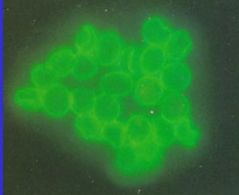


## Ecology of Disease

Weekly theme: **WATER**  
Lecture: **Biota**



Prof Peter O'Donoghue

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## Water as a medium

for carriage of passive infectious stages

- non-motile encysted forms (ova/cysts/spores)
- resistant to external environmental conditions
- no amplification in water (non-proliferative)


for transmission of active infectious stages

- motile host-seeking forms (larvae/miracidia/cercaria)
- adapted to aquatic existence
- amplification during aquatic cycle (multiplication)



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## TREMATODES (flukes)

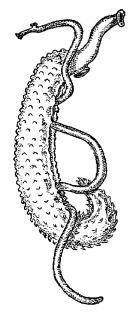
VASCULAR	- blood flukes	
ENTERIC	- intestinal flukes	
TISSUE	- liver flukes	

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## *Schistosoma* spp. (blood flukes)

Order: Strigeatida  
Family: Schistosomatidae

- blood flukes – adults in blood vessels
- unique trematodes as sexes separate (female lying in male gynecophoral canal) [schisto-soma = split body]
- no metacercaria, cercaria penetrate skin
- important human and animal parasites in Africa, Asia & South America
- haematuria (bloody urine) well known throughout history (Egyptian mummies 3000-1000BC, Napoleon's army 1800AD)




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## Vascular trematodes


Parasite	Definitive host	Snail vector	Locality
<b>Schistosomes</b>			
<i>S. mansoni</i>	human/rodents	<i>Biomphalaria</i>	Africa, America
<i>S. japonicum</i>	human/ruminants	<i>Oncomelania</i>	SE Asia
<i>S. haematobium</i>	human/primates	<i>Bulinus</i>	Africa

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## *Schistosoma* spp. (blood fluke)



adults 10-20 mm  
in blood vessels



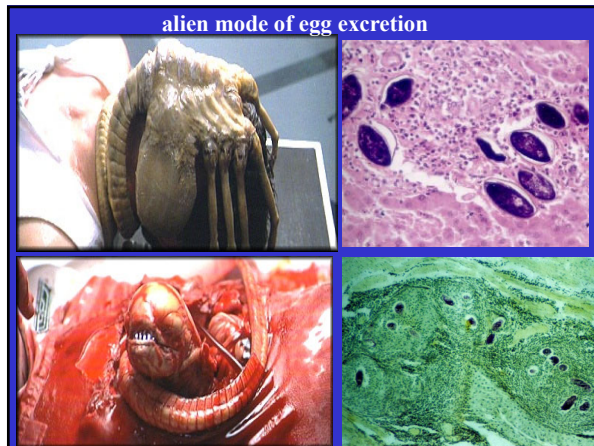
snail vector



non-operculated eggs  
passed in faeces/urine

cause bilharzia (fibrosis, portal hypertension)

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**Schistosoma spp. adults**

<i>S. mansoni</i>	<i>S. japonicum</i>	<i>S. haematobium</i>
inferior/superior mesenteric veins	inferior/superior mesenteric veins	vesical/prostate/uterine plexuses

eggs migrate into intestines and passed in faeces	eggs migrate into intestines and passed in faeces	eggs migrate into bladder/urethra passed in urine
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**Schistosoma spp. eggs**

<i>S. mansoni</i>	<i>S. haematobium</i>	<i>S. japonicum</i>
120-180 x 45-75 $\mu\text{m}$	110-170 x 40-70 $\mu\text{m}$	55-85 x 40-60 $\mu\text{m}$

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**Amplification in snail hosts**

miracidium

freshwater snails

sporocysts/redia

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**Snail hosts**

<i>S. mansoni</i>	<i>S. haematobium</i>	<i>S. japonicum</i>
<i>Biomphalaria</i>	<i>Bulinus</i>	<i>Oncomelania</i>

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**Host finding**

- fork-tailed cercaria 400-600  $\mu\text{m}$
- rapid swimmer
- penetration machine

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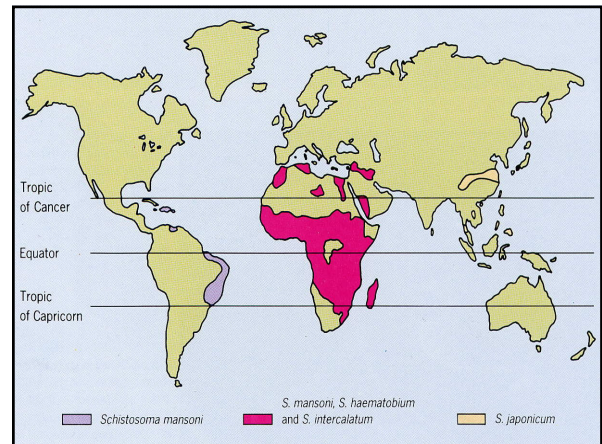
**Water-borne transmission**

prepatent period  
5-10 weeks

- eggs voided
- f-I miracidia
- sporocysts in snail
- f-I cercariae
- penetrate skin
- schistosomulum carried to liver
- adults in vasculature

**Schistosoma (Bilharzia)**  
*S. mansoni* SAMBON 1907, *S. haematobium* WEINLAND 1858,  
*S. japonicum* KATSURADA 1904

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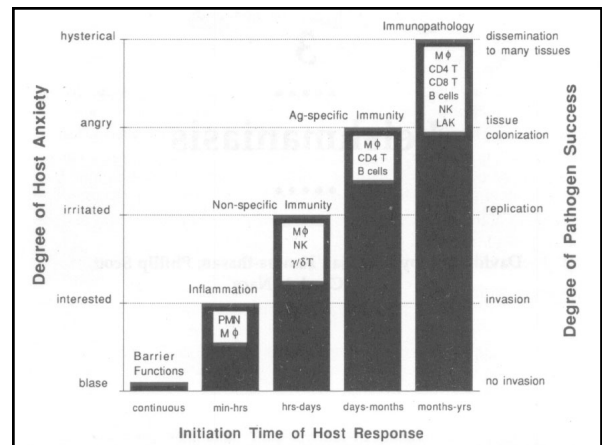
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**Pathogenesis**

Three disease phases

- migratory phase, characterized by cercarial dermatitis ('swimmers itch' more marked with bird schistosomes)
- acute phase (Katayama fever), characterized by serum sickness coincident with first egg release
- chronic phase, characterized by host granulomatous responses to eggs deposited in tissues

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**Pathogenesis – migratory phase**

- often asymptomatic
- transient dermatitis due to cercarial penetration in sensitized patients
- occasionally pulmonary lesions, pneumonitis

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**Pathogenesis – acute phase**

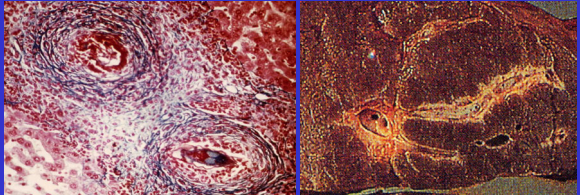
- acute allergic responses when eggs first produced
- eggs pass through tissues aided by enzymes from enclosed miracidia
- cause haematuria, pyrexia, lymphadenopathy, eosinophilia, liver tenderness, diarrhoea ('Katayama syndrome')

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### Pathogenesis – chronic phase

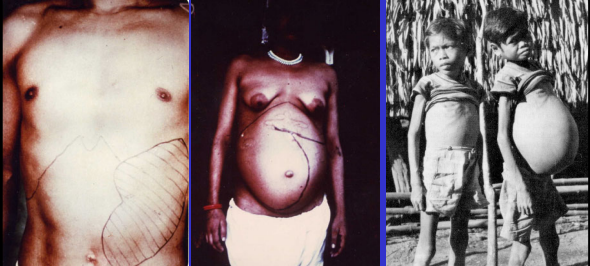
- eggs trapped in tissues surrounded by inflammatory cells (forming characteristic pseudotubercles)
- these coalesce to form larger granulomatous reactions (polyps) and eggs eventually calcify
- Symer's periportal fibrosis, intestinal polyposis, glomerulonephritis, cardiopulmonary problems



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### Pathogenesis - chronic


- portal hypertension leads to hepatomegaly, splenomegaly, and possibly ascites
- also gross enlargement of oesophageal and gastric veins (varices) which sometimes burst



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### Problem


- contamination of water by urine/faeces
- free-swimming miracidia seeking water snails
- sporocyst amplification in snail tissues (vectors or intermediate hosts)
- liberated cercaria seeking definitive hosts
- active penetration of skin
- chronic infections
- inflammation/immunopathology



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### Control

- stop water contamination (faeces/urine)
- treat water (disinfectants, standing)
- restrict immersion in water (rice paddies?)
- reduce snail populations
  - drain swamps
  - chemical treatment
- treat infected individuals
- vaccination?



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### TREMATODES (flukes)

VASCULAR	- blood flukes	
ENTERIC	- intestinal flukes	
TISSUE	- liver flukes	

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### *Fasciola* (liver fluke)


Order: Echinostomatida  
Family: Fasciolidae

- large leaf-shaped flukes (unmistakable)
- adults in bile ducts
- metacercariae on plants
- primarily a zoonotic disease




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### *Fasciola hepatica* (liver fluke)



adults 2-3 cm  
in bile ducts



operculate eggs 140x80µm  
passed in faeces

snail vector

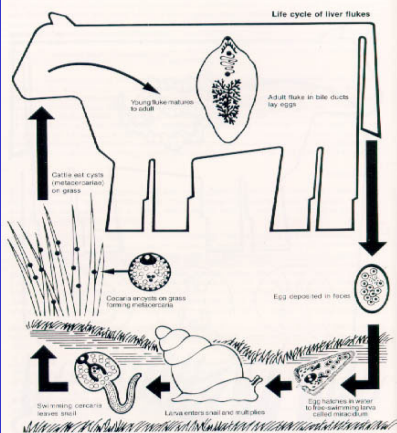
common in sheep/cattle on wet/irrigated pasture  
may also infect humans, goats, pigs, macropods, rats, rabbits, etc

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### Heteroxenous transmission

prepatent period  
8-13 weeks

- eggs voided
- f-1 miracidium
- form sporocysts rediae in snails
- f-1 cercariae
- form metacercariae on vegetation
- eaten by herbivore
- adult in liver

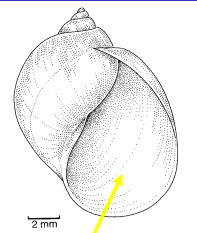


Life cycle of liver flukes

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### Snail intermediate hosts


- snails of Family Lymnaeidae
- snails "dextral" (right-handed)  
[aperture on right]
- lack operculum (trapdoor)
- high population density under right conditions (up to 3,000/m<sup>2</sup>)
- capable of aestivation over dry summers by burrowing into soil

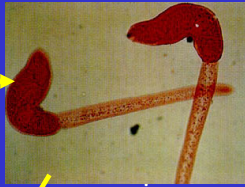


aperture

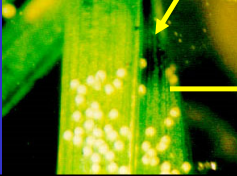
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### Amplification in snail and pasture contamination





cercaria



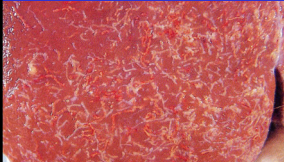
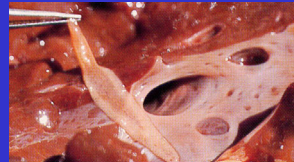


metacercaria

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### Pathogenesis - acute



- ingested worms penetrate intestine, wander in body cavity, penetrate liver, then enter main bile ducts (~7 weeks)
- acute disease (liver rot) caused by mass migration of juveniles
- traumatic tissue damage, coagulative necrosis, haemorrhage, urticaria, eosinophilia, leukocytosis, pallor, anaemia
- predisposes for anaerobic *Clostridium perfringens/novyi* which produce toxins leading to rapidly fatal 'black disease' in sheep

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### Pathogenesis - chronic

- chronic infections cause fibrotic hardened liver, biliary epithelial hyperplasia, duct pipestem fibrosis, cholangitis
- mechanical damage, metabolic by-products, obstruction
- acute epigastric pain, pruritis, jaundice, weight loss
- metacercariae may wander (subcutaneous lumps common)

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## Problem

- contamination of water by faeces
- free-swimming miracidia seeking water snails
- sporocyst amplification in snail tissues
- cercaria encysting on aquatic vegetation
- ingested with pasture/water plants
- inflammation/fibrosis/obstruction



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## Control

- reduce contamination (faeces)
- reduce snail populations
  - habitat destruction (drain swamps)
  - chemical treatment (molluscicides)
- avoid grazing wetlands/irrigated pastures
- avoid aquatic/semi-aquatic vegetables
- treat infected individuals

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## Water - beware!



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## Water-borne diseases

- ingestion of contaminated drinking water not the only route of transmission
- ingestion of aquatic plants contaminated with encysted metacercariae
- contact with water containing motile host-seeking cercariae

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