chronic disease-most important
    - Fatigue, bowel and bladder symptoms, hepatic dysfunction
    - Hepatosplenomegaly because of portal obstruction
    - S. hematobium: urinary tract infection, inflammation, squamous cell epithelioma, hydronephrosis and fibrosis of ureters

SCHISTOSOMA HAEMATOMIUM

causes urinary schistosomiasis, schistosomal hematuria, vesical schistosomiasis, or urinary bilharziasis

Differences in Life Cycles of 3 species
**Geographic distribution**

- **Schistosoma mansoni**
  - Africa (52 countries), Caribbean, Eastern Mediterranean, South America

- **Schistosoma japonicum**
  - Asian countries and the Pacific region

- **Schistosoma haematobium**
  - Africa (54 countries), Eastern Mediterranean

- **Schistosoma intercalatum**
  - African Countries (10)

**Geographical distribution**

Tropical and subtropical. Africa, Iran, Iraq, Saudi Arabia, Yemen, Syria, India, Mauritius, Malagasy Republic, Zanzibar

**Pathogenesis**

**Eggs trapped in the bladder wall and surrounding tissues cause inflammatory formation of granulomata reactions with the**

(contains egg, toxic products, eosinophils, epitheloid cells, and lymphocytes).

Many of the eggs die and become calcified known as “sandy producing what are patches” in the bladder.

In heavy infection, eggs can be carried to other parts of the body.

**Schistosoma haematobium**

Distribution - through Africa and in parts of the Middle East
Co-exists with ___________ in much of Africa especially along the Nile River Valley.

**PATHOGENESIS**

Skin rash at site of cercarial penetration (swimmer’s itch) within a few days after penetration, the young flukes become coated with host red cell antigens and histocompatibility antigens, so they are not recognized as foreign and live free from host attack to develop and produce eggs for long periods.

It is the eggs not the adult flukes which are responsible for the clinical features and damage to the bladder or ureters.
SYMPTOMATOLOGY

In some areas, S. haematobium infection has been linked to an increase in Salmonella typhi and S. paratyphi carriers following acute infection. Patients are more likely to become urinary rather than fecal carriers. Patients may also exhibit a syndrome of chronic, intermittent, enteric bacteremia that clinically resembles Kala-azar. Both of these chronic bacterial infections have been attributed to a mechanism of adhesion of the bacteria to the integument of the intravascular schistosomes.

LABORATORY DIAGNOSIS:

Specific finding the eggs or occasionally the hatched miracidia in the urine. Occasionally, eggs can be found in faeces detecting eggs in rectal biopsy or bladder mucosal biopsy.

Eggs of S. haematobium. White ciliated, and rapidly motile arrows show the position of the miracidium.

Pathology of Schistosoma haematobium

1. Eggs in the tissues of the urinary bladder cause inflammation. Intense pain occurs at end of urination.

2. Blood in the urine is common.

3. Deposition around eggs in the urinary bladder wall is common and tissues lose their elasticity.

4. Infection also increases the likelihood of...

Again, all pathology is caused by the eggs. Adults cause no pathology.
NON-SPECIFIC FINDINGS
hematuria
proteinuria
cells, especially eosinophils can
often be found in the urine
bacteriuria may accompany urinary
schistosomiasis

Schistosoma haematobium egg

Schistosoma mansoni

Diagnosis
- Travel history (in non-endemic areas).
- A history of dermatitis or Katayama fever.
- Urine dipstix for blood +/- protein
  (terminal haematuria)
- Blood eosinophilia
- Urine microscopy (for the ova)
- Stool microscopy
- Rectal biopsy
- Serological markers

Schistosoma mansoni adult male
and female
GEOGRAPHICAL DISTRIBUTION

Parts of Africa, Middle East, South America (Brazil), West India

Biomphalaria snail

PATHOGENESIS/SYMPTOMATOLOGY

Gastrointestinal symptoms:
- skin rash after cercarial penetration
- flukes acquire host antigen protecting them from host immune response
- eggs penetrate through the intestinal wall and are excreted in the faeces often with blood and mucus
- host reaction to eggs leads to the formation of granulomata, ulceration, and thickening of the bowel
- a proportion of the eggs reach the liver through the portal vein
- reaction to the eggs causes thickening of the portal vessels known as claypipe-stem fibrosis
- hepatomegaly with fibrosis

Splenomegaly
Portal hypertension
Ascites
Ova can be deposited in the spinal cord, lungs, and other organs of the body
Salmonella infections can become chronic and prolonged
Pathology of *Schistosoma mansoni*

1. Eggs in the ____________ stimulate host inflammatory reaction followed by fibrous scar tissue deposition.
   - Fibrous scar tissue forms nodules along the large intestine.
   - Some eggs make their way through the intestine to enter the feces.

2. Eggs are swept up the hepatic portal vein into the ____________
   - is followed by scar tissue formation of ____________
   - Cirrhosis and hepatosplenomegaly occur

**Schistosoma mansoni egg in section of liver**

**Schistosoma mansoni egg in section of intestine**

**LABORATORY DIAGNOSIS**

- finding *S. mansoni* ova in faeces occasionally may also be found in the urine following fecal contamination
- Rectal biopsy especially after a patient has been partially treated

**Pathology of Schistosoma mansoni**

- **Inflammatory reaction to eggs**
- **Fibrous scar tissue nodules**
- **Cirrhosis** – areas of scar tissue in liver
- **Ascites** (accumulation of fluid in abdominal cavity) is common
- This produces the typical "swollen belly" associated with chronic schistosomiasis
SCHISTOSOMA JAPONICUM

OTHER FINDINGS
mucus and blood are often present in fecal specimens
blood eosinophilia for patients with hepatic involvement,
increased liver enzymes,
low serum albumin,
increased serum protein due to increased globulin

GEOGRAPHICAL DISTRIBUTION
China, Philippines, Western Indonesia
Eastern Visayas and Mindanao
Oncomelania snail

PATHOGENESIS/SYMPTOMATOLOGY
skin rash at the site of cercarial penetration
20-60 days after infection, patient develop fever,
muscular and abdominal pain, spleen enlargement, urticaria, and eosinophilia (Katayama reaction or Katayama fever)
Reactions to eggs in the tissue can cause intestinal or hepatosplenic disease with dysentery, liver fibrosis, marked hepatosplenoegaly egg deposition in the lungs, CNS, and other parts of the body
Portal hypertension with prominent ascites

LABORATORY DIAGNOSIS
Finding of the ova in faeces
Typical ova on rectal biopsy
Serologic tests
Circumoval Precipitin Test (COPT)
ELISA
OTHER SCHISTOSOMA
SCHISTOSOMA INTERCALATUM
similar to Schistosoma mansoni in terms of life cycle, pathology and clinical feature
intermediate host is the Bulinus snail
SCHISTOSOMA MEKONGI
similar to Schistosoma japonicum in terms of life cycle, pathology, and clinical features
intermediate host is the snail Lithoglyphopsis aperta

OTHER FINDINGS
Mucus and blood in fecal specimen
blood eosinophilia
in patients with hepatic involvement
raised hepatic enzymes,
low serum albumin,
increased total protein due to increased globulin

**Treatment of schistosomiasis**

<table>
<thead>
<tr>
<th>Compound</th>
<th>Type of Compound</th>
<th>Active against</th>
<th>Dosage</th>
<th>Cure Rate</th>
<th>Main Side Effects</th>
<th>Price/adult dose</th>
<th>Mode of Action</th>
<th>Effective treatment also requires host responsiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Praziquantel</td>
<td>Isoquinoline</td>
<td>All Species</td>
<td>40-60mg/kg</td>
<td>70%-100%</td>
<td>Diarrhea, Nausea</td>
<td>$0.25 - $3</td>
<td>Calcium Channel</td>
<td>As with egg excretion</td>
</tr>
<tr>
<td>Oxamniquine</td>
<td>Tetrahydroquinoline</td>
<td>S. mansoni</td>
<td>15-40mg/kg</td>
<td>80%-100%</td>
<td>Dizziness</td>
<td>$2 - $5</td>
<td>Delayed action</td>
<td></td>
</tr>
</tbody>
</table>

**Treatment**
Praziquantrel
40-50 mg/kg Single dose
25 mg/kg Two doses
20 mg/kg Three doses

**Prevention and Control**

Health Education
Control snail vector
Environmental method
Removing the environmental requirement of the snails drainage of breeding sites and proper management of irrigation system
removal of shade or shelter from the sun by clearing vegetation armed bodies of water
prevention of breeding on the banks of streams or irrigation canals by living these concretes or making these more perpendicular
acceleration of flow of water by proper grading and clearing of the stream bed and removal of debris
construction of ponds if the area cannot be drained
covering snail habitats with land fills
Chemical method
Environmental sanitation
Swimmer's Itch

3 genera of schistosomes cause swimmer's itch in the Midwest:
- **Gigantobilharzia** - adult blood flukes occur in mesenteric veins of
- **Trichobilharzia** - adults in mesenteric veins of
- **Schistosomatium** - adults in mesenteric veins of

Swimmer's itch was first described at U. Michigan Biological Station in 1936 and extensively studied in Wisconsin in the 1930's & 40's. The problem is common in Wisconsin lakes and occasional outbreaks occur (recent outbreak in Half Moon Lake in Eau Claire).

### Life Cycle of Swimmer's Itch

Humans become infected when snail intermediate hosts of these parasites release cercariae, which mistake humans for the definitive host.

Infection results when cercariae penetrate epidermis but are unable to enter the dermis. Cercariae die in the epidermis and an immunological response occurs against the dead cercariae. Formation of a dermatitis (dermatitis) occurs at the sites of cercarial penetration. Intense itching also occurs.

### Pathology of Swimmer's Itch

- Dermatitis is mild in initial exposures but may become severe in persons exposed to previous outbreaks – called rash.
- Itching and breaking of skin may lead to secondary infections.
- Rash has been misdiagnosed as a rash.

**Swimmer's Itch**

Swimmer's itch is caused by _____________________________

Swimmer's itch is common in Midwest and along coastal areas of the U.S.
- Itch was first described at U. Michigan Biological Station in 1936.
- Extensively studied in Wisconsin in the 1930's & 40's.
- Problem is common in Wisconsin lakes and occasional outbreaks occur (recent outbreak in Half Moon Lake in Eau Claire).

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Swimmer's Itch

Swimmer's itch is a problem to ___________________________________________
- Swimming beaches are common sources of infection.
- First outbreaks occur in late June and early July when vacationers are numerous.

But problem is often kept hushed, as resort owners don’t want you to know that swimmer’s itch is a problem in their lake, as they don’t want to lose business.

Treatment and Prevention of Swimmer’s Itch

TREATMENT - topical creams such as ___________________________ reduce the itching and ___________________________ reduce the inflammatory response

PREVENTION – best way to reduce swimmer’s itch is to

- Snail control efforts (using copper sulfate as molluscicide) have been attempted but generally have been unsuccessful.
- Removal of bird or mammal definitive hosts is not possible.