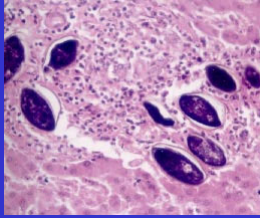


Biomedical Parasitology


Trematodes - tissues



Prof. Peter O'DONOGHUE

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TISSUE TREMATODES



- Fasciola* spp.
- Clonorchis sinensis*
- Opisthorchis* spp.
- Dicrocoelium dendriticum*
- Paragonimus* spp.

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Tissue trematodes

Parasite	Definitive host	Vector	Metacercaria	Locality
Liver flukes				
<i>Fasciola</i>	human/ruminant	snails	plants	worldwide
<i>Clonorchis</i>	human/dog/cat	snails	fish	China
<i>Opisthorchis</i>	human/dog/cat	snails	fish	Eurasia
<i>Dicrocoelium</i>	human/ruminants	snails	ants	Old World
Lung flukes				
<i>Paragonimus</i>	human/tiger/mink	snails	crabs	Indochina

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Fasciola (liver fluke)

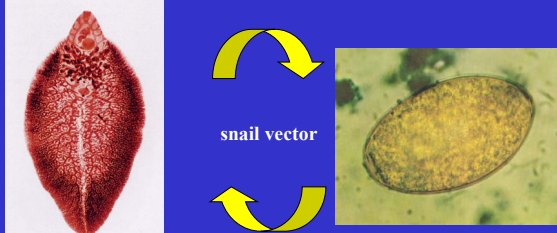
Order: Echinostomatida
Family: Fasciolidae



- 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

snail vector

operculate eggs 140x80µm passed in faeces

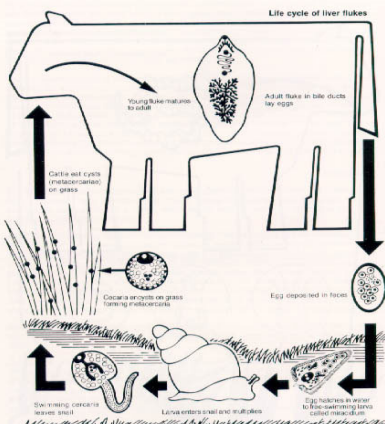
common in sheep/cattle on wet/irrigated pasture
may also infect humans, goats, pigs, macropods, rats, rabbits, etc

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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 metacercaria on vegetation
- eaten by herbivore
- adult in liver

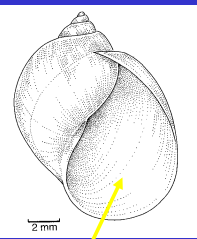


Life cycle of liver flukes

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Snail intermediate hosts

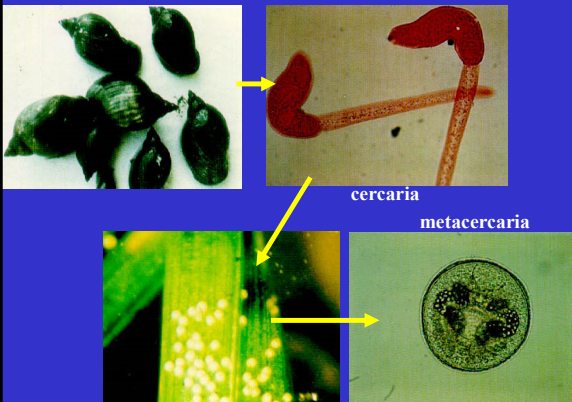
- snails of Family Lymnaeidae
- snails "dextral" (right-handed)
[aperture on right]
- lack operculum (trapdoor)
- high population density under right conditions (up to 3,000/m²)
- capable of aestivation over dry summers by burrowing into soil



aperture

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Amplification in snail and pasture contamination

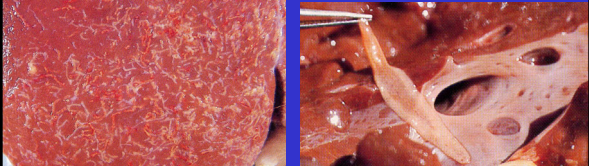


cercaria
metacercaria

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Pathogenesis - acute

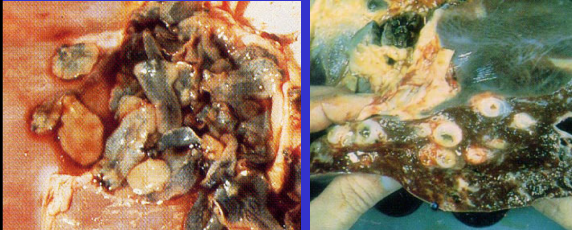
- ingested worms penetrate intestine, wander in body cavity, penetrate liver, then enter main bile ducts (~7 weeks)
- acute disease (liver rot) caused by mass migration of juveniles
- traumatic tissue damage, coagulative necrosis, haemorrhage, urticaria, eosinophilia, leukocytosis, pallor, anaemia
- 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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Clonorchis/Opisthorchis (liver flukes)

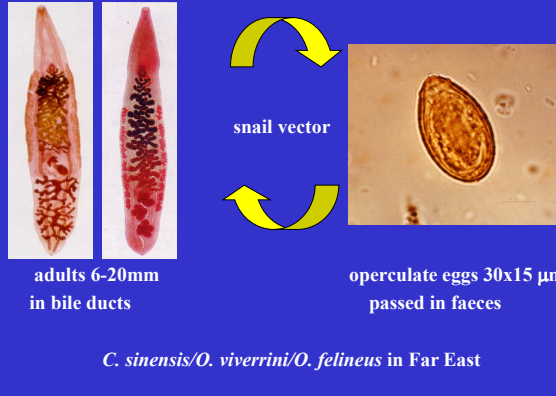
Order: Opisthorchiidae
Family: Opisthorchidae

- narrow elongate adults
- in distal bile ducts
- form metacercariae in freshwater fish



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Clonorchis/Opisthorchis (liver flukes)



adults 6-20mm
in bile ducts

operculate eggs 30x15 μm
passed in faeces

C. sinensis/O. viverrini/O. felineus in Far East

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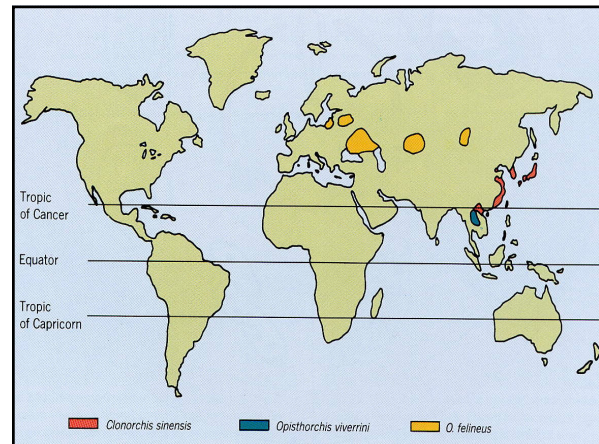
Food-borne transmission

prepatent period
2-5 weeks

- eggs voided
- ingested by snails
- miracidia form sporocysts, rediae
- f-1 cercariae
- metacercaria in freshwater fish
- eaten by humans

Clonorchis sinensis LOOSS 1907
Opisthorchis felinus BLANCHARD 1895

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Pathogenesis

- lesions due to mechanical irritation/toxic products
- light infections, mild inflammation
- heavy infections, thickening of bile ducts, fibrosis, hyperplasia of mucinous glands
- resulting in biliary obstruction, jaundice, aggravated by bile stones, liver abscesses
- recurrent pyogenic cholangitis

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Paragonimus (lung fluke)

Order: Plagiorchiida
Family: Troglotrematidae

- thick fleshy flukes
- adults live as pairs in cysts in lungs
- form metacercariae in freshwater crabs/crayfish

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Paragonimus westermani (lung fluke)

adults 12 mm in lungs

operculate eggs 100x55 μm passed in faeces

snail vector

in crustacean-eating carnivores in Asia, Africa, Americas
prevalent in humans in Korea (crayfish juice cure for measles)

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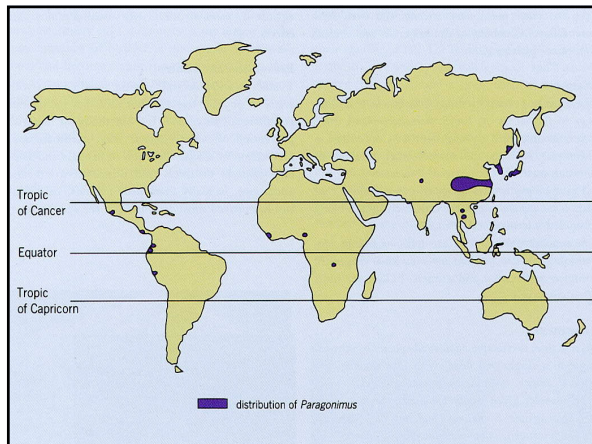
Food-borne transmission

prepatent period
8-12 weeks

- eggs coughed up and voided
- f-1 miracidia
- sporocyst/redia in snails
- f-1 cercaria
- metacercaria in crabs/crayfish
- eaten by humans
- migrate through viscera to lungs

Paragonimus westermani BRAUN 1899
P. kellicotti WARD 1908

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Pathogenesis

- migration through tissues to lungs produces localized haemorrhage and leucocytic infiltrates
- in lungs, pronounced tissue reaction, infiltration of eosinophils and neutrophils, fibrotic capsule
- cysts contain purulent fluid with 'iron-filing' flecks (=eggs)
- perforate bronchioles to release eggs, viscous bloody sputum, dyspnea with chronic bronchitis

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Pathogenesis

- some eggs in lungs swept away by circulation to other tissues (liver, muscles, brain) causing granulomas, calcification
- cerebral complications of paragonimiasis
- symptoms include fever, headache, nausea, vomiting, visual disturbances, motor weakness, localized or generalized paralysis, possibly death

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VASCULAR TREMATODES

Schistosoma mansoni
Schistosoma japonicum
Schistosoma haematobium

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Vascular trematodes

Parasite	Definitive host	Snail vector	Locality
Schistosomes			
<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 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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Schistosoma spp. (blood fluke)

snail vector


adults 10-20 mm in blood vessels 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 μ m	110-170 x 40-70 μ m	55-85 x 40-60 μ m




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Amplification in snail hosts

miracidium
 freshwater snails
 sporocysts/redia

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Snail hosts

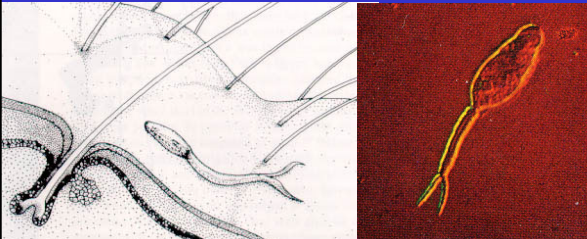
<i>S. mansoni</i>	<i>S. haematobium</i>	<i>S. japonicum</i>
		
<i>Biomphalaria</i>	<i>Bulinus</i>	<i>Oncomelania</i>



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Host finding

- fork-tailed cercaria 400-600 μ m
- rapid swimmer
- penetration machine



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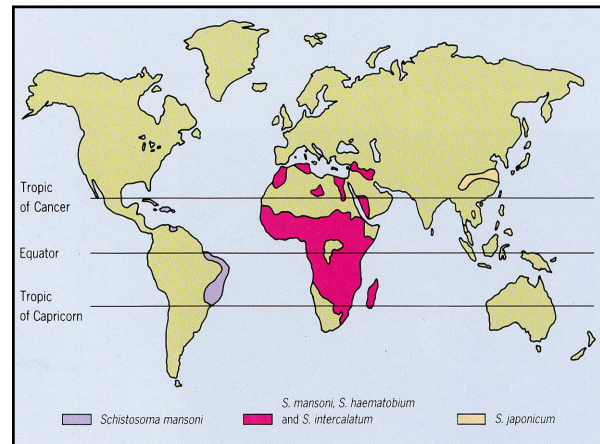
Water-borne transmission

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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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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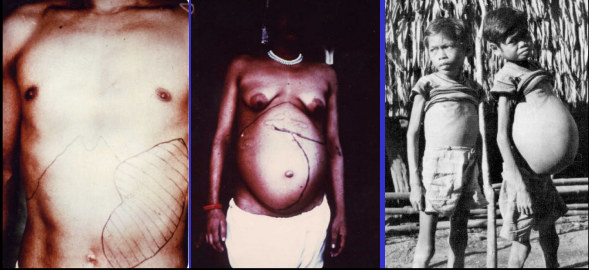
Pathogenesis – chronic phase

- eggs trapped in tissues surrounded by inflammatory cells (forming characteristic pseudotubercles)
- these coalesce to form larger granulomatous reactions (polyps) and eggs eventually calcify
- Symmer'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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TREATMENT - trematodocides

<i>Schistosoma</i> (blood fluke)	praziquantel, niridazole, metrifonate, oxaminiquine
<i>Fasciola</i> (large liver fluke)	bithionol, triclabendazole
<i>Clonorchis/Opisthorchis</i> (small liver flukes)	praziquantel, albendazole
<i>Paragonimus</i> (lung fluke)	praziquantel, bithionol

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Control

- stop water contamination (faeces/urine)
- treat water (disinfectants, standing)
- avoid grazing wetlands/irrigated pastures
- avoid raw aquatic vegetables, shellfish
- restrict immersion in water (rice paddies?)
- reduce snail populations
 - drain swamps
 - chemical treatment
- treat infected individuals
- vaccination?



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Summary

Parasite	Site	Definitive host	Intermediate hosts
<i>Echinostoma</i>	intestines	humans	snails clams
<i>Fasciolopsis</i>	intestines	humans/pigs	snails water plants
<i>Fasciola</i>	bile ducts	humans/ruminants	snails water plants
<i>Clonorchis</i>	bile ducts	humans/piscivores	snails fish
<i>Opisthorchis</i>	bile ducts	humans/piscivores	snails fish
<i>Paragonimus</i>	lung	humans/carnivores	snails crabs
<i>Schistosoma</i>	veins	humans	snails -

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