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Helmintic Infections

M1 Infectious Diseases Sequence
Vernon Carruthers
Cary Engleberg

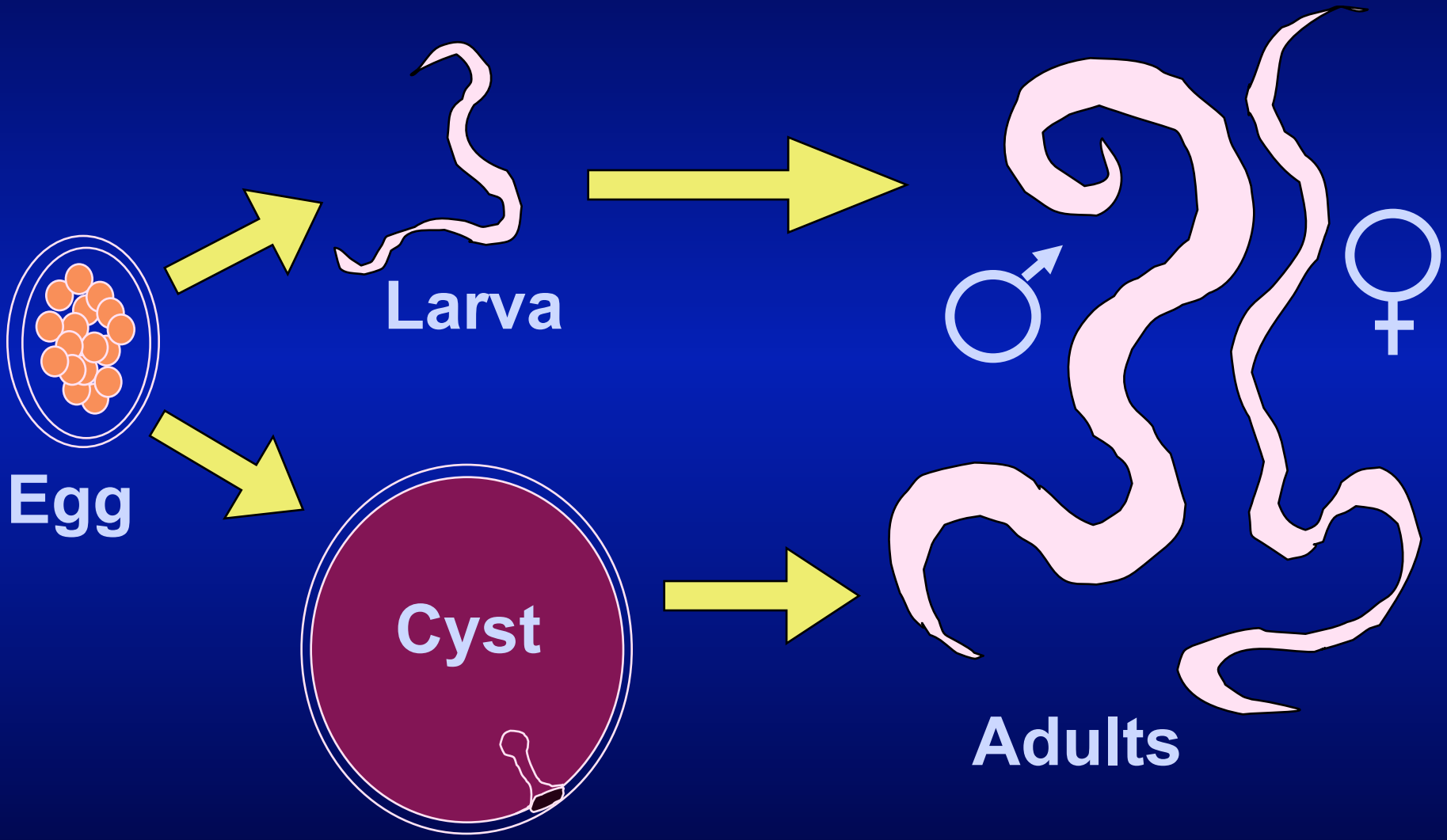
Spring 2009

University of Michigan
Medical School

Infection vs. disease

- successful parasites live in, but do not kill their hosts
- protozoa multiply within hosts
  - expression of disease depends on host factors
- helminths do not multiply within hosts
  - severity of disease depends on parasite burden and immunologic response to parasites

Helminth forms



Helminth modes of entry

- Ingestion (eggs or cysts)
- Arthropod bites (larvae)
- Penetration of intact skin or mucous membranes (larvae)

Spread and tropisms

- Some parasites must migrate to certain locations within the host in order to complete their life cycle
- Non-human parasites, in humans, often fail to migrate properly and become “dead-end infections”

PD-INEL Cary Engleberg

Mechanisms for evading the host response

- antigenic variation - trypanosomes, malaria, giardia
- intracellular infection - malaria, toxoplasma
- encystation* - Toxoplasma, cestodes
- camouflage - schistosomes
- cleavage of ABs or C' components - amoebae, leishmania
- suppression/redirection of the cellular immune response - malaria, leishmania, schistosomes

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* "cyst" has multiple meanings

Tissue damage and host response

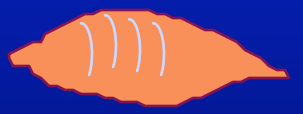
- direct destruction of tissue
- hypersensitivity reactions
- eosinophila
  - —occurs with helminths, not protozoa
  - —results from tissue migration

Classification of helminths

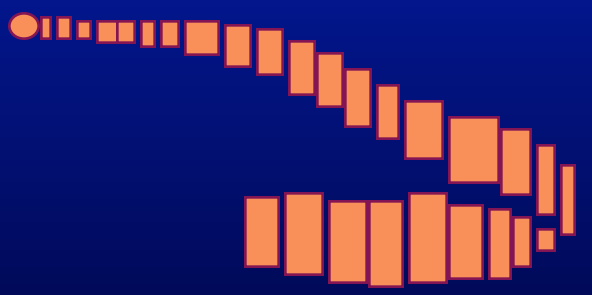
Nematodes (roundworms)

Platyhelminthes (flatworms)

- Trematodes (“flukes”)



- Cestodes (“tapeworms”)



Helmintic diseases

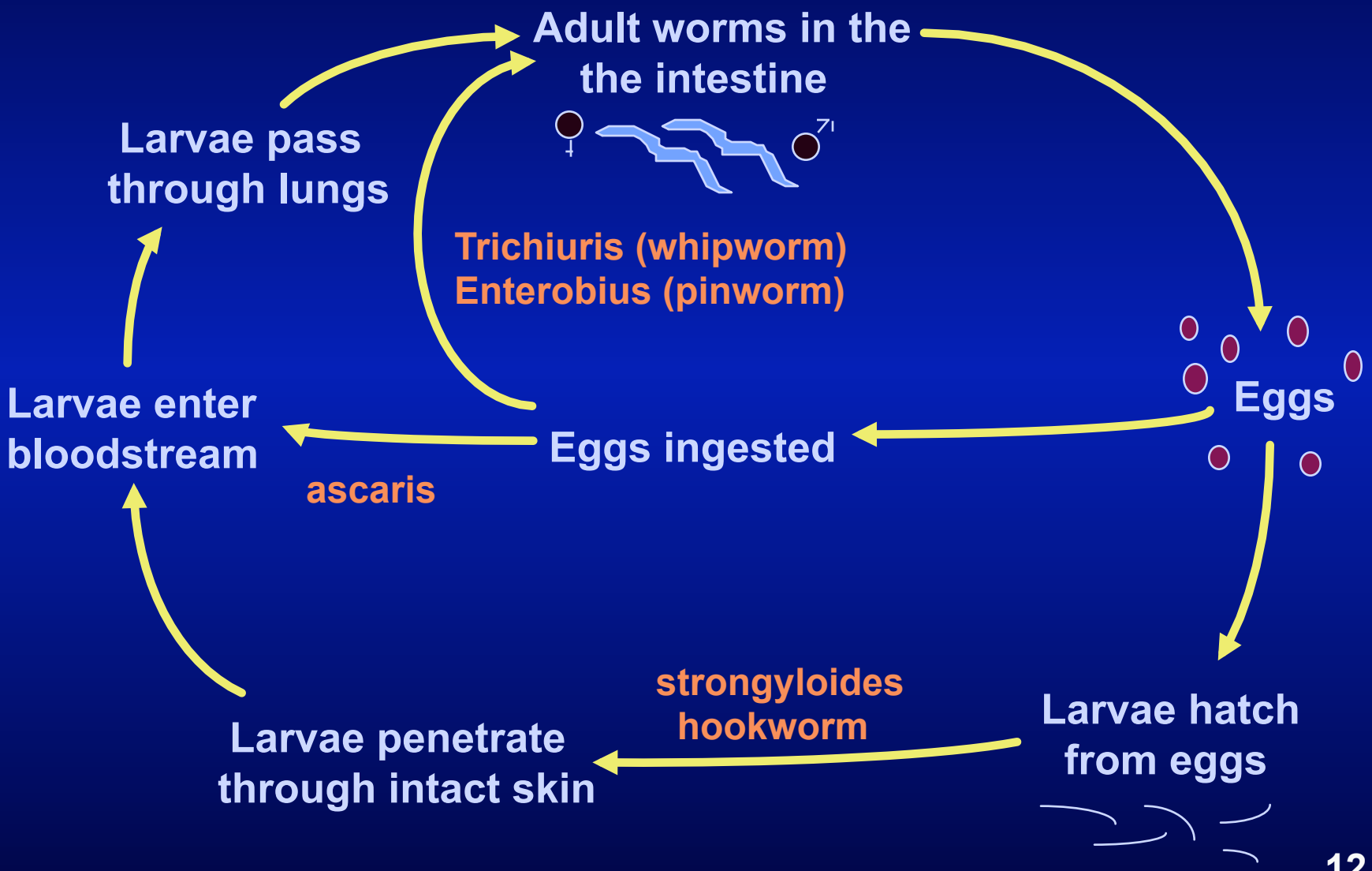
- **Intestinal**
  - —**Others**
  - —**Strongyloides** (autoinfection cycle)
- **Invasive**
  - —**Trichinosis** (muscle pain, uncooked carnivores)
  - —**Filaria** (worms in lymphatics or under skin)
  - —**Schistosomiasis** (liver or urinary tract granulomas and fibrosis)
  - —**Cysticercosis** (cysts in brain, seizures)
  - —**Echinococcus** (massive cysts in liver or lung)

roundworms

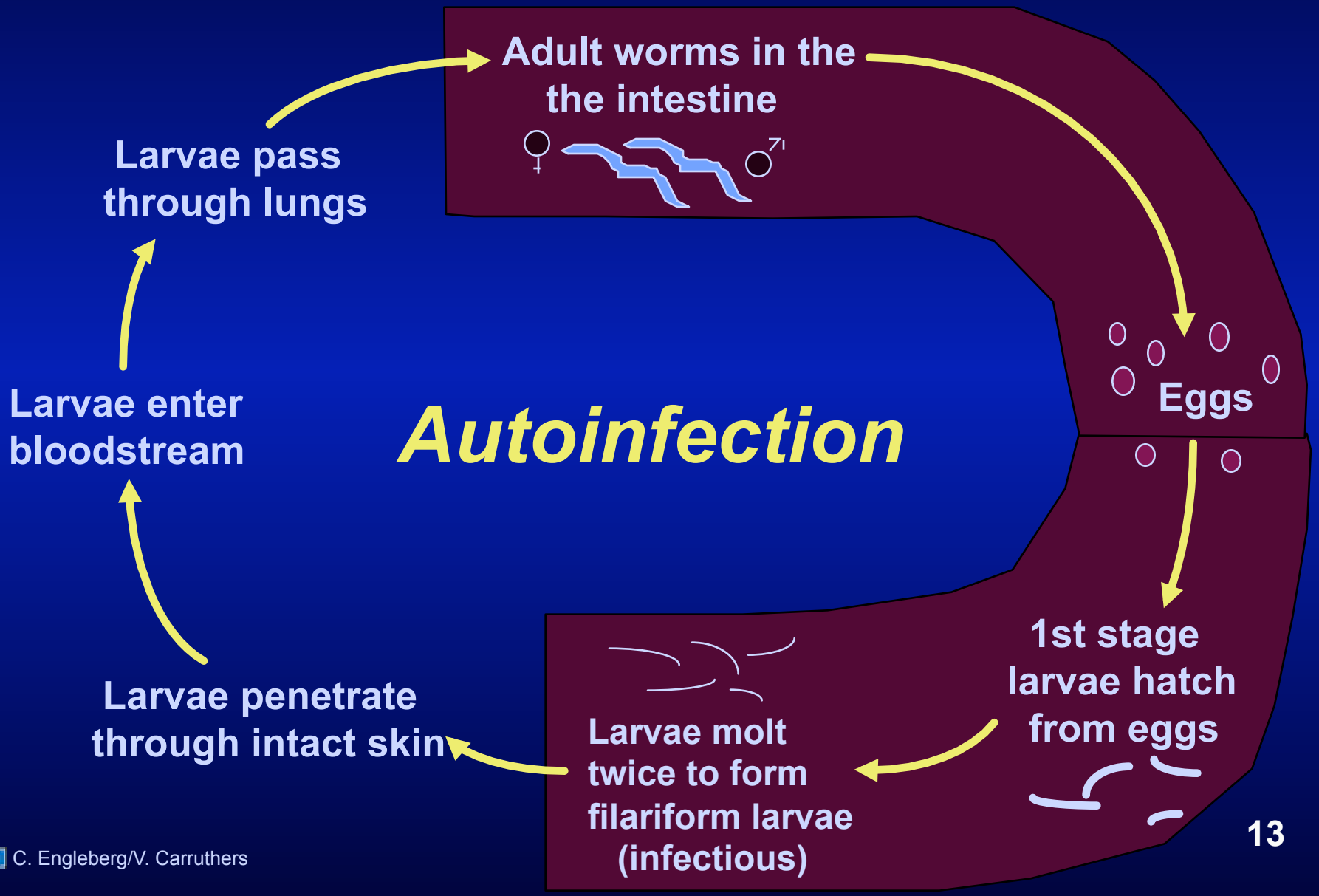
flukes

tapeworms

Intestinal nematodes



Strongyloides life cycle



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Strongyloides - clinical features

- uncomplicated
  - —GI upset
- autoinfection
- hyperinfection
  - —rash
  - —bronchspasm, chest X-ray infiltrates
  - —diarrhea
  - —profound eosinophilia
  - —recurrent Gram-negative bacteremia

Trichinosis

Trichinella spiralis - life cycle

- "cycle of carnivorism" among hogs and rats
- humans ingest encysted larvae in infected, undercooked pork
- larvae exist in stomach and burrow into small intestinal mucosa
- adult males and female reemerge and produce larvae which penetrate intestine and circulate in bloodstream
- larvae enter skeletal muscle cells and encyst

PD-INEL Source Undetermined

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Trichinosis cases, by source of infection, U.S.,1981

Pork products	
sausage	93
other	44
unspecified	9
Non-pork products	
hamburger	18
bear	10
other wild animals	7
Unknown	7
	188

Clinical features of trichinosis

- Most common sxs:
  - —muscle pain and tenderness
  - —fever +/- chills
  - —edema (often periorbital)
- >10% eosinophilia (often ~50%)
- elevated creatine phosphokinase (CPK)
- +/- chronic neurologic/myocardial sxs
- self-limited (2% mortality)

Treatment of trichinosis

- antihelmintic (albendazole) to kill any intestinal adults
- steroids to relieve inflammatory reactions
- antipyretics

Filaria

Life cycles of two types of filaria

	Arthropod vector	Adult worm pairs	Larvae (microfilariae)
Lymph-dwelling (e.g, *Wuchereria bancroftii*)	mosquitoes	peripheral lymphatics	circulate in bloodstream
Skin-dwelling	biting flies	skin nodules or migratory	migrate through dermis

Microfiliaria found in the blood of lymph dwelling species

Long-term consequences of persistent lymph-dwelling filarial infection:

Blockage of lymph drainage with chronic lymphedema (elephantiasis)

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Life cycles of two types of filaria

	Arthropod vector	Adult worm pairs	Larvae (microfilariae)
Lymph-dwelling (e.g, *Wuchereria bancroftii*)	mosquitoes	peripheral lymphatics	circulate in bloodstream
Skin-dwelling (e.g., *Onchocerca volvulus* & *Loa loa*)	biting flies	skin nodules or migratory	migrate through dermis

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Black fly: vector of Onchocerciasis

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O. volvulus microfilaria (a skin-dwelling species) in skin snip

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Depigmentation due to chronic microfilarial production, degradation, and allergic host responses in the skin

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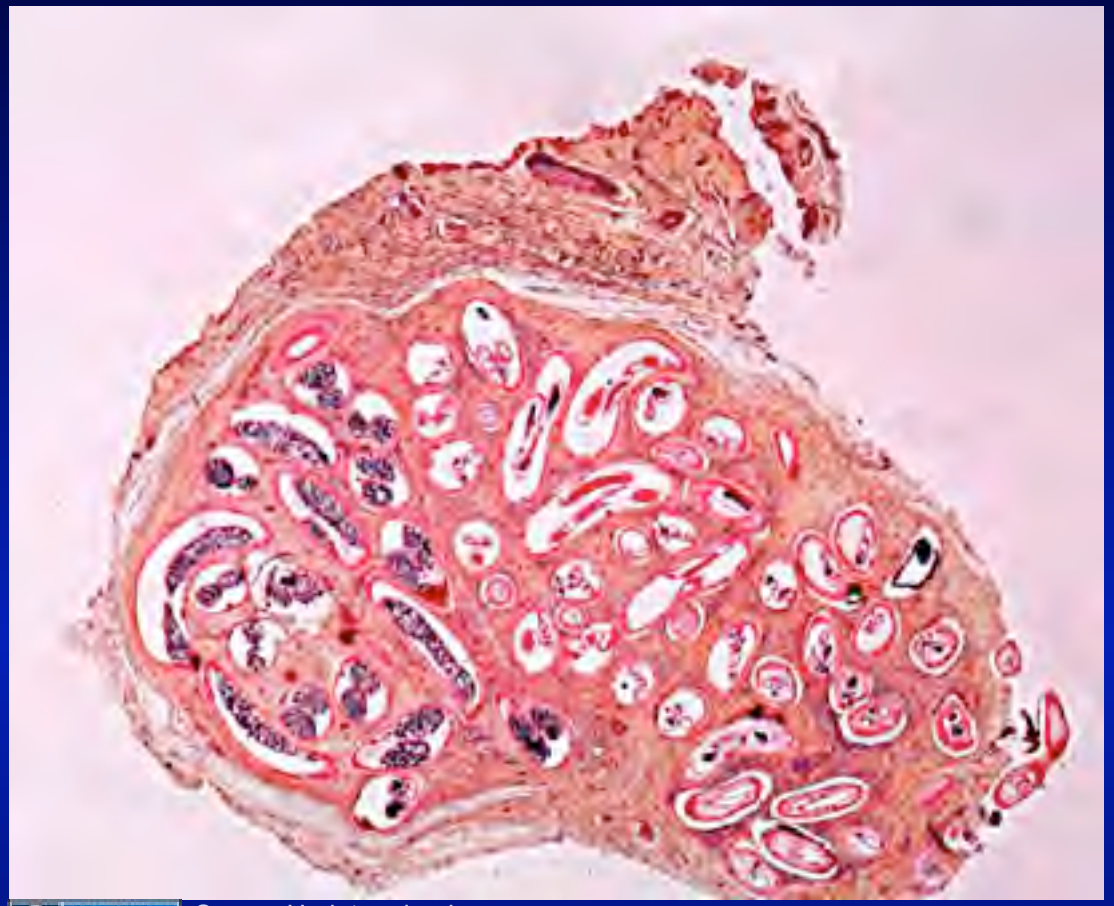

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O. volvulus **skin nodule removed and sectioned, showing cross-sections of male and female adult worms (source of microfiliariae)**

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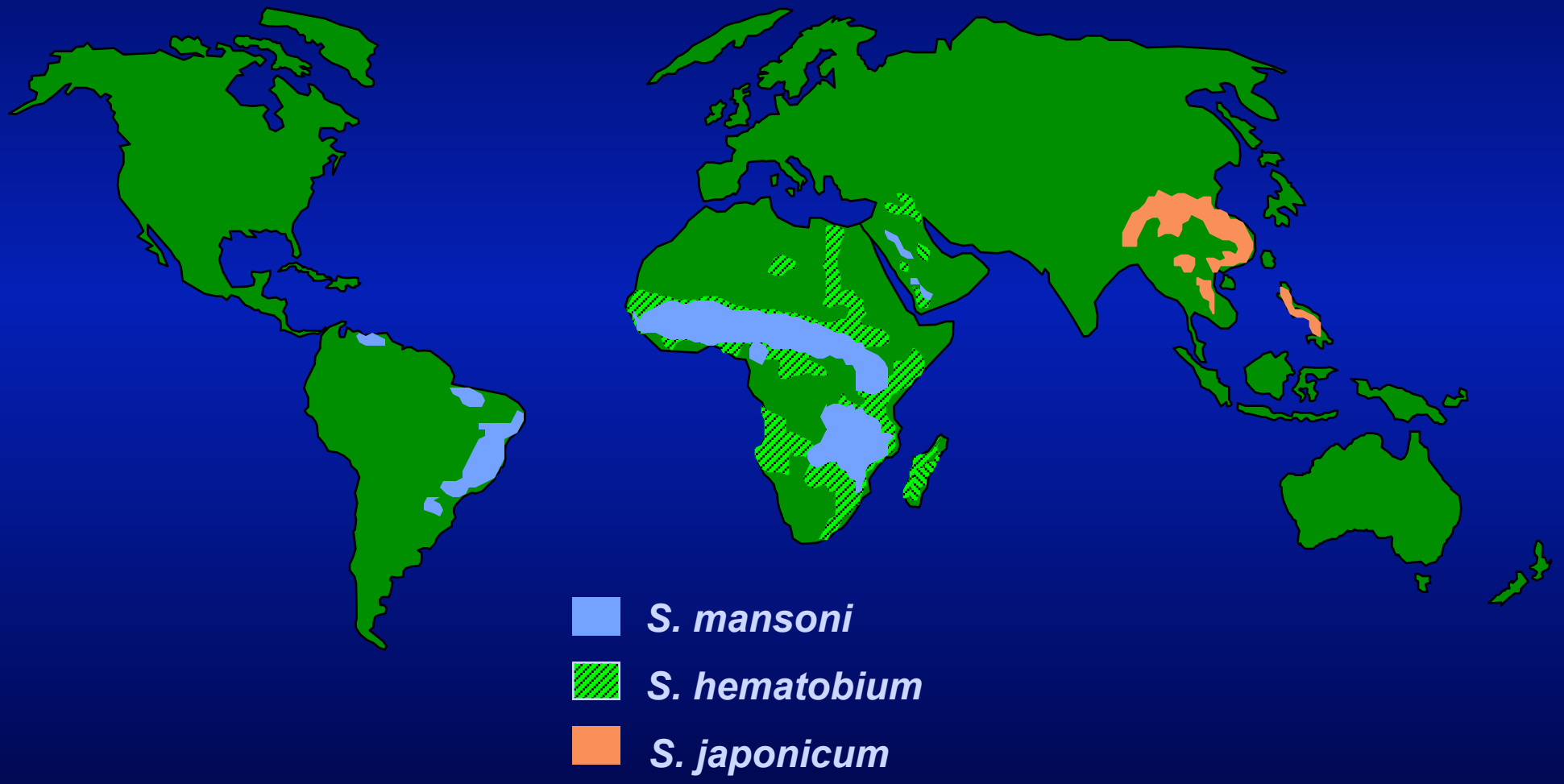
Onchocerciasis (”River blindness”)

Role of endosymbiont *Wohlbachia* sp. in filiaria infection

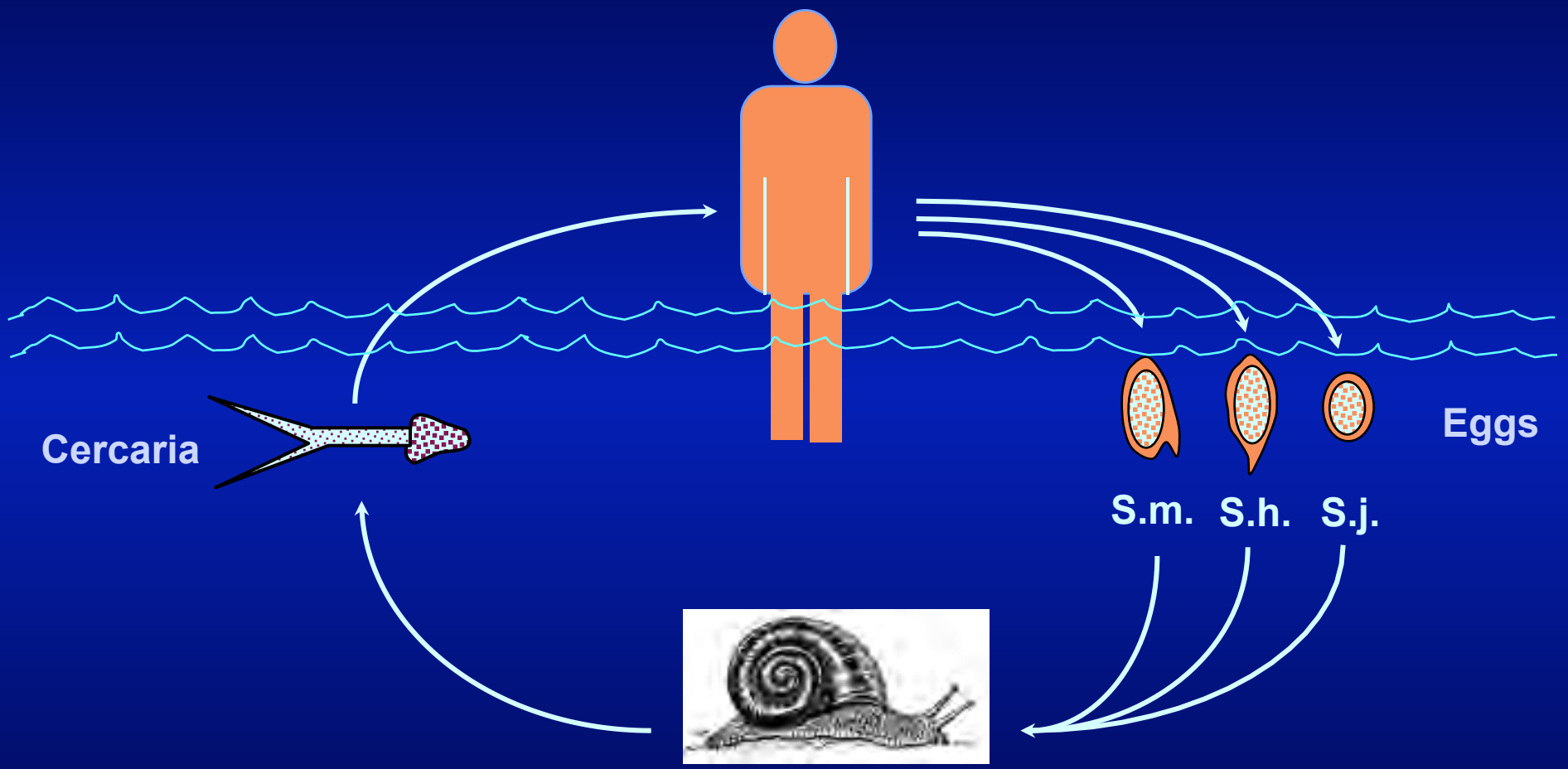
- Rickettsia-like organisms required for fecundity and viability of filaria
- Wohlbachia-free worms produce less inflammation in tissue (? LPS)
- Implications for rx:
  - —ivermectin kills microfilaria only
  - —tetracycline may destroy adult worms

Schistosomiasis

Geographic distribution of schistosomiasis



Schistosomiasis - life cycle



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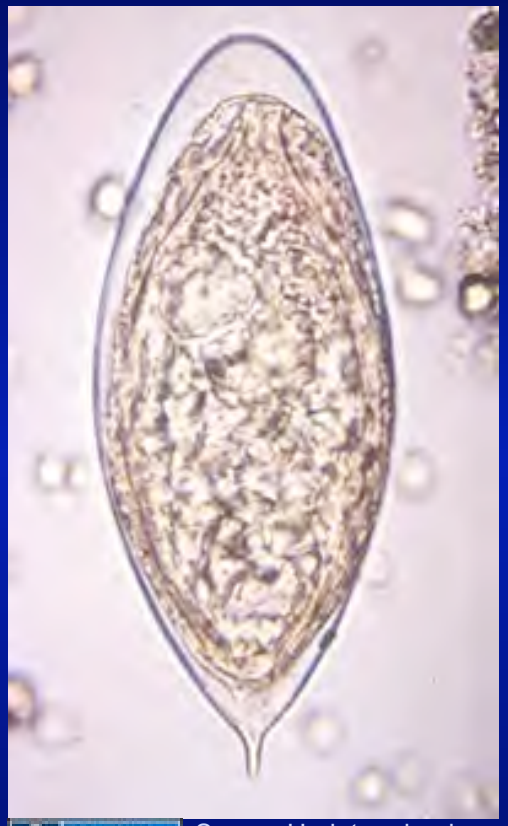
S. mansoni



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S. haematobium

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S. japonicum

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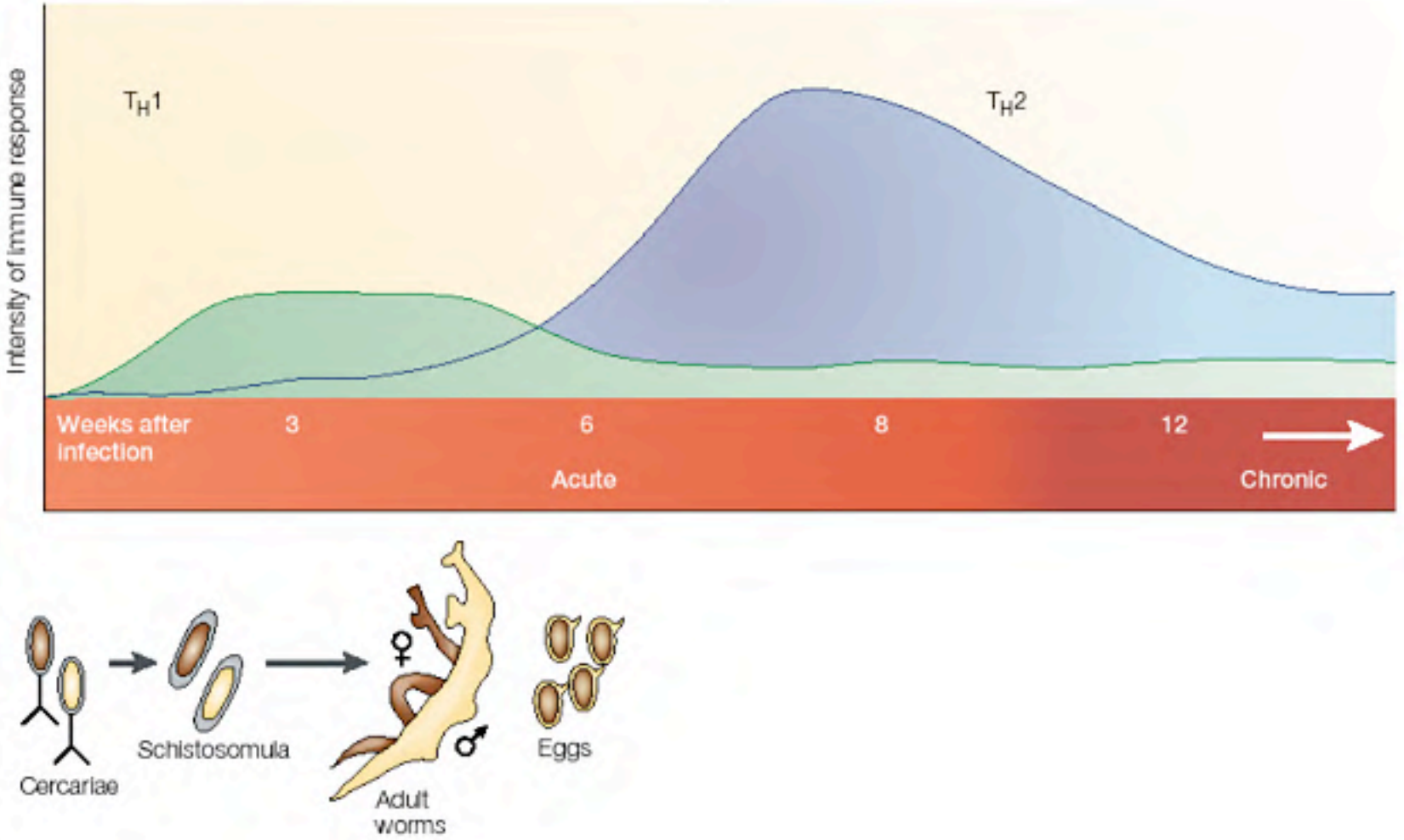
Events following cercarial penetration

1. Larva migrate to lungs and develop as "schistosomulae" (this may trigger a self-limited febrile illness).
2. Male and female schistomulae migrate to the abdominal venules:
   I. Superior mesenteric (*S. japonicum*)
   II. Inferior mesenteric (*S. mansoni*)
   III. Bladder plexus (*S. hematobium*)
3. Males and females pair off and egg production begins
4. Eggs migrate out of the body through visceral organs or become trapped and die in tissues.

Immune response to schistosoma infection



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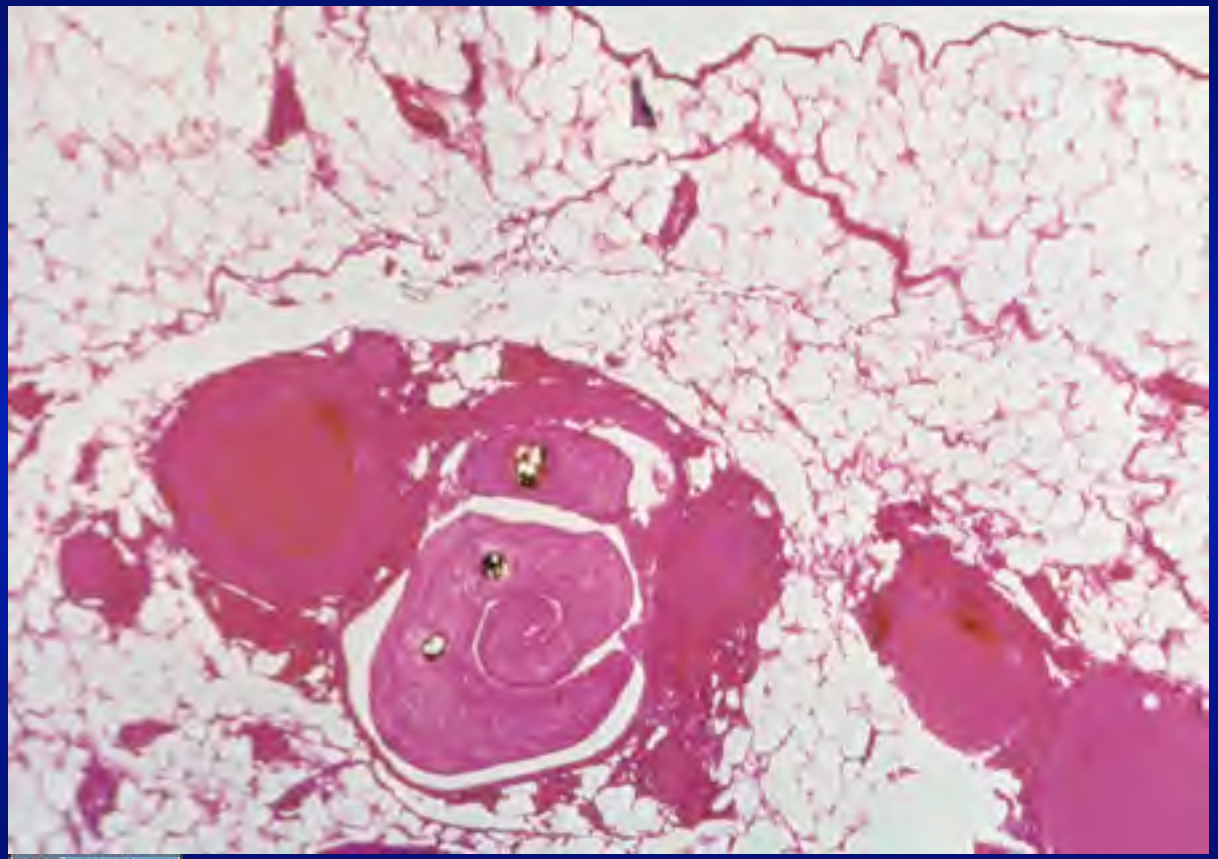

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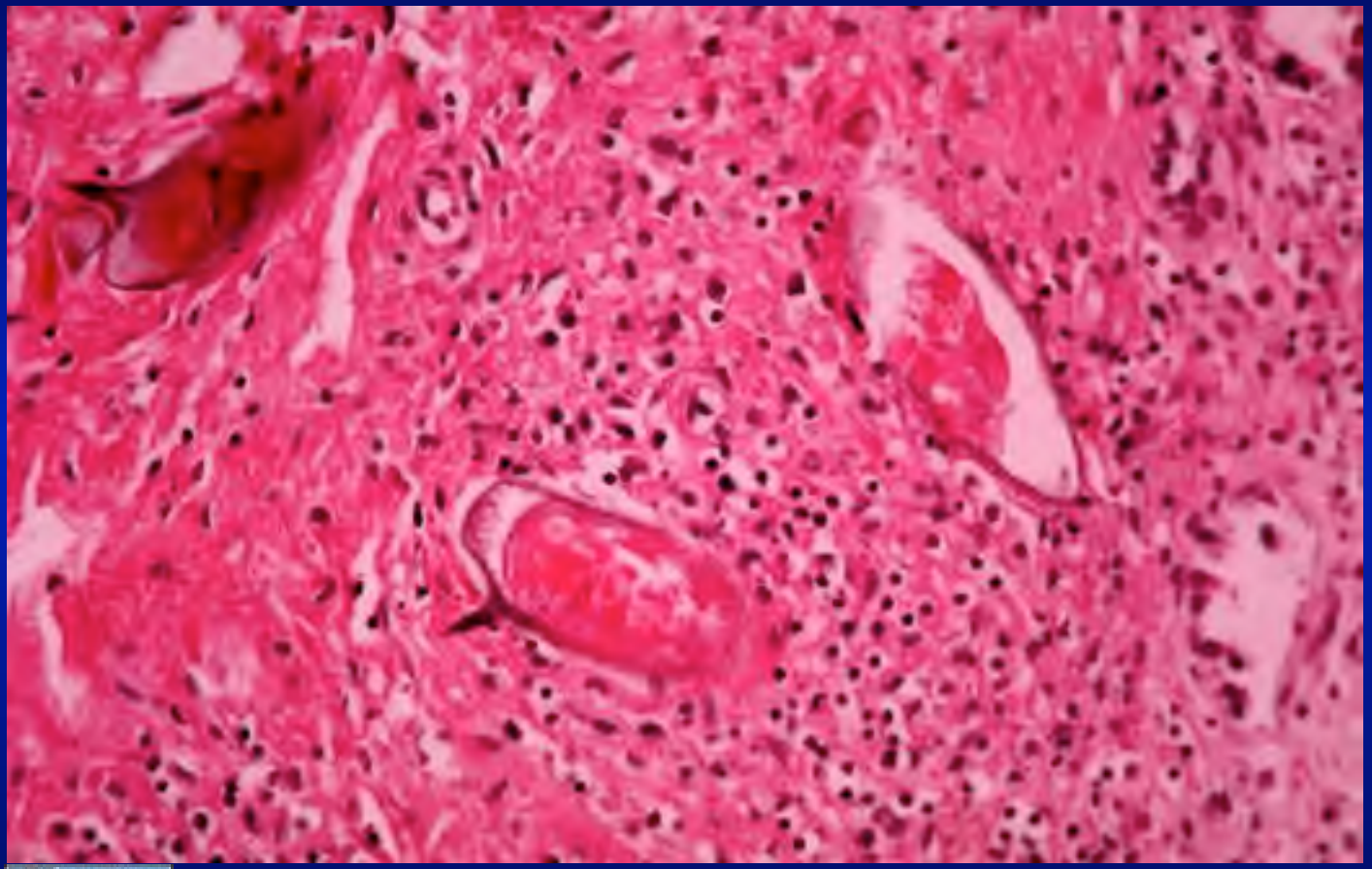

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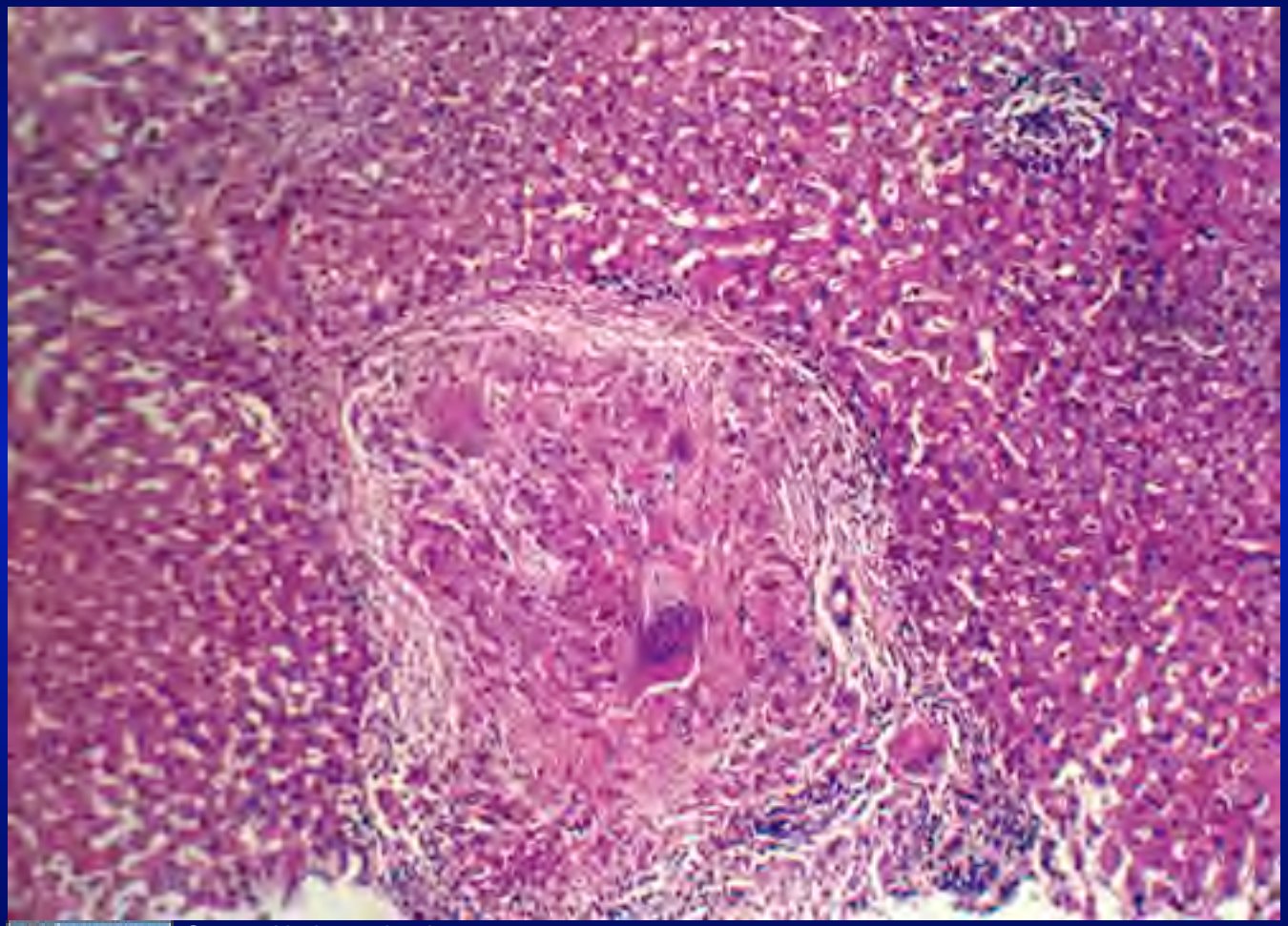

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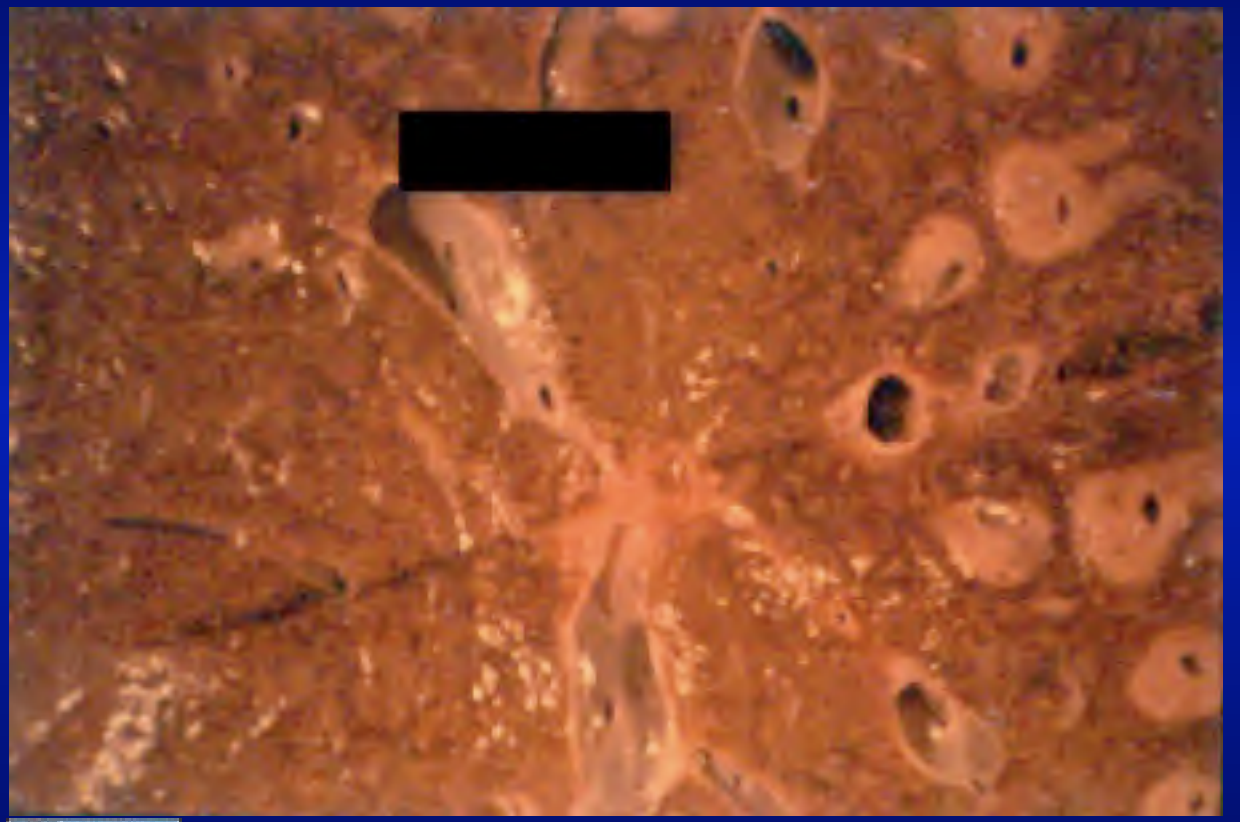

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“pipestem” fibrosis

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Schistosomiasis - pathogenesis

- egg granuloma (type IV reaction)--> fibrosis
- morbidity ~ worm (egg) burden
- concomitant immunity to schistosomula
- adult worms: invisible to the immune system (survive for years)

Schistosomiasis- clinical features

- Cercarial dermatitis
- Intestinal schistosomiasis (granulomas --> polyps, protein loss, malabsorption, strictures)
- Hepatosplenic schistosomiasis (portal hypertension --> ascites, varices, splenomegaly, normal hepatic function)
- Urinary schistosomiasis (hematuria, chronic infection, obstruction)
- Other (cardiopulmonary, CNS, etc.)

Drug treatment of schistosomiasis

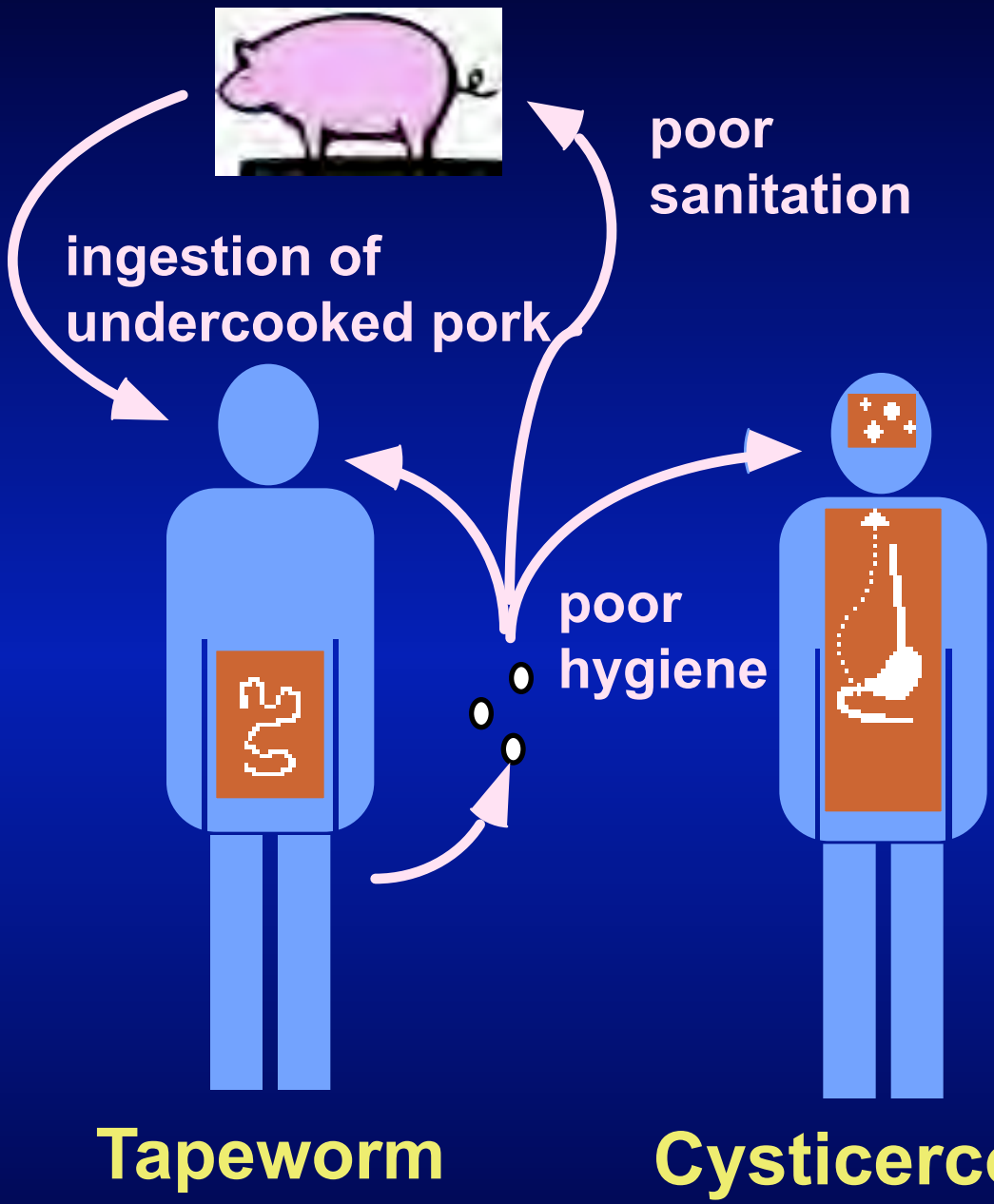
- Praziquantel increases permeability of adult parasite to Ca^{++}.
- Tetanospasm --> death

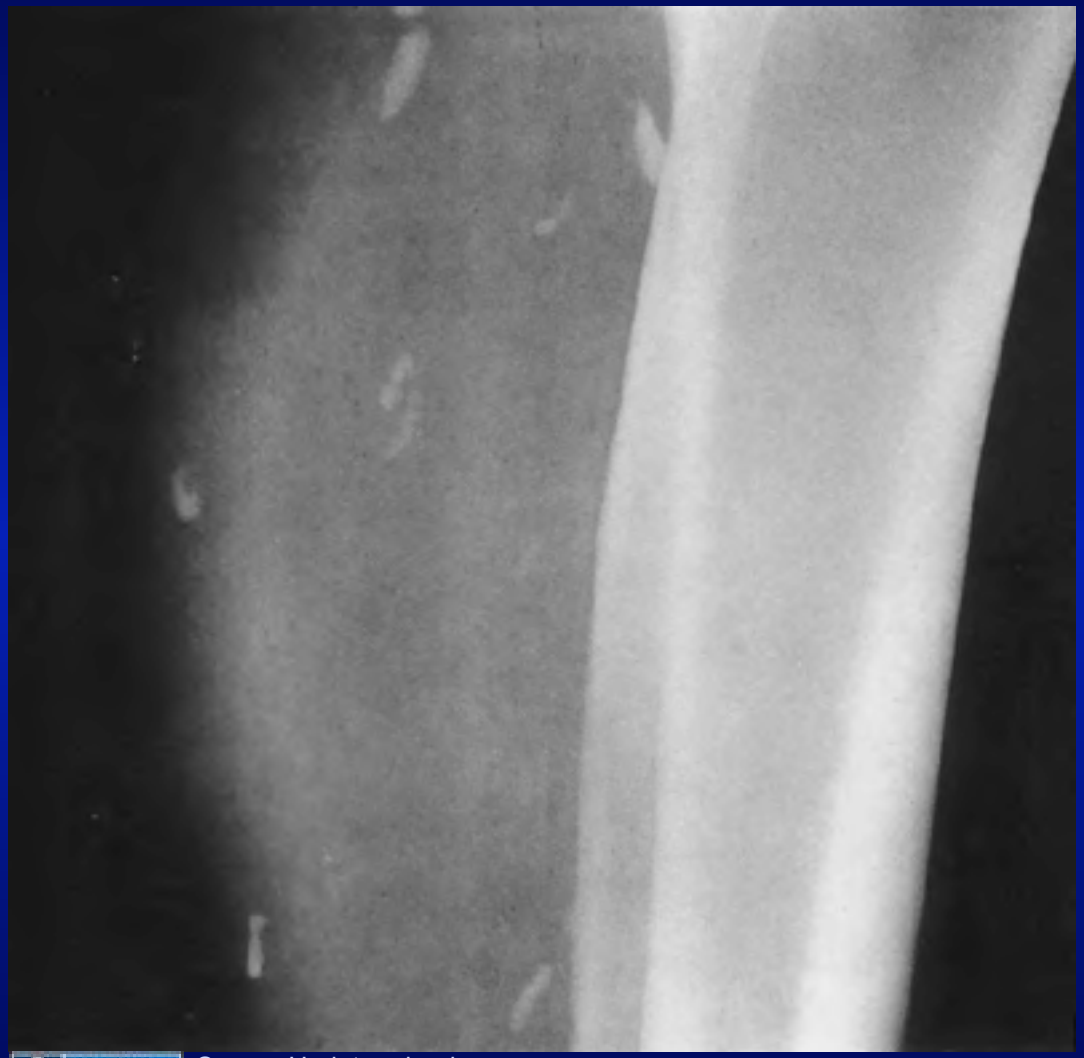
Cestode infections

Tapeworms

- Definitive hosts: harbor adult worms
- Intermediate hosts: harbor tissue cysts (containing worm heads)
- Humans acquire infection two ways:
  - —ingestion of eggs from feces (to acquire tissue cysts) = Intermediate host
  - —ingestion of tissue cysts in undercooked meat (to acquire a tapeworm) = Definitive host

Taeniasis





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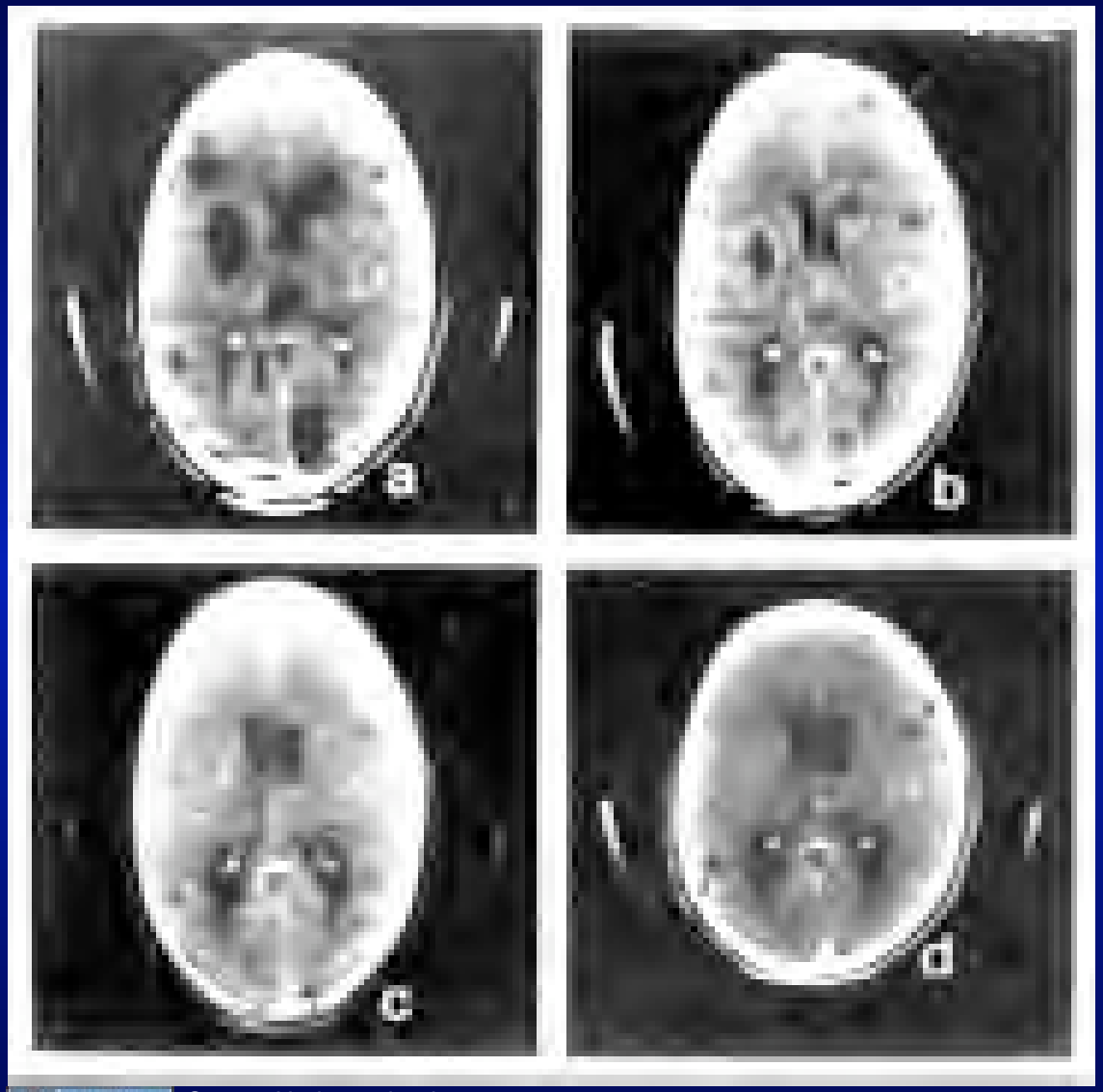

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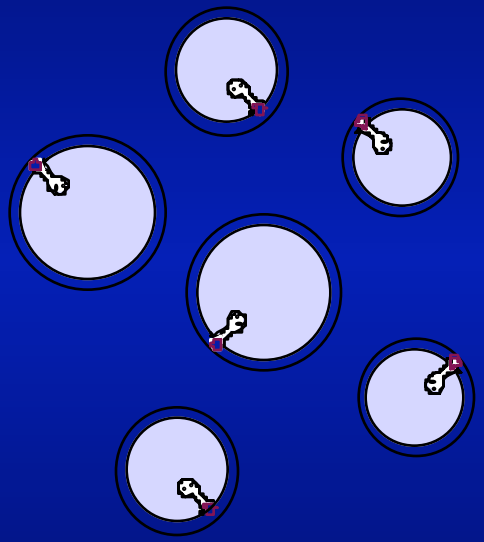
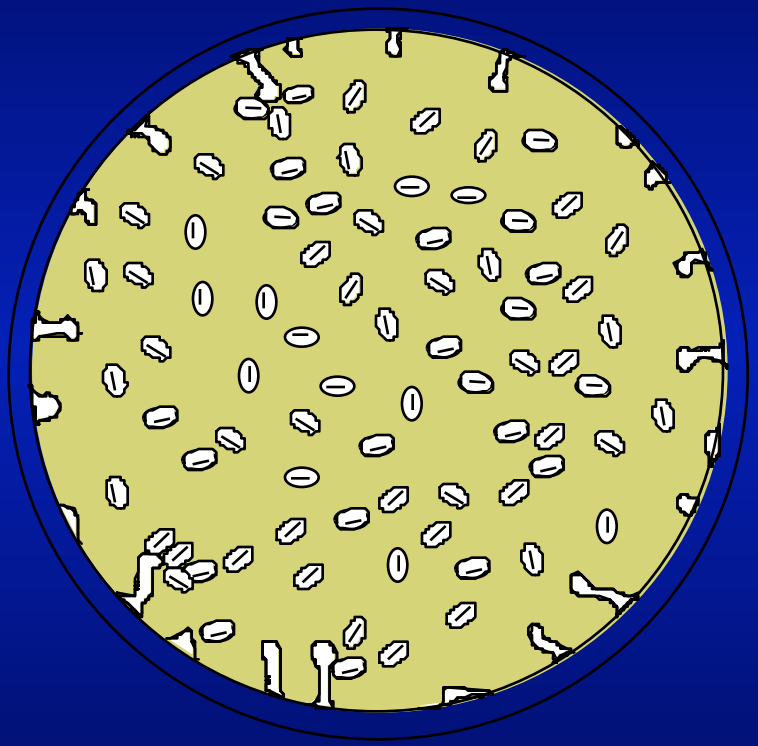
Cysticerci



Hydatid Cyst



Isolated cysticerci

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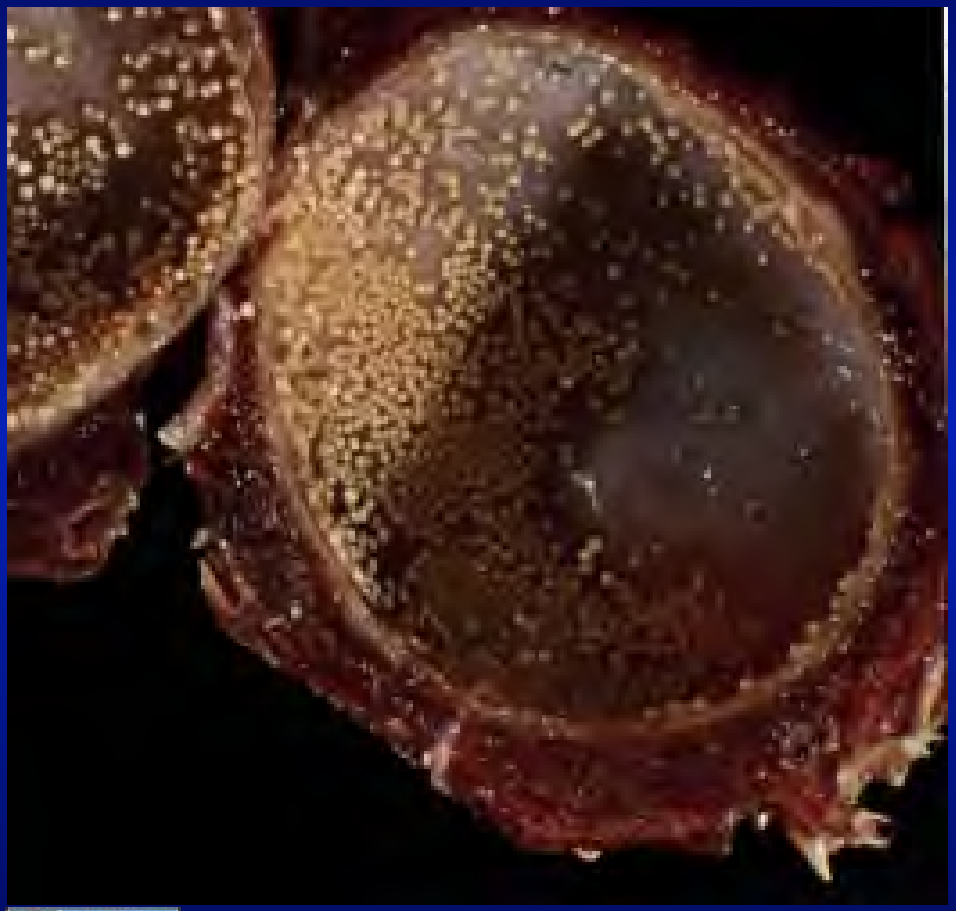
Hydatid cyst



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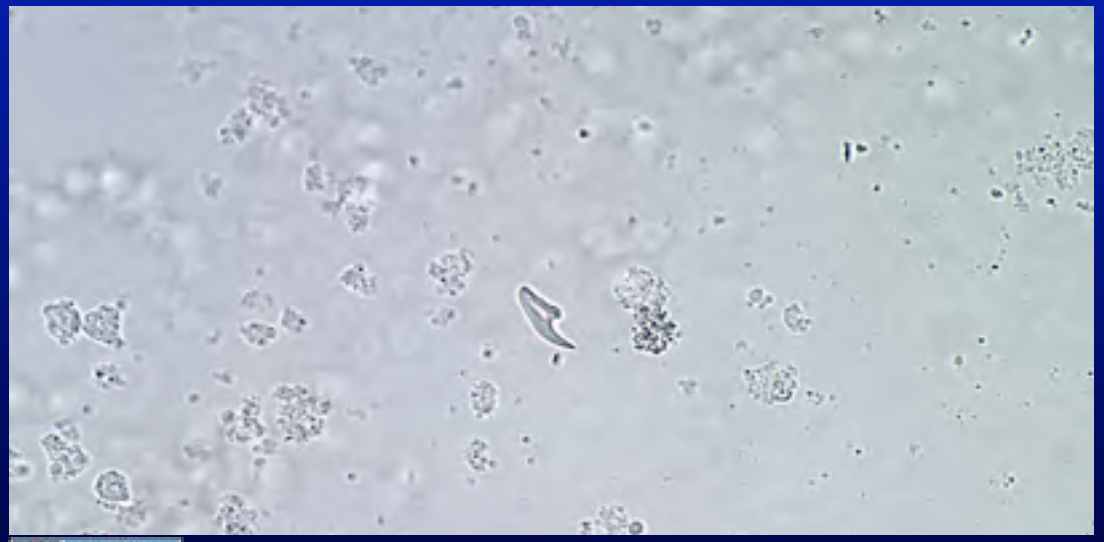
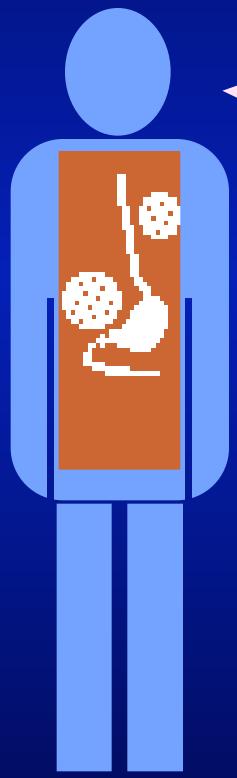


PD-INEL Duane Newton

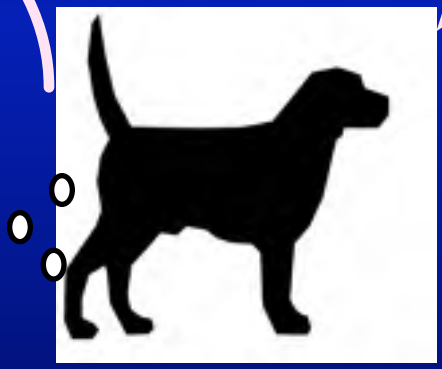
Echinococcosis



ingestion of eggs in pastures

ingestion of entrails

contact with dogs



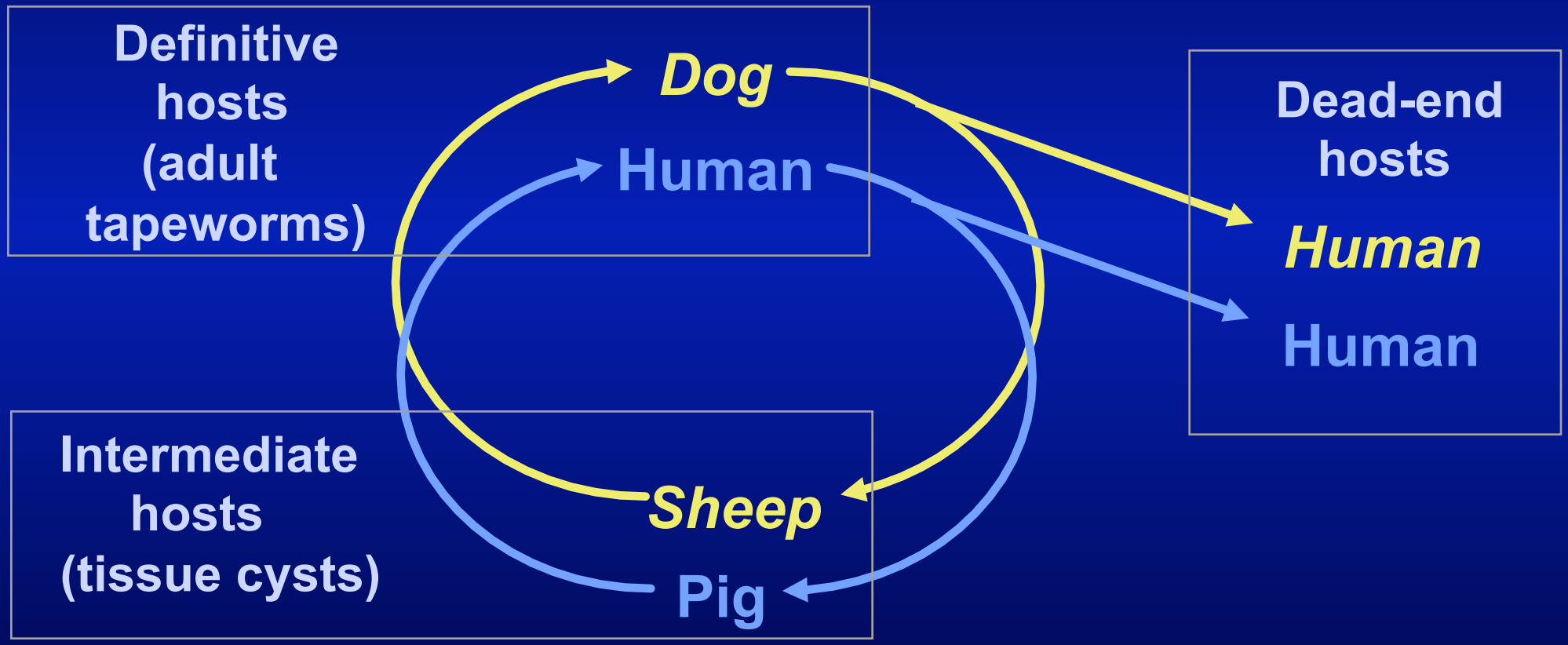
Cystic Hydatid Disease

PD-INEL Cary Engleberg

Treatment of cysticercosis and echinococcosis

- Antihelminthic therapy (e.g., albendazole, praziquantel)
- (Echinococcus only)
  - —Surgical removal
  - —Irrigation-evacuation of cysts

Comparison of pork tapeworm and *Echinococcus* life cycles



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