Parasitology (4th stage)/ 2023-2024
Helminthes/ Trematodes
Blood Trematodes
Schistosoma

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Trematodes (Flukes)

- The most significant trematodes from a clinical point of view are blood flukes, *Schistosoma mansoni*, *S. japonicum* and *S. hematobium*. Other trematodes of significance are intestinal fluke, *Fasciolopsis buski*, liver fluke, *Clonorchis sinensis* and lung fluke, *Paragonimus westermani*.

SCHISTOSOMIASIS (BILHARZIASIS)

- Three species of Schistosoma have different geographic distributions. ***S. hematobium*** is prevalent in Africa, ***S. mansoni*** is found in Africa and America and ***S. japonicum*** is common in the far east.
- **Epidemiology**
- Approximately 250 million people are infected with schistosomes and 600 million are at risk.

Morphology

- Adult worms are 10 to 20 mm long; the male has an unusual lamelliform shape with marginal folds forming a canal in which the slender female worm resides. Unlike other trematodes, schistosomes have separate sexes (figure 1).

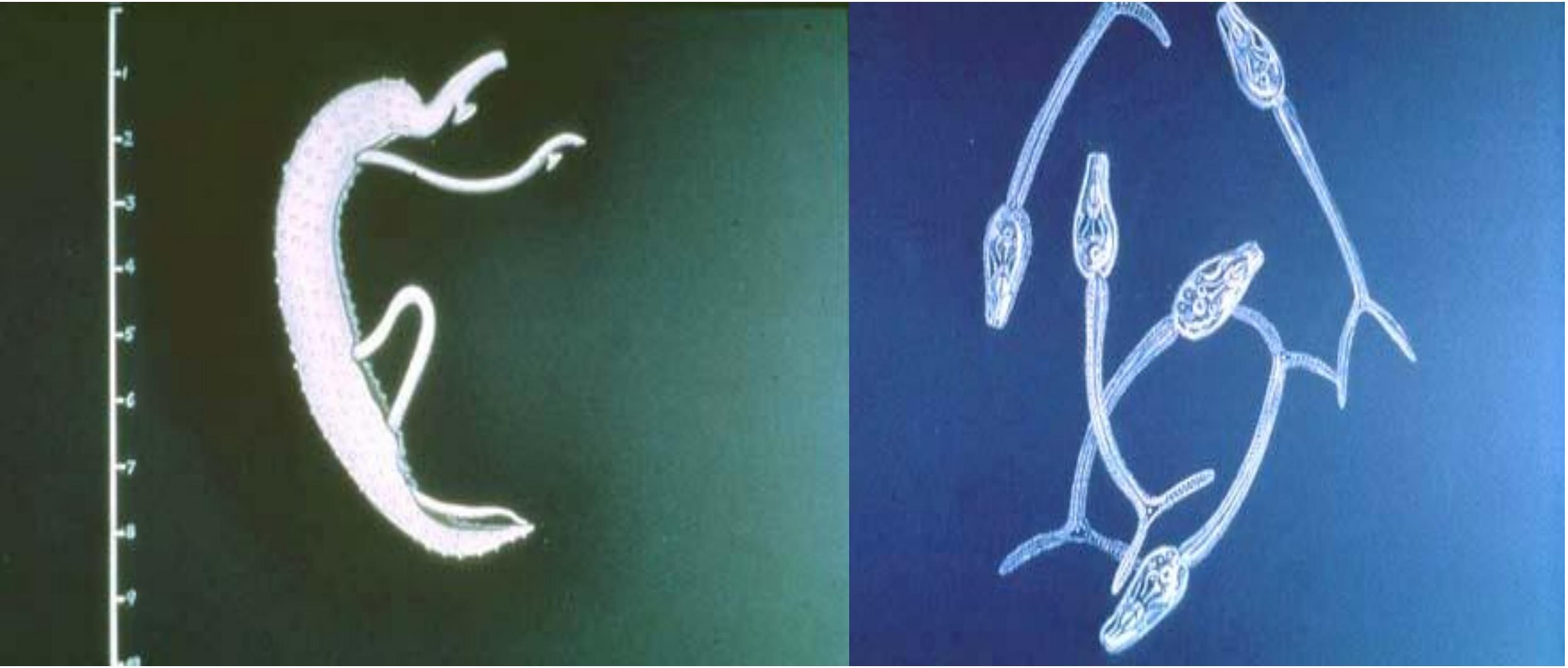


Fig 1 A Schistosomes. WHO

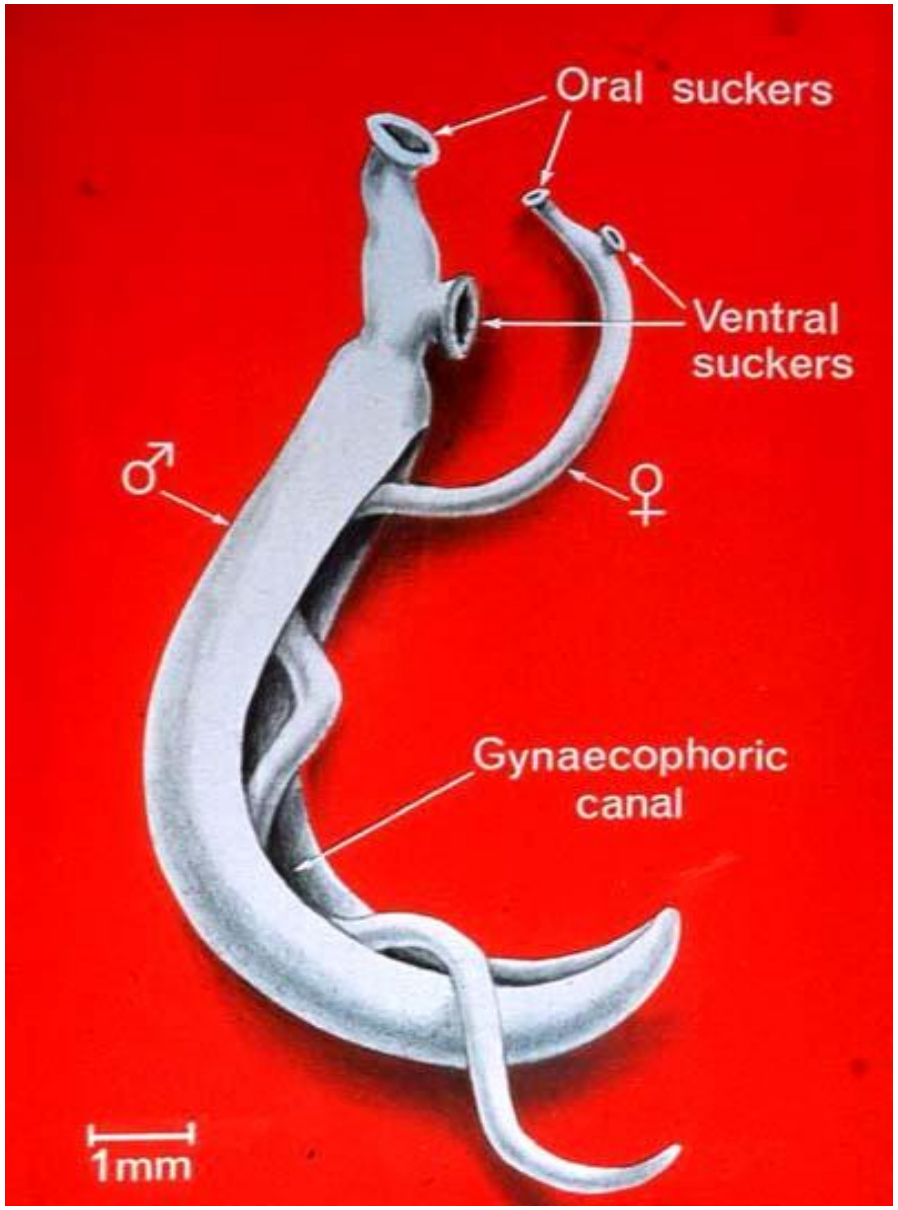


Figure 1B Male and Female SchistosomesWHO

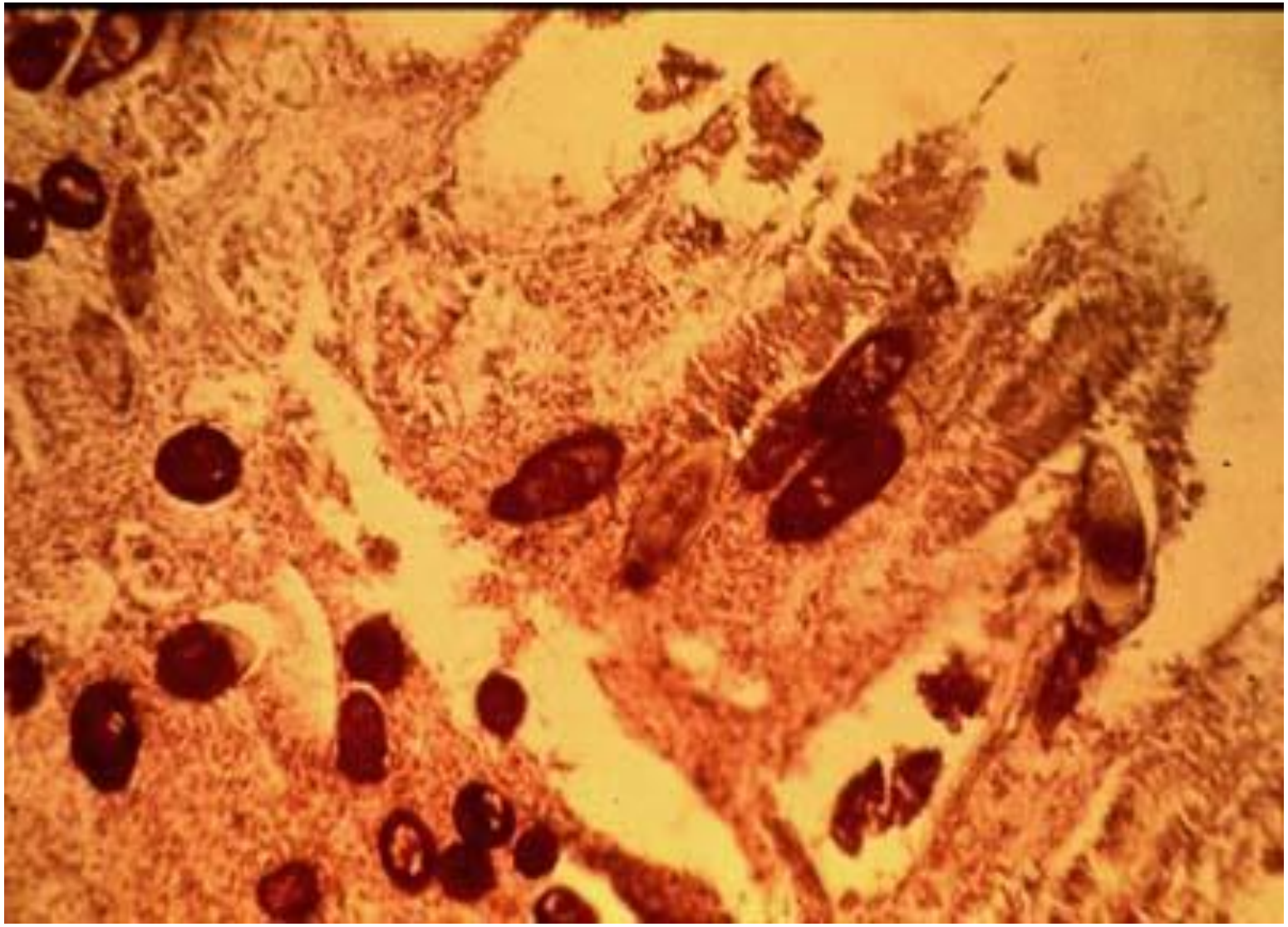


Figure 1C Intestinal schistosomiasis: eggs in the wall of the gut. WHO

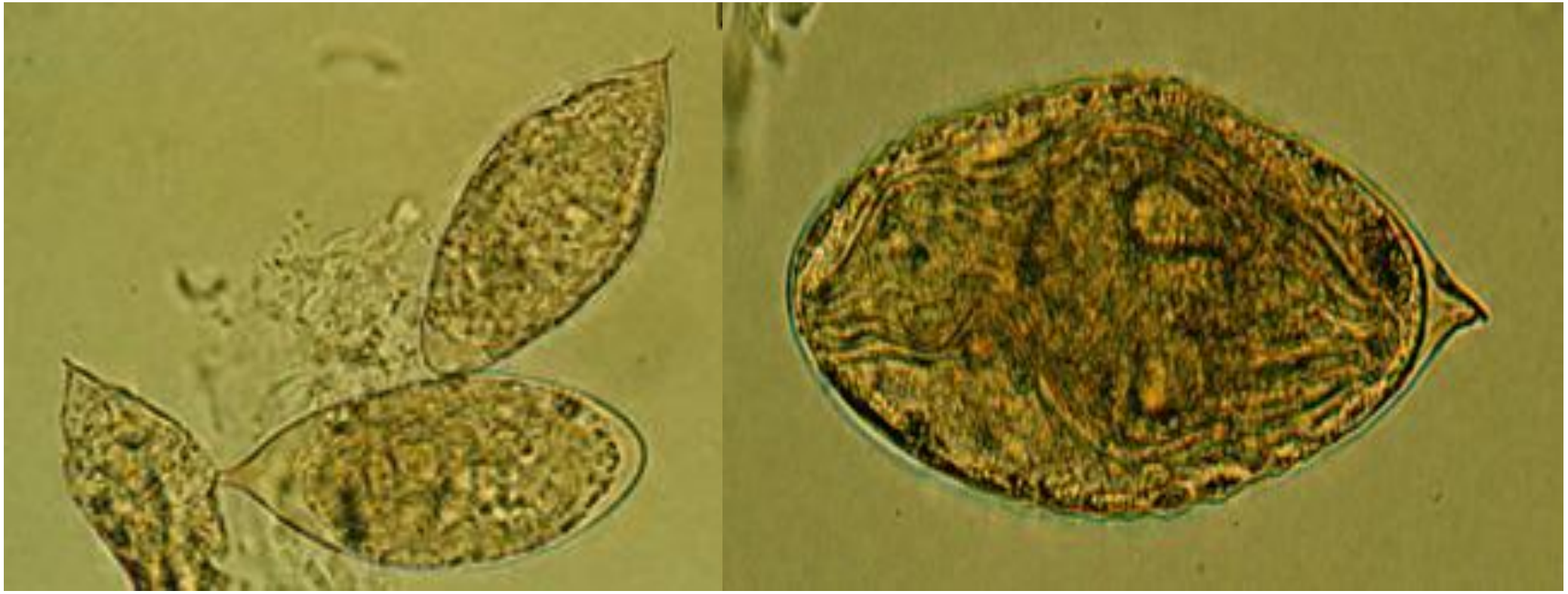


Figure 1D

Eggs of *Schistosoma haematobium* (A). In this species, the eggs are large and have a prominent terminal spine at the posterior end. Length 112-170 µm. In (B), a greater magnification shows the miracidium inside the egg.

Figure 1E *Schistosoma haematobium* adult male

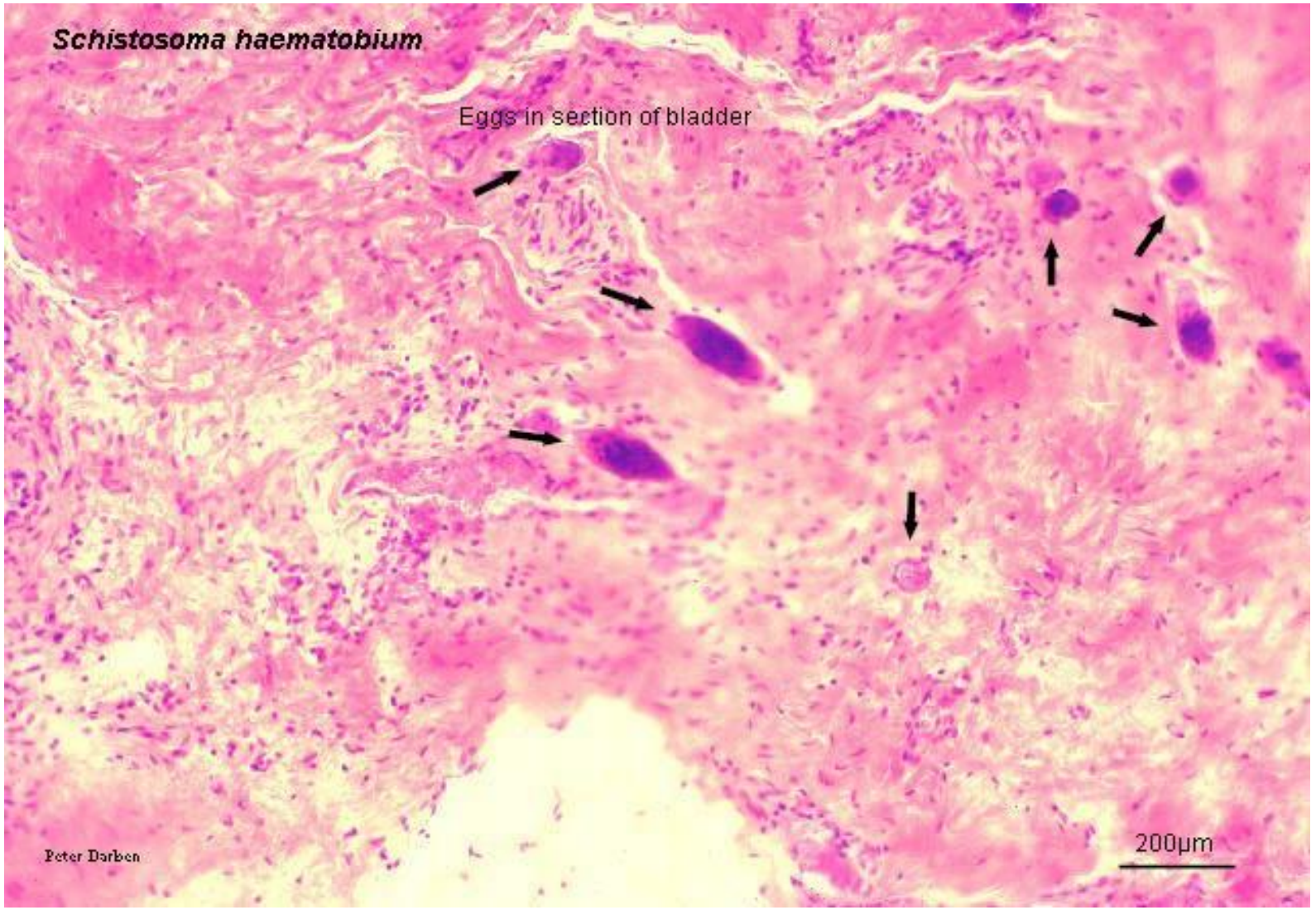


Figure 1F *Schistosoma haematobium* eggs in section of bladder (H&E)

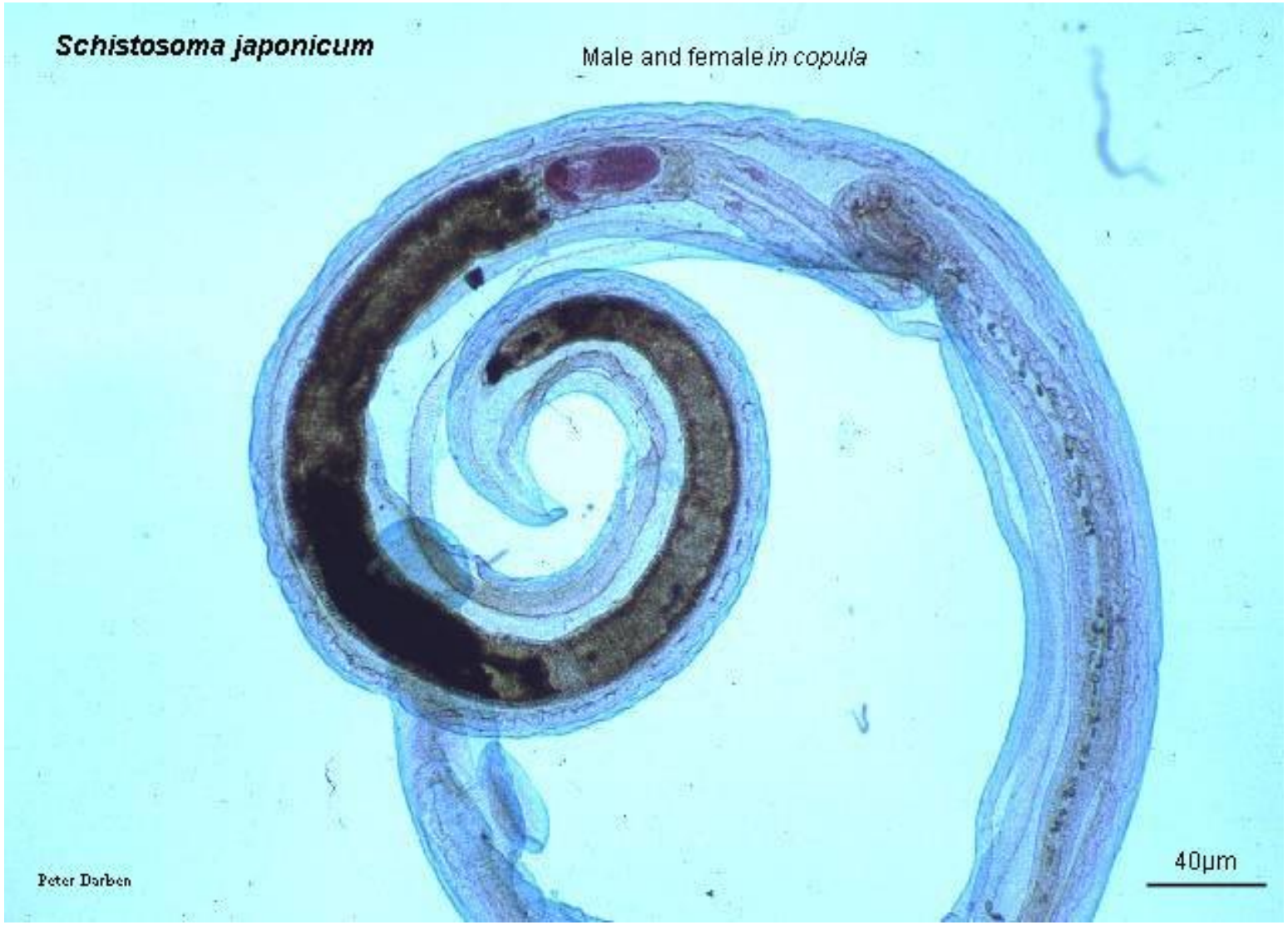


Figure 1G *Schistosoma japonicum* adult male and female, in copula

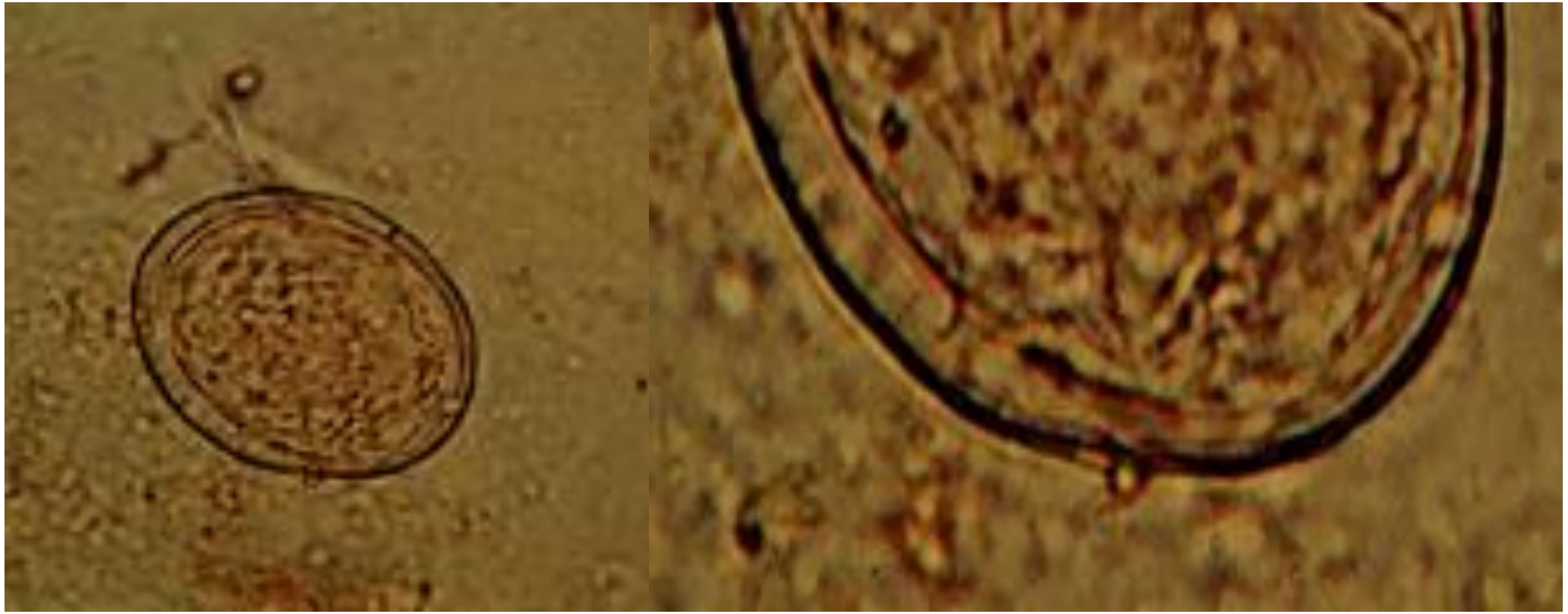


Figure HA **Figure HB**

Egg of *Schistosoma japonicum* (A). The egg is typically oval or subspherical, and has a vestigial spine, which is better shown in (B). *Schistosoma japonicum* eggs are smaller (68 - 100 µm by 45 - 80 µm) than those of the other species

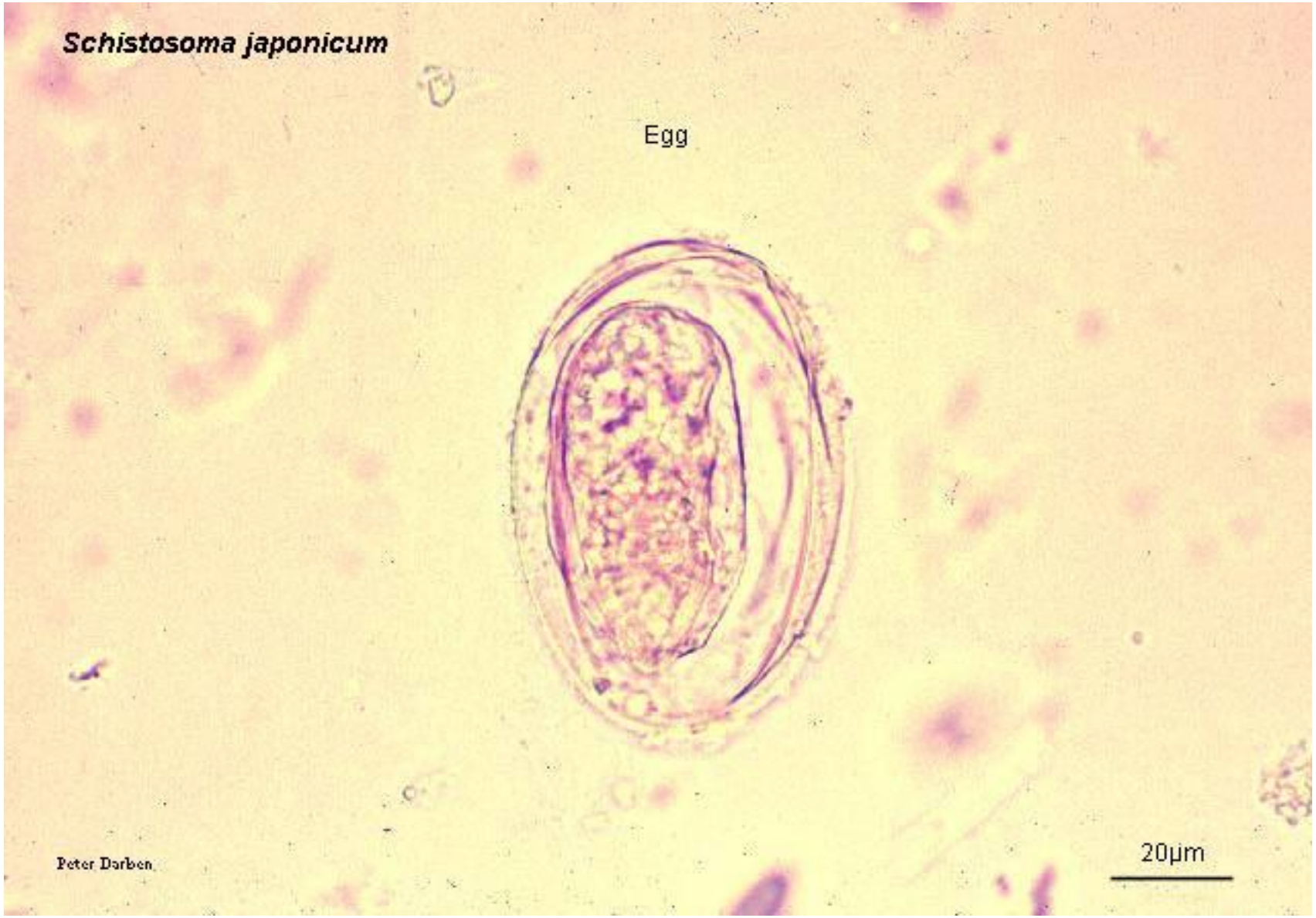


Figure 1I *Schistosoma japonicum* egg

Figure 1 J *Schistosoma japonicum* adult male and female

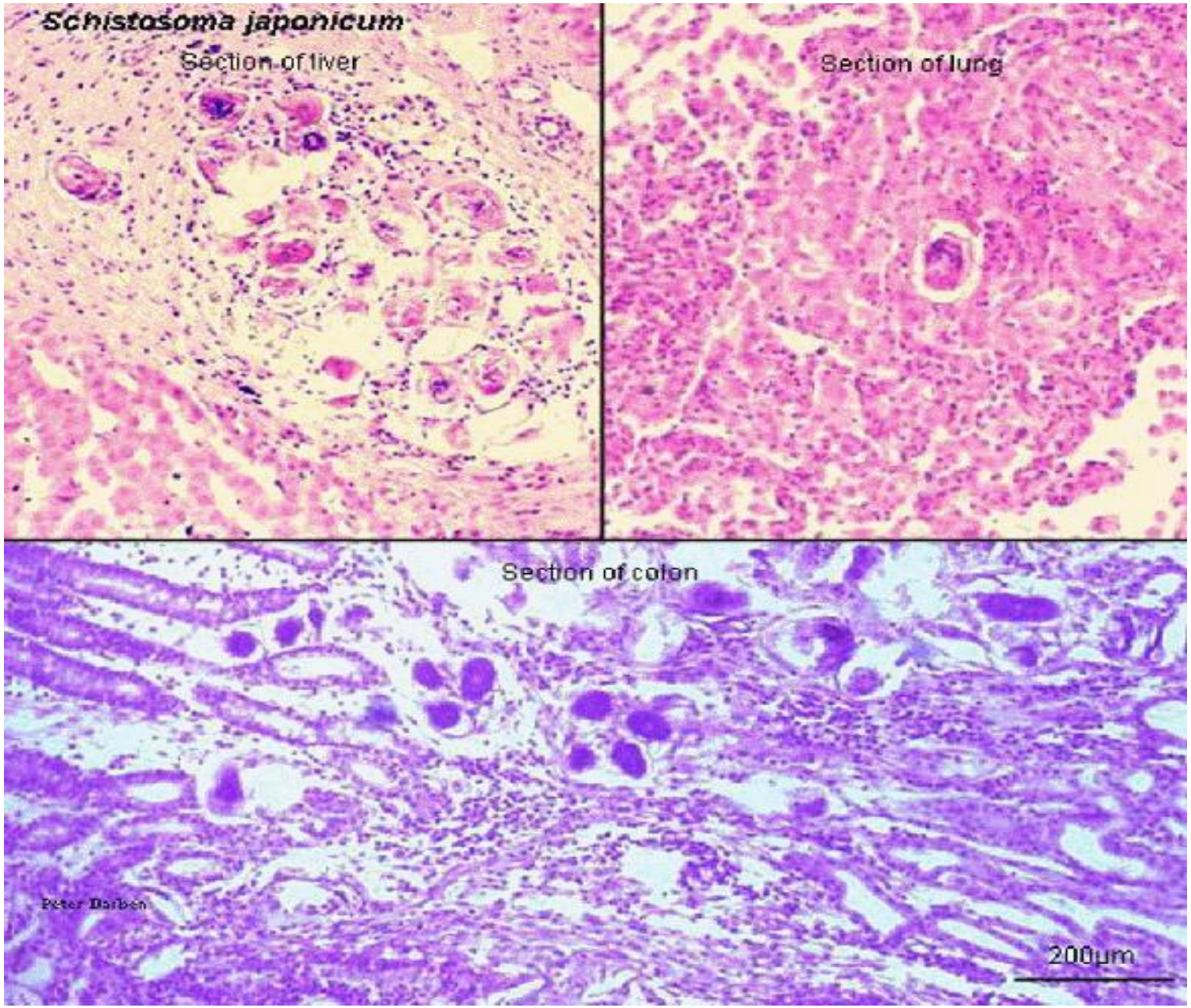


Figure 1K *Schistosoma japonicum* eggs in tissue section (H&E)

Figure 1L A **Figure 1L B**

Eggs of *Schistosoma mansoni* in a patient from Egypt. These eggs are large (length 114 - 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 (visible especially in A). CDC

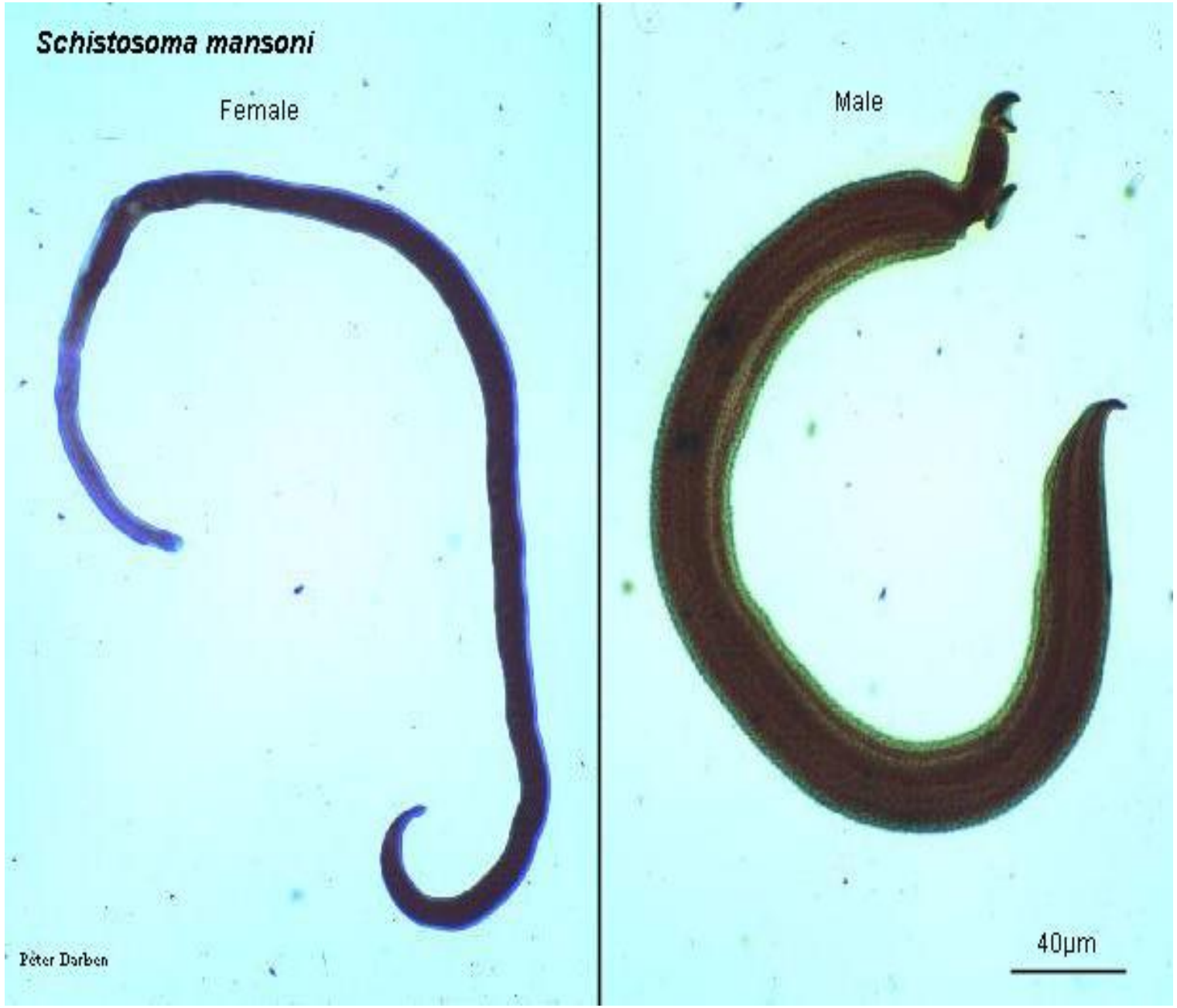


Figure 1M *Schistosoma mansoni* adult male and female

Figure 1N Schistosoma mansoni adult male and female, in copula

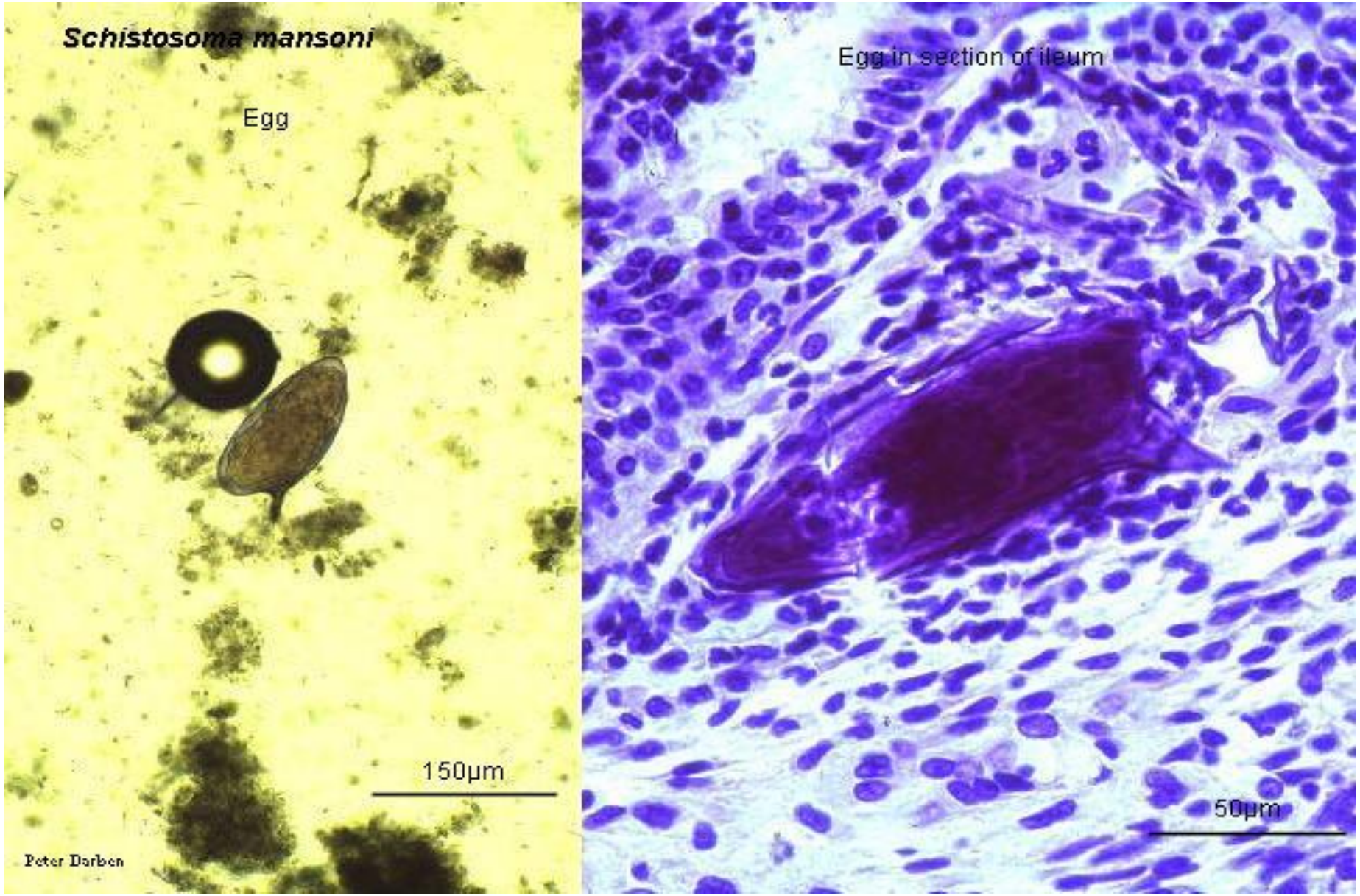


Figure 1O *Schistosoma mansoni* egg, whole and in section (H&E)

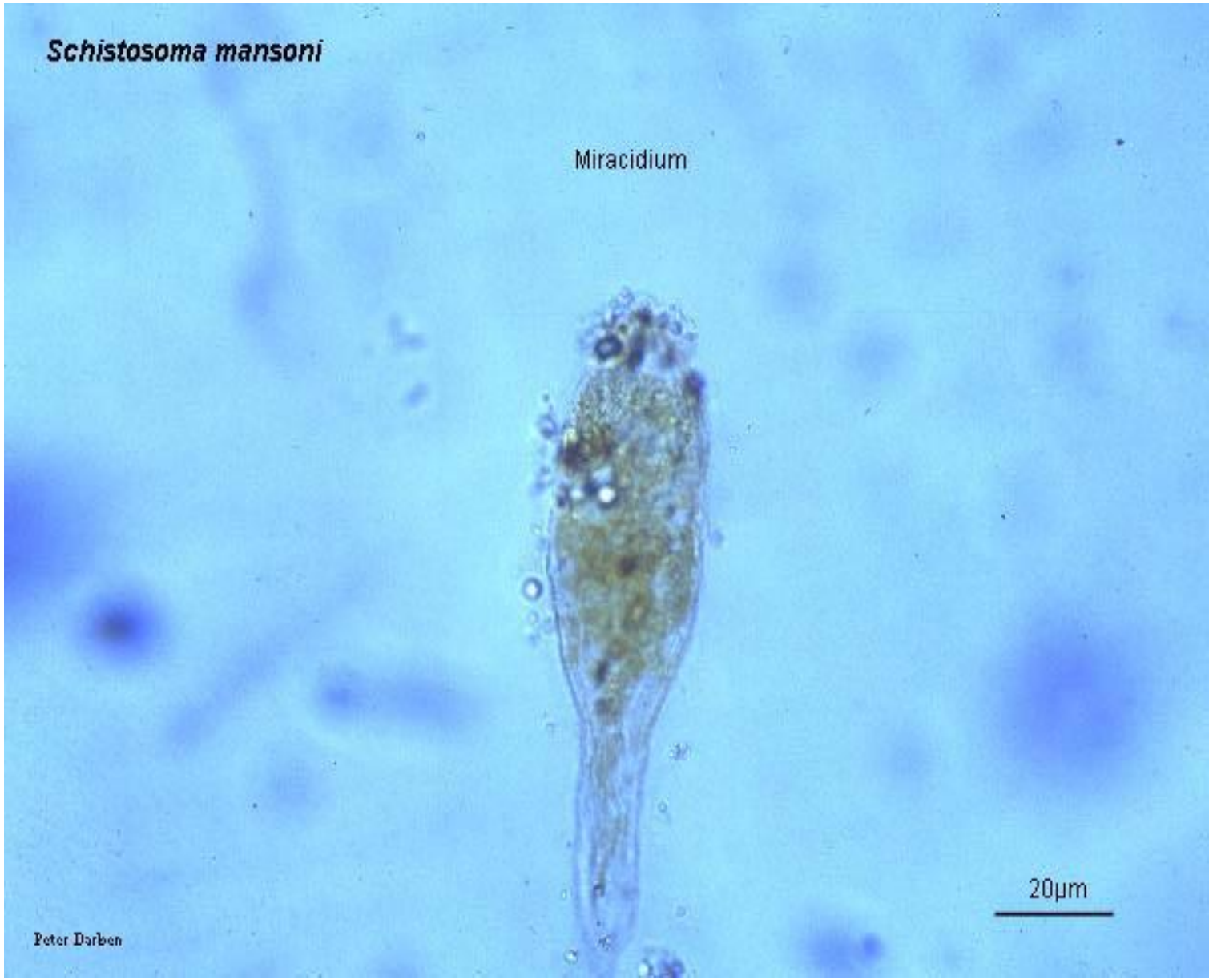


Figure 1P *Schistosoma mansoni* miracidium

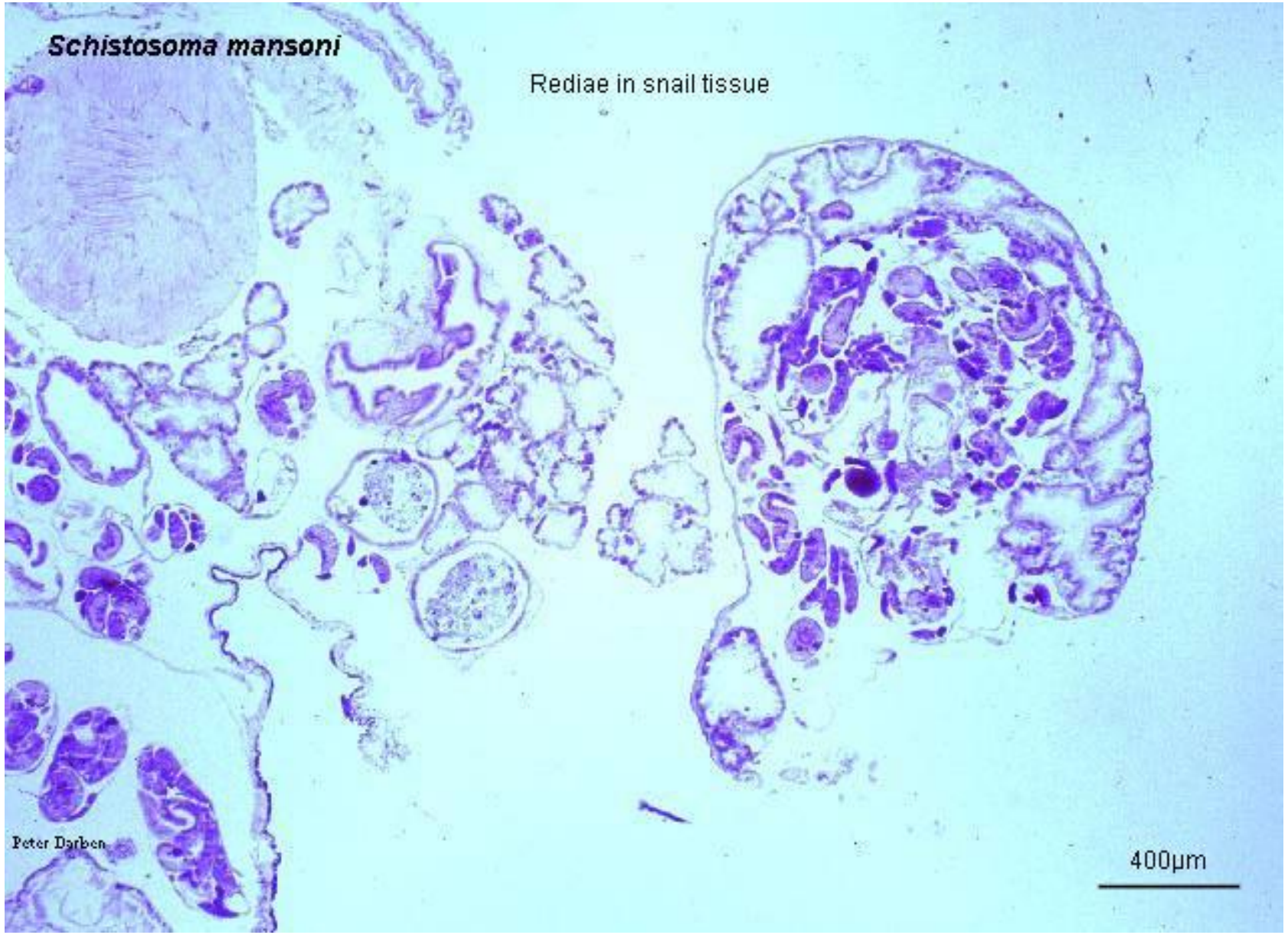


Figure 1Q *Schistosoma mansoni* in section of snail tissue (H&E)

Figure 1R *Schistosoma mansoni* cercaria

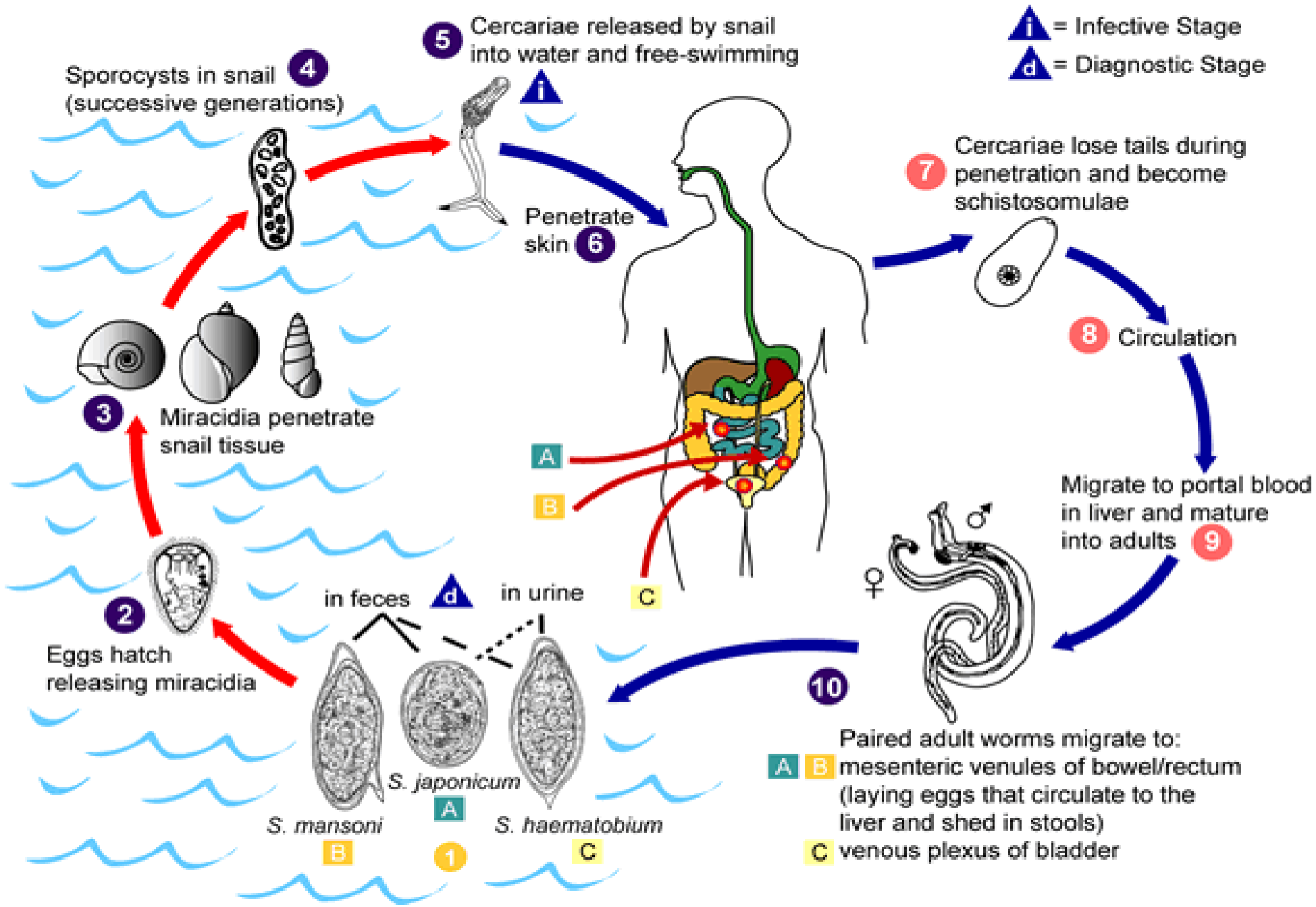

i = Infective Stage
d = Diagnostic Stage
1
Sporocysts in snail
(successive generations)
4
5
Cercariae released by snail
into water and free-swimming
i
Penetrate
skin
6
7
Cercariae lose tails during
penetration and become
schistosomulae
8
Circulation
Migrate to portal blood
in liver and mature
into adults
9
10
Paired adult worms migrate to:
A B mesenteric venules of bowel/rectum
(laying eggs that circulate to the
liver and shed in stools)
C venous plexus of bladder
3
Miracidia penetrate
snail tissue
2
Eggs hatch
releasing miracidia
in feces
d
in urine
S. japonicum
A
S. mansoni
B
S. haematobium
C
A
B
C

Figure 1S

Eggs are eliminated with feces or urine **1**. Under optimal conditions the eggs hatch and release miracidia **2**, which swim and penetrate specific snail intermediate hosts **3**. The stages in the snail include 2 generations of sporocysts **4** and the production of cercariae **5**. Upon release from the snail, the infective cercariae swim, penetrate the skin of the human host **6,** and shed their forked tail, becoming schistosomulae **7**. The schistosomulae migrate through several tissues and stages to their residence in the veins **(8,9)**. Adult worms in humans reside in the mesenteric venules in various locations, which at times seem to be specific for each species **10**.

For instance, *S. japonicum* is more frequently found in the superior mesenteric veins draining the small intestine **A**, and *S. mansoni* occurs more often in the superior mesenteric veins draining the large intestine **B**. However, both species can occupy either location, and they are capable of moving between sites, so it is not possible to state unequivocally that one species only occurs in one location. *S. haematobium* most often occurs in the venous plexus of bladder **C,** but it can also be found in the rectal venules. The females (size 7 to 20 mm; males slightly smaller) deposit eggs in the small venules of the portal and perivesical systems.

The eggs are moved progressively toward the lumen of the intestine (*S. mansoni* and *S. japonicum*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively **1**.

Pathology of *S. mansoni* and *S. japonicum* schistosomiasis includes: Katayama fever, presinusoidal egg granulomas, Symmers' pipe stem periportal fibrosis, portal hypertension, and occasional embolic egg granulomas in brain or spinal cord. Pathology of *S. haematobium* schistosomiasis includes: hematuria, scarring, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord.

Human contact with water is thus necessary for infection by schistosomes. Various animals, such as dogs, cats, rodents, pigs, hourse and goats, serve as reservoirs for *S. japonicum*, and dogs for *S. mekongi*.

Life cycle

Man is infected by cercaria in fresh water by skin penetration. The cercaria travel through the venous circulation to the heart, lungs and portal circulation. In about 3 weeks, they mature and reach the mesenteric (*S. japonicum* and *S. mansoni*) or the bladder (*S. hematobium*) vessels where they live and ovulate for the duration of the host's life.

Eggs germinate as they pass through the vessel wall into the intestine or bladder and are excreted in feces (*S. japonicum* and *S. mansoni*) or urine (*S. hematobium*). In fresh water, the larval miracidium hatches out of the egg and swims about until it finds an appropriate snail. After two generations of multiplication in the snail, the fork-tailed cercariae emerge into the water and infect another human **(figure 2).**

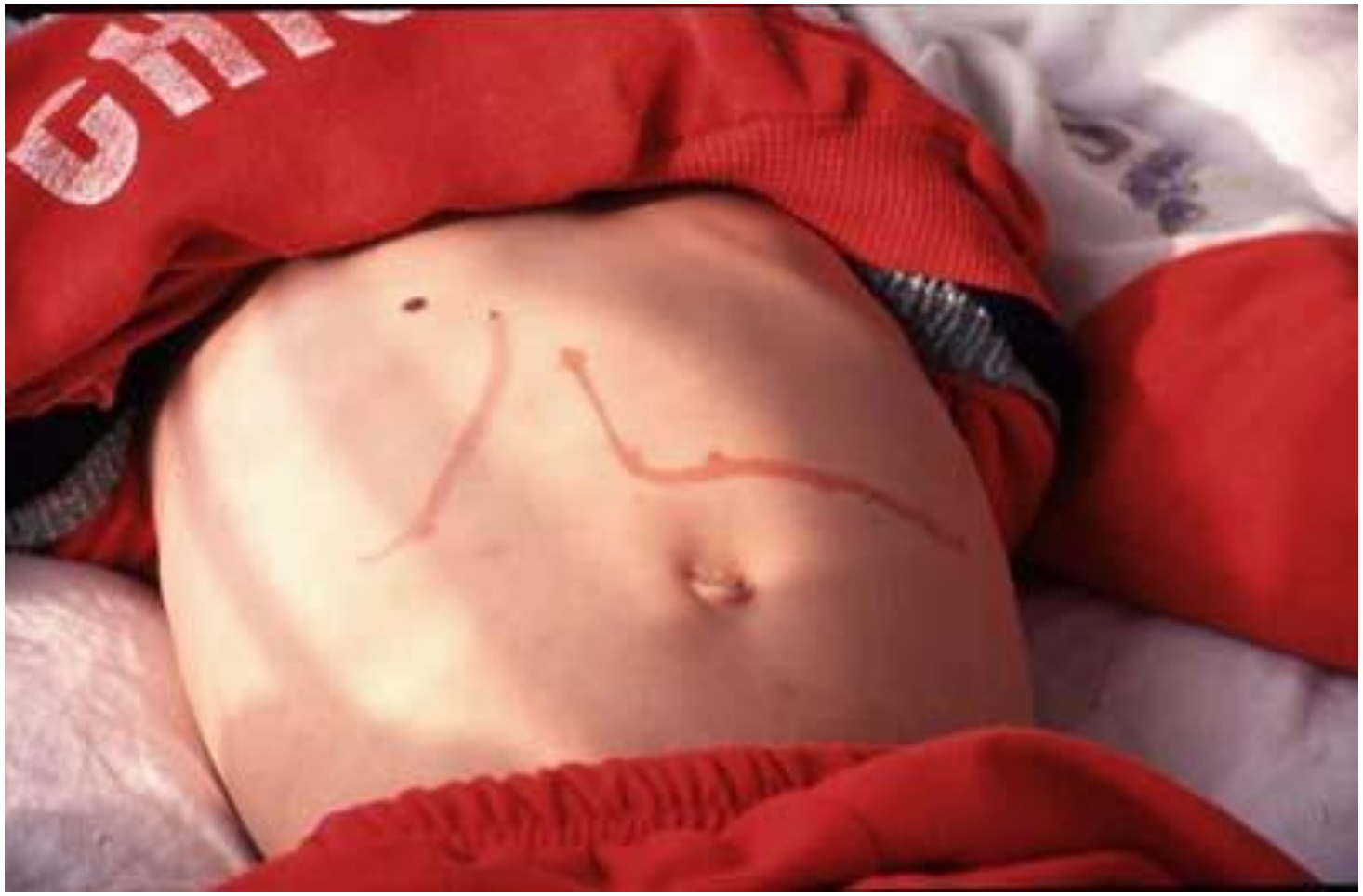


Figure 2A The abdomen of an 11-year-old boy with intestinal schistosomiasis with the size and extent of the liver and spleen marked. Both are well below the midline, indicating the severity of infection. The disease has caused a stunting of the boy's growth, he is only 120cms tall and weighs 22 kg. WHO/TDR/Crump

Figure 2B

Two boys, victims of schistosomiasis showing typical distension of the abdomen. WHO

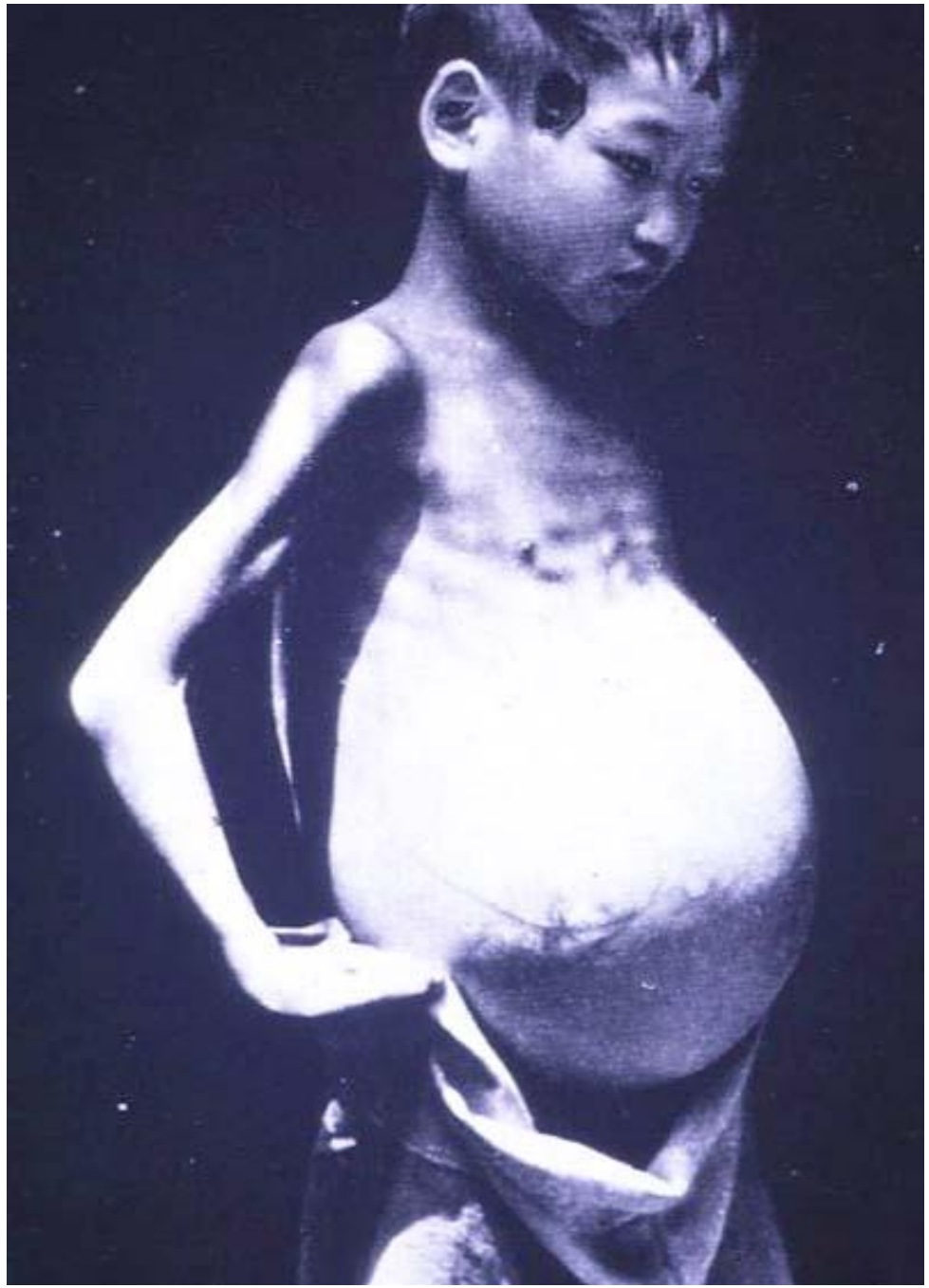


Figure 2C

Symptoms

Penetration of cercariae causes transient dermatitis (swimmers' itch). The symptoms of schistosomiasis are primarily due to a reaction against the eggs and include splenomegaly, lymphadenopathy and diarrhea.

In the bladder, they produce granulomatous lesions, hematuria and sometimes urethral occlusion. Most bladder cancers in endemic areas are associated with chronic infection. In the intestine, they cause polyp formation which, in severe cases, may result in life threatening dysentery. In the liver, the eggs cause periportal fibrosis and portal hypertension resulting in hepatomegaly, splenomegaly and ascites.

A gross enlargement of the esophageal and gastric veins may result in their rupture. *S. japonicum* eggs are sometimes carried to the central nervous system and cause headache, disorientation, amnesia and coma. Eggs carried to the heart produce arteriolitis and fibrosis resulting in enlargement and failure of the right ventricle **(figure 2a).**

Pathology and Immunology

The 'swimmers' itch is due to physical damage to the skin by proteases and other toxic substances secreted by the cercaria. The host develops both type I and type IV hypersensitivity reactions to schistomal secretions and egg constituents.

Embryonated eggs cause collagenase-mediated damage to the vascular endothelium. Host immune responses, both humoral and cell mediated, have been shown to be of some protective value. IgE and eosinophil mediated cytotoxicity has been suggested as a mechanism of killing the adult worm.

Diagnosis

Diagnosis is based on a history of residence in an endemic area, swimmers' itch and other symptoms. The eggs are very characteristic and confirm diagnosis. *S. hematobium* eggs in urine (55 to 65 by 110 to 170 micrometers) have an apical spine or knob. *S. mansoni* eggs in feces (45 to 70 by 115-175 micrometers) have a spine on the side. *S. japonicum* eggs (55 to 65 by 70 to 100 micrometers) are more round with a vague spine on the side.

Treatment and control

Praziquantel is effective against all species. Contaminated water should be avoided. Control measures include sanitary disposal of sewage and destruction of snails. No vaccine is available.

Thank you for listening

Parasitology
Helminthes/NEMATODES
(Round Worms)

INTESTINAL HELMINTHS

Intestinal nematodes of importance to man are:

- ***Ascaris lumbricoides* (roundworm)**
- ***Trichinella spiralis* (trichinosis)**
- ***Trichuris trichiura* (whipworm)**
- ***Enterobius vermicularis* (pinworm)**
- ***Strongyloides stercoralis* (Cochin-china diarrhea)**
- ***Ancylostoma duodenale* and *Necator americanes* (hookworms)**
- ***Dracunculus medinensis* .**

E. vermicularis* and *T. trichiura are exclusively **intestinal parasites.**

Other helminths listed above have **both intestinal and tissue phases.**

Ascaris lumbricoides (Large intestinal roundworm)

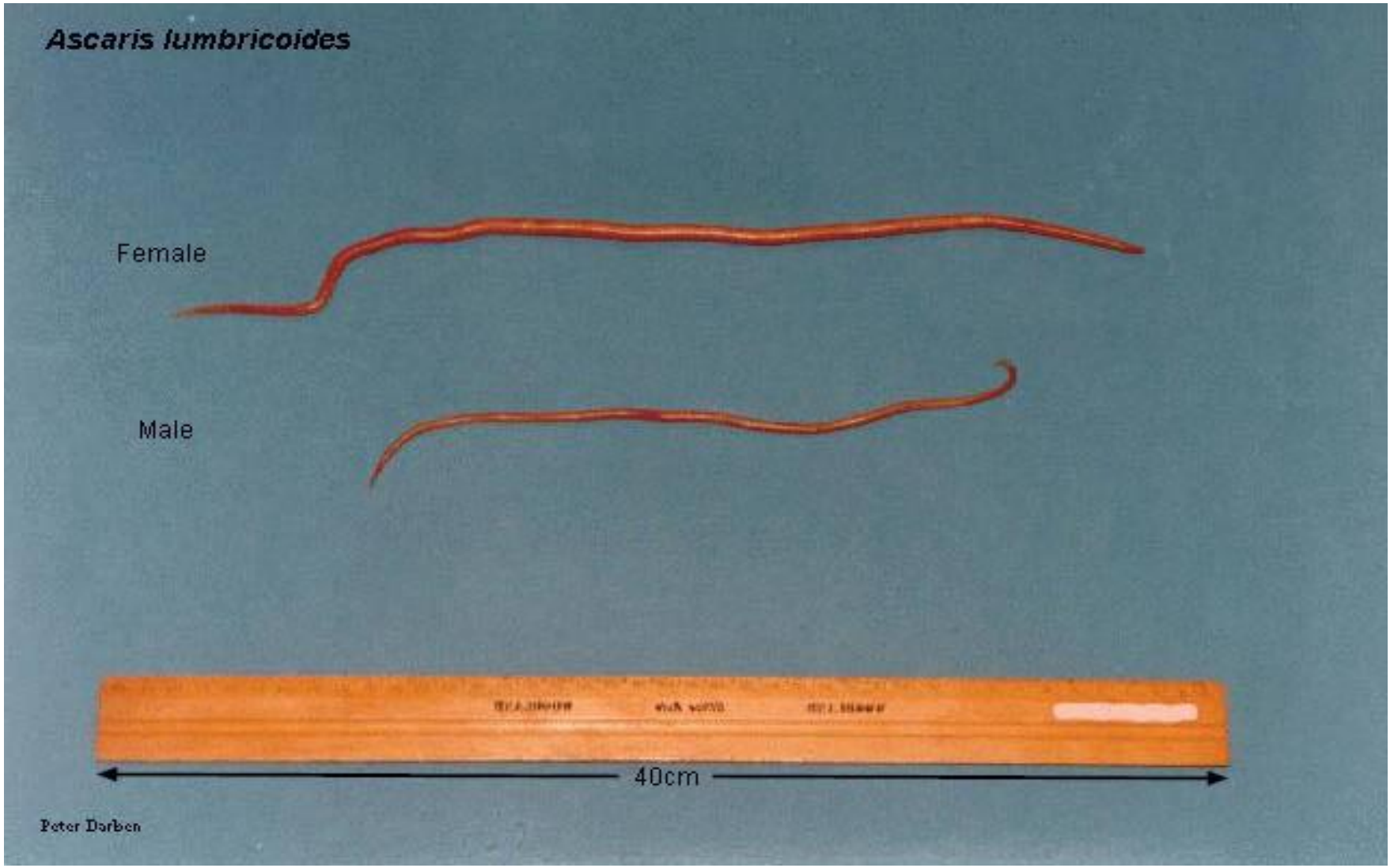
Epidemiology

The annual global morbidity due to **ascaris** infections is estimated at **1 billion** with a mortality of 20,000. **Ascariasis** can occur at all ages, but it is more prevalent in the 5 to 9 years age group. The incidence is higher in **poor rural populations.**

Morphology

The average female worm measures **30 cm x 5 mm**. The male is smaller.

An adult Ascaris worm. Diagnostic characteristics: tapered ends; length 15-35 cm (the females tend to be the larger ones). This worm is a female, as evidenced by the size and genital girdle (the dark circular groove at bottom area of image). Worm passed by a female child in Florida.



***Ascaris lumbricoides* adult male and female**

Life cycle (figure 1)

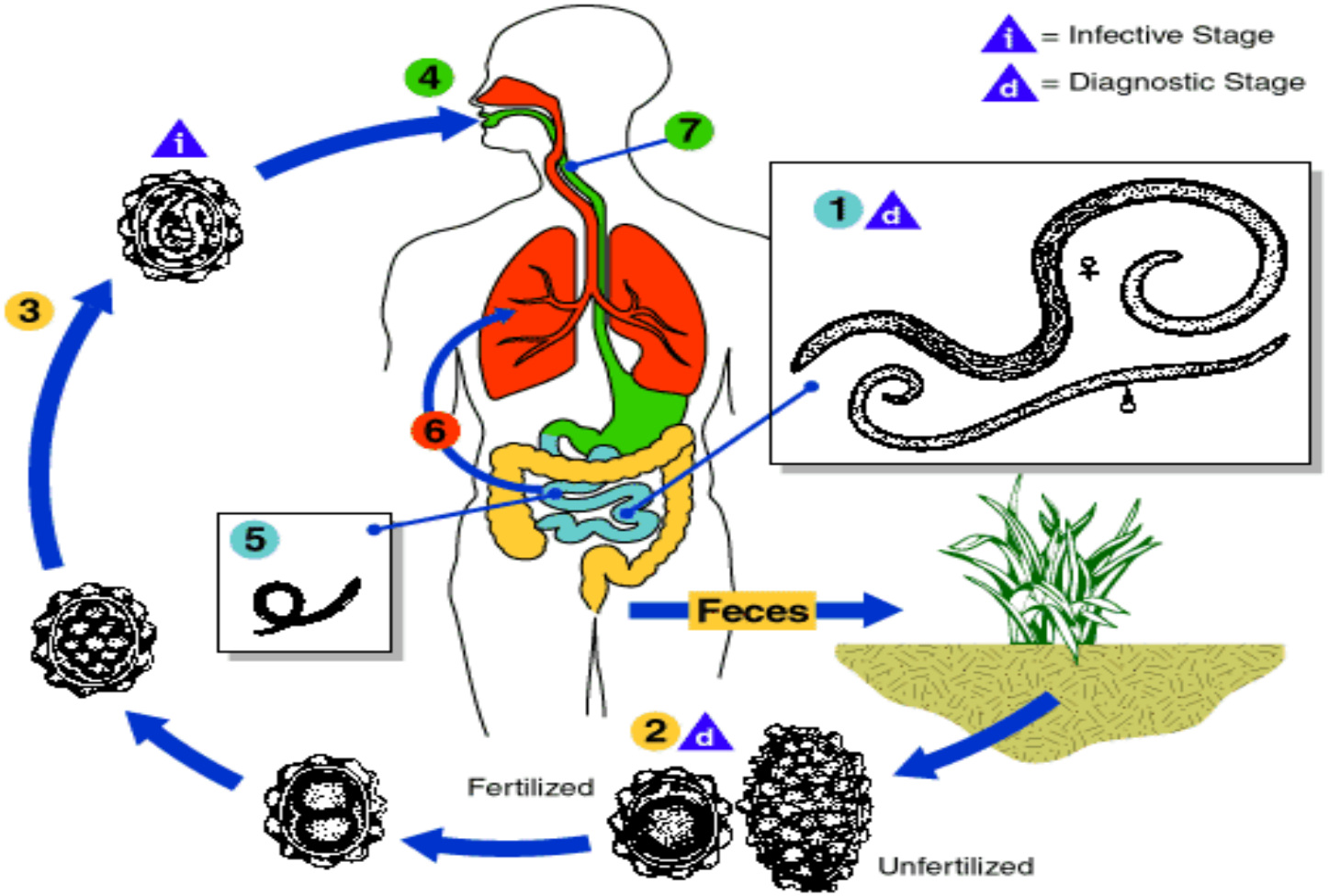
The infection occurs by ingestion of food contaminated with **infective eggs** which **hatch in the upper small intestine.**

The larvae (250 x 15 micrometers) penetrate the **intestinal wall** and enter the **venules or lymphatics.** The **larvae** pass through the **liver, heart** and **lung** to reach **alveoli** in 1 to 7 days during which period they grow to 1.5 cm.

They migrate up the **bronchi**, ascend the **trachea** to the **glottis,** and pass down the **esophagus** to the **small intestine** where they mature in 2 to 3 months. A **female** may live in the **intestine** for 12 to 18 months and has a capacity of producing **25 million eggs** at an average **daily** output of **200,000 (figure 2).**

The **eggs are excreted** in **feces**, and under suitable conditions (21 to 30 degrees C, moist, aerated environment) infective larvae are formed

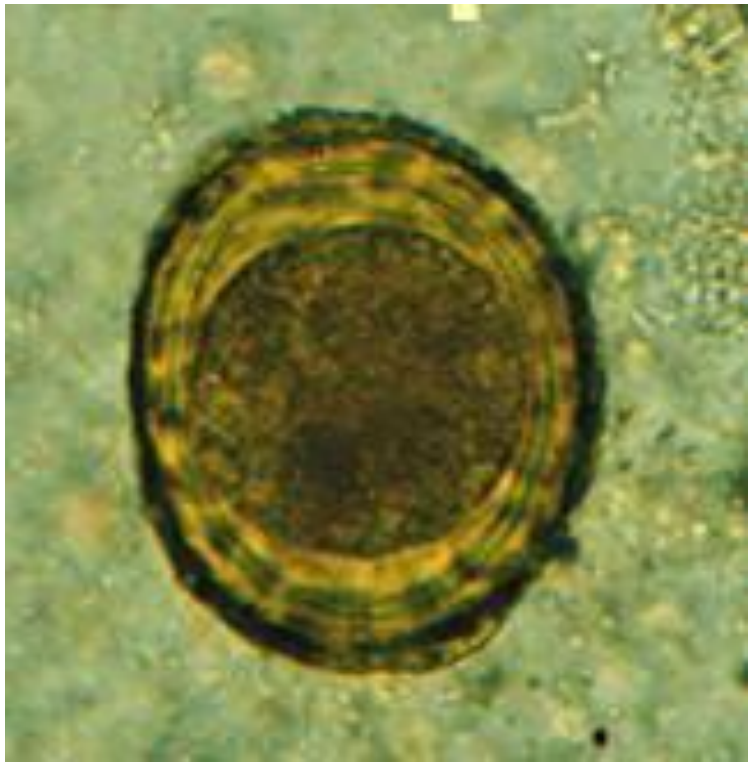
within the egg. The **eggs** are resistant to chemical disinfectant and survive for months in sewage, but are killed by heat (40 degrees C for 15 hours). The infection is man to man. Auto infection can occur.



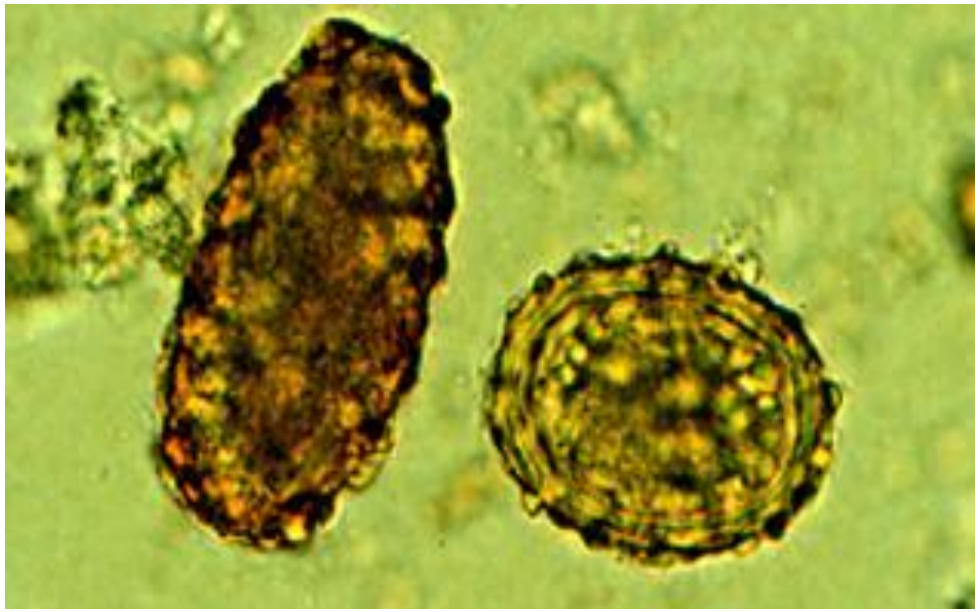
Ascaris Life Cycle

Adult worms 1 live in the **lumen of the small intestine.** A **female** may produce approximately **200,000 eggs per day,** which are passed with the feces **2.** Unfertilized eggs may be ingested but are not infective. **Fertile eggs** embryonate and become infective after 18 days to several weeks **3,** depending on the environmental conditions (optimum: moist, warm, shaded soil). After **infective eggs** are swallowed **4,** the **larvae** hatch **5,** invade the **intestinal mucosa**, and are carried via the **portal**, then **systemic circulation** to the **lungs 6.** The **larvae mature** further in the **lungs** (10 to 14 days), penetrate the **alveolar walls**, ascend the **bronchial tree** to the **throat,** and are **swallowed 7.** Upon reaching the **small intestine,** they develop into **adult worms 1**.

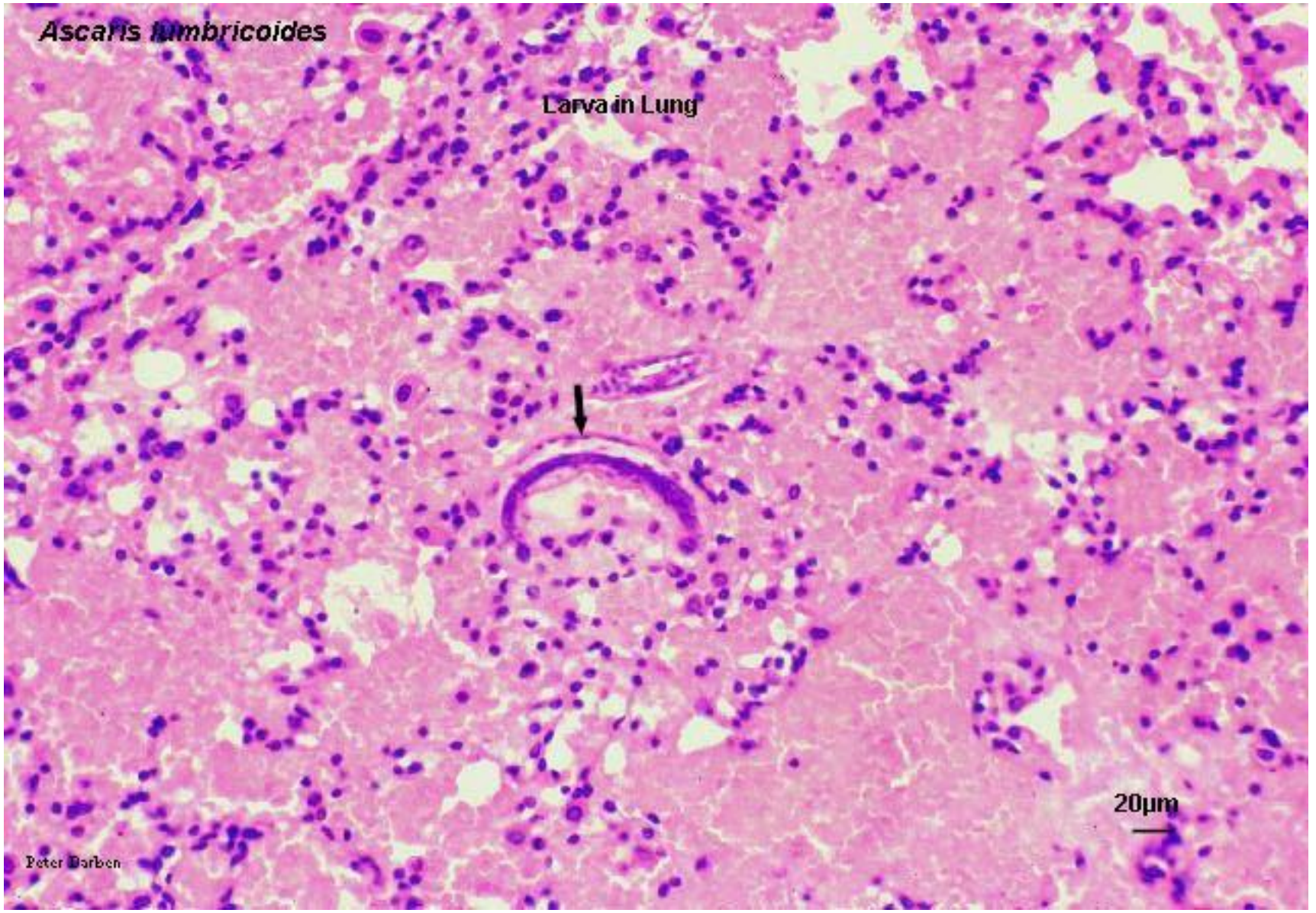
Between 2 and 3 months are required from ingestion of the **infective eggs** to **oviposition by the adult female**. **Adult worms** can live 1 to 2 years. CDC



A fertilized Ascaris egg, still at the unicellular stage, as they are when passed in stool. Eggs are normally at this stage when passed in the stool (Complete development of the larva requires 18 days under favorable conditions). CDC DPDx Parasite Image Library



Eggs, unfertilized (left) and **fertilized (right).** Patient seen in Haiti. CDC DPDx Parasite Image Library



Ascaris lumbricoides larva in section of **lung** (H&E)

Symptoms

Symptoms are related to the **worm burden.** Ten to twenty worms may go unnoticed except in a routine stool examination. The commonest complaint is **vague abdominal pain.**

In more **severe cases,** the patient may experience **listlessness, weight loss**, **anorexia**, **distended abdomen**, **intermittent loose stool** and occasional **vomiting**. During the **pulmonary stage**, there may be a brief period of **cough, wheezing,** dyspnea and **sub-sternal discomfort**. Most symptoms are due to the **physical presence of the worm.**

Diagnosis

- **Diagnosis** is based on **identification of eggs** (40 to 70 micrometers by 35 to 50 micrometers - figure 2) in the **stool.**

Treatment and Prevention

Mebendazole, 200 mg, for **adults** and 100 mg for **children**, for 3 days is effective. **Good hygiene** is the best **preventive** measure.

Trichinella spiralis (Trichinosis)

Epidemiology

Trichinosis is related to the quality of **pork** and consumption of poorly cooked **meat**. **Autopsy** surveys indicate about 2 percent of the population is infected. The mortality rate is low.

Morphology

The **adult female** measures 3.5 mm x 60 micrometers. The **larvae** in the **tissue** (100 micrometers x 5 micrometers) are coiled in a lemon-shaped capsule.

Life cycle

Infection occurs by **ingestion of larvae**, in **poorly cooked meat**, which immediately invade **intestinal mucosa** and sexually differentiate within 18 to 24 hours.

The female, after fertilization, burrows deeply in the **small intestinal mucosa**, whereas the male is dislodged (intestinal stage).

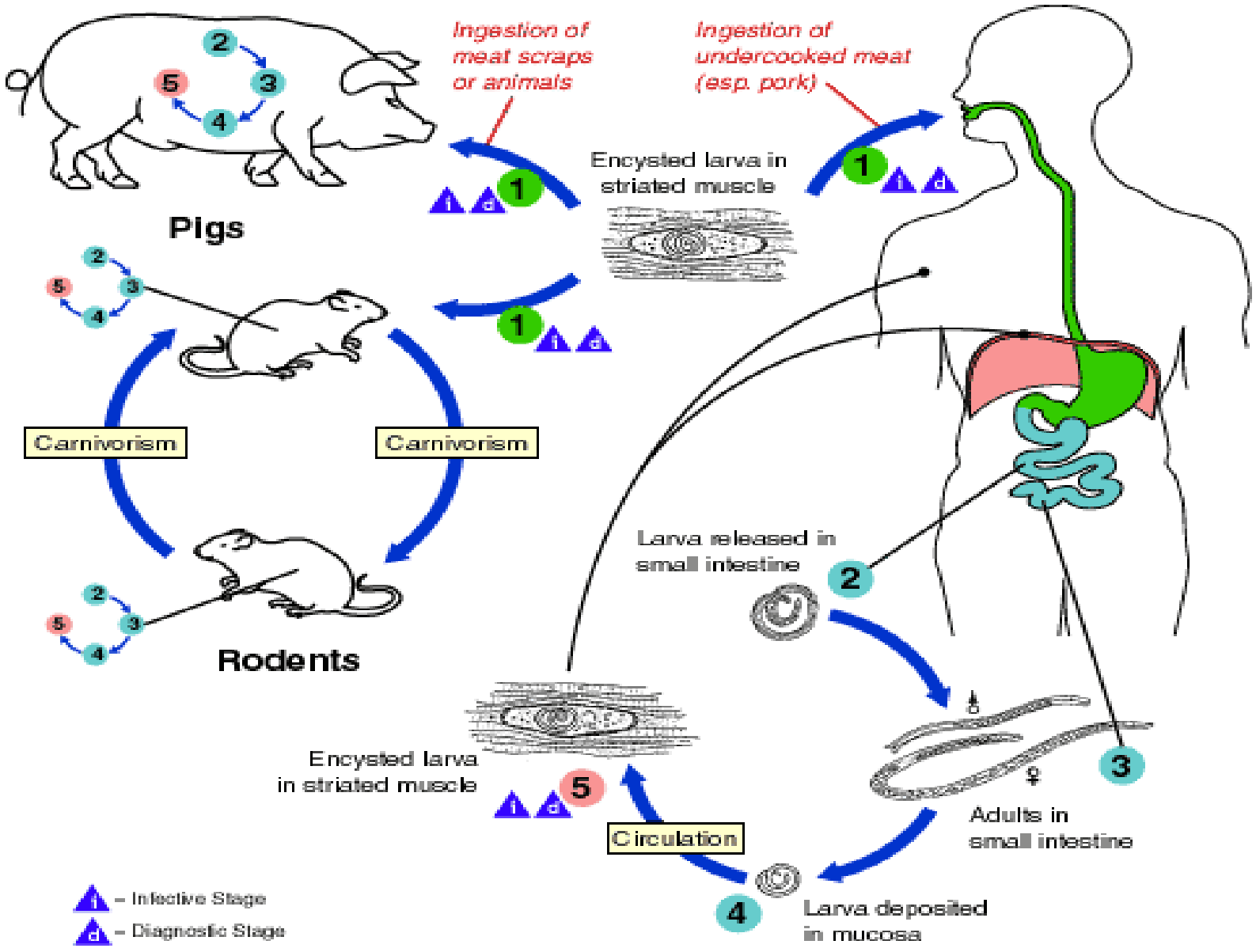
On about the 5th day **eggs** begin to **hatch** in the **female worm** and young **larvae** are deposited in the **mucosa** from where they reach the **lymphatics, lymph nodes** and the **blood stream** (**larval migration**).

Larval dispersion occurs 4 to 16 weeks after infection.

The **larvae** are deposited in **muscle fiber** and, in **striated muscle**, they form a capsule which calcifies to form a cyst.

In non-striated tissue, such as **heart** and **brain**, the larvae do not calcify; they die and disintegrate.

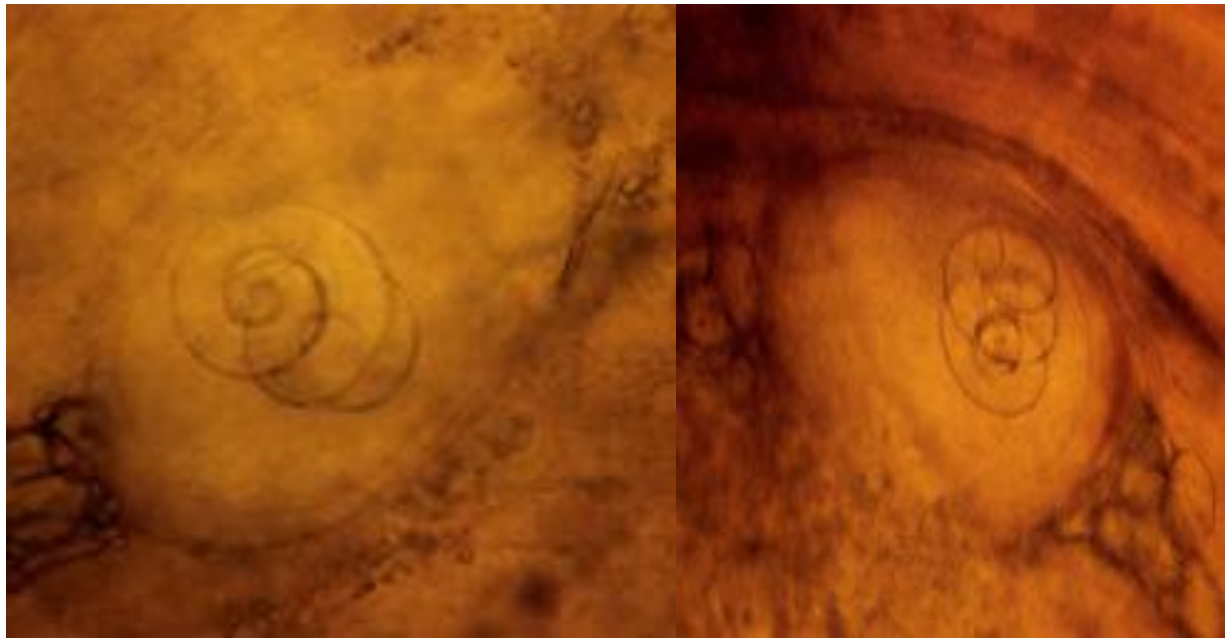
The **cyst** may persist for several years. One **female** worm produces approximately **1500 larvae**. Man is the terminal host. The **reservoir** includes most **carnivorous** and **omnivorous animals (Figure 3 and 4).**


Ingestion of meat scraps or animals
Ingestion of undercooked meat (esp. pork)
Encysted larva in striated muscle
Pigs
Carnivorism
Carnivorism
Rodents
Larva released in small intestine
Encysted larva in striated muscle
Adults in small intestine
Circulation
Larva deposited in mucosa
i – Infective Stage
d – Diagnostic Stage

Trichinellosis is acquired by ingesting **meat containing cysts** (**encysted larvae**) **1** of ***Trichinella*.** After exposure to gastric acid and pepsin, the **larvae** are released **2** from the cysts and invade **the small bowel mucosa** where they develop into **adult worms 3** (female 2.2 mm in length, males 1.2 mm; life span in the small bowel: 4 weeks). After 1 week, the females release **larvae 4** that migrate to the **striated muscles** where they **encyst 5.** Encystment is completed in 4 to 5 weeks and the **encysted larvae** may remain viable for several years. Ingestion of the **encysted larvae** perpetuates the cycle. **Rats and rodents** are primarily responsible for maintaining the endemicity of this infection.

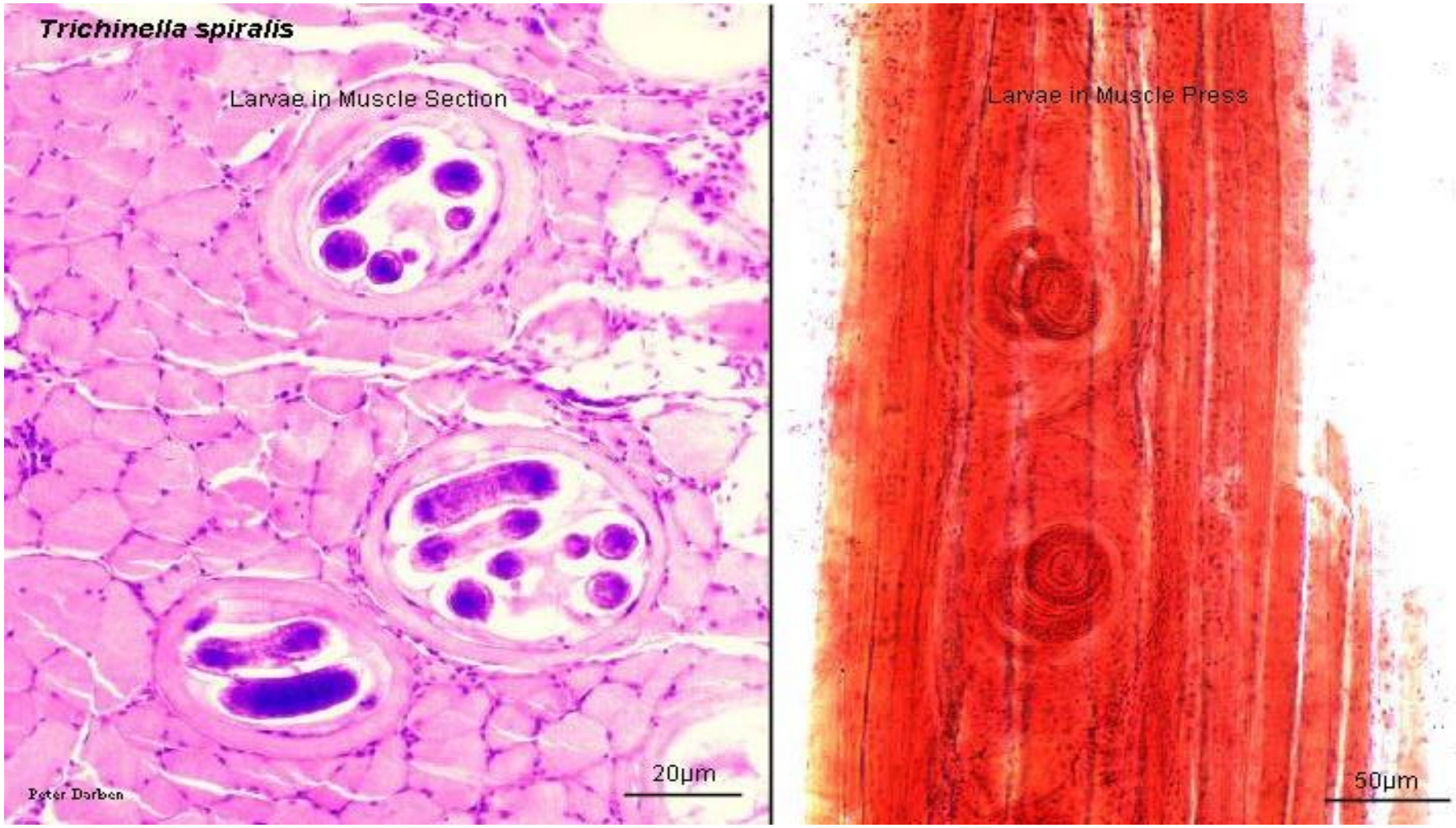
Carnivorous/omnivorous animals, such as pigs or bears, feed on infected **rodents or meat from other animals**.

Humans are **accidentally** infected when eating **improperly processed meat** of these **carnivorous animals** (or eating food contaminated with such meat). CDC DPDx Parasite Image Library



Encysted larvae of **Trichinella** in pressed muscle tissue. The coiled larvae can be seen inside the cysts.

Larvae of Trichinella, freed from their cysts, typically coiled; length: .8 to 1 mm. Alaskan bear.



***Trichinella spiralis* larvae** in muscle section (H&E) and muscle press

Symptoms

Trichinosis symptoms depend on the **severity of infection**: **mild** infections may be **asymptomatic**. A **larger bolus** of infection produces **symptoms** according to the **severity** and **stage of infection and organs involved** (Table 1).

Table 1 Trichinosis symptomatology			
Intestinal mucosa (24-72 hrs)	Circulation and muscle (10-21 days)	Myocardium (10-21 days)	Brain and meninges (14-28 days)
Nausea, vomiting diarrhea, abdominal pain, headache.	Edema, peri-orbital conjunctivitis, photo phobia, fever, chill, sweating, muscle pain, spasm, eosinophilia.	Chest pain, tachycardia, EKG changes, edema of extremities, vascular thrombosis.	Headache (supraorbital), vertigo, tinnitus, deafness, mental apathy, delirium, coma, loss of reflexes.

Pathology and Immunology

- Trichinella pathogenesis is due the presence of **large numbers of larvae in vital muscles** and **host reaction to larval metabolites.**

- The muscle fibers become **enlarged edematous and deformed.** The **paralyzed muscles** are **infiltrated** with **neutrophil, eosinophils and lymphocytes.**

- Splenomegaly is dependent on the **degree of infection.**

- The worm induces a **strong IgE response** which, in association with **eosinophils,** contributes to **parasite death.**

Treatment and Control

- Steroids are use for treatment of **inflammatory symptoms** and **Mebendazole** is used to **eliminate worms. Elimination of parasite** infection in **hogs and adequate cooking of meat** are the best ways of **avoiding infection.**

Parasitology
Helminthes/ Trematodes
Pulmonary Trematodes/
Helminthes/Cestododa

PARAGONIMUS WESTERMANI (LUNG FLUKE)

Epidemiology

Lung fluke is most commonly encountered in parts of Asia, Africa and South America.

Morphology

It is a plump reddish brown oval worm measuring 10 by 4 mm. The ovum measures 85 by 55 micrometers (figure 1).

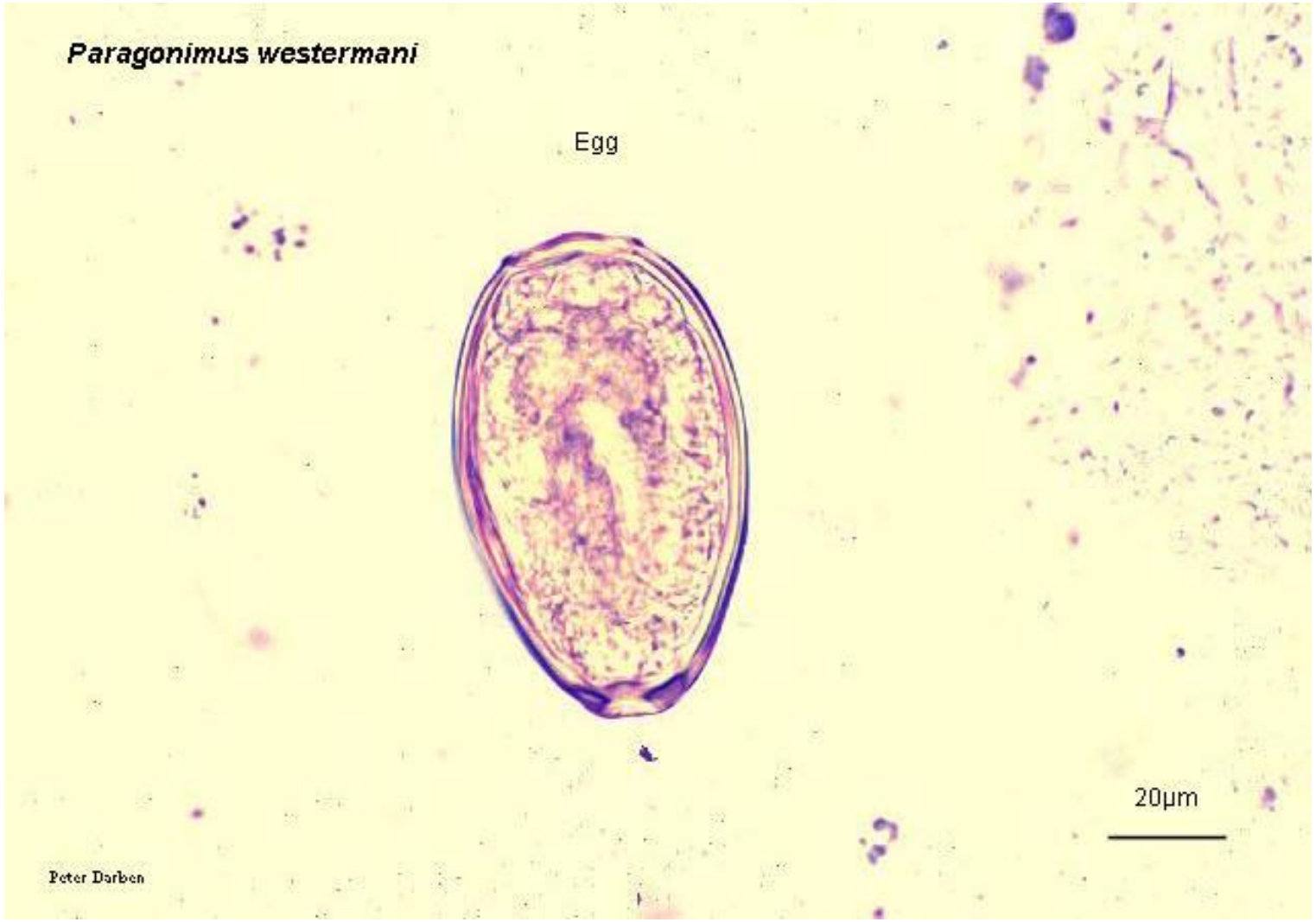


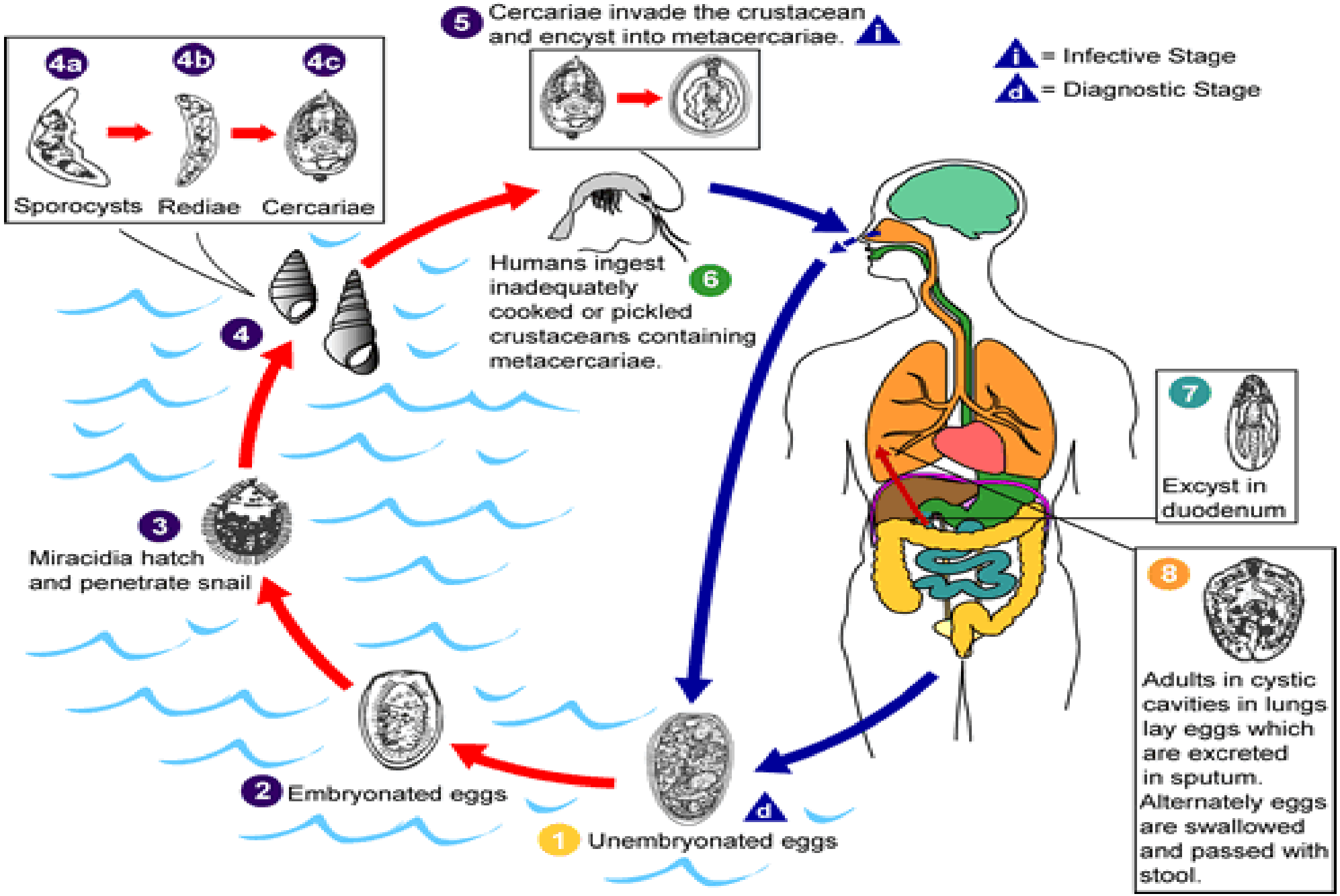
Figure. 1a, *Paragonimus westermani* egg

***Figure 1 b, Paragonimus westermani* adult, carmine stain**

Figure 1c, *Paragonimus westermani* adult in section of lung (H&E)

Life cycle

Lung fluke infects man (and domestic carnivores) when **crabmeat** infested with **encysted metacercaria** is consumed. The **metacercaria** reach the small intestine, exit their shell and bore their way, as young flukes, through the intestinal wall, through the thoracic diaphragm and penetrate the lung. There, they become enclosed in 1 to 2 cm cysts and reach maturity. The **eggs** are found in the sputum or, if swallowed, in the feces, 2 to 3 months after infection. The **eggs**, when introduced in fresh water produce a **miracidia** which penetrates the suitable **snail**. In the snail they develop into **cercaria** which break out in water and penetrate gills, muscle or viscera of **fresh water crabs** and become encysted in flesh as **metacercaria** (**figure 2**).


5 Cercariae invade the crustacean and encyst into metacercariae.
i = Infective Stage
d = Diagnostic Stage
4a
4b
4c
Sporocysts
Rediae
Cercariae
Humans ingest inadequately cooked or pickled crustaceans containing metacercariae.
6
4
3
Miracidia hatch and penetrate snail
2 Embryonated eggs
1 Unembryonated eggs
d
7
Excyst in duodenum
8
Adults in cystic cavities in lungs lay eggs which are excreted in sputum. Alternately eggs are swallowed and passed with stool.

Paragonimus westermani (Lung Fluke) Life Cycle

The **eggs** are excreted unembryonated in the sputum, or alternately they are swallowed and passed with stool **1**. In the external environment, the eggs become embryonated **2**, and **miracidia** hatch and seek the first **intermediate host**, **a snail**, and penetrate its soft tissues **3**. Miracidia go through several developmental stages inside the snail **4**: **sporocysts 4a**, **Rediae 4b**, with the latter giving rise to many **cercariae 4c**, which emerge from the snail. The cercariae invade the second intermediate host, a crustacean such as a crab or crayfish, where they encyst and become **metacercariae**. This is the **infective stage** for the mammalian host **5**. Human infection with *P. westermani* occurs by eating inadequately cooked or pickled crab or crayfish that harbor metacercariae of the parasite **6.**

The **metacercariae** excyst in the duodenum **7**, penetrate through the intestinal wall into the peritoneal cavity, then through the abdominal wall and diaphragm into the lungs, where they become encapsulated and develop into **adults 8** (7.5 to 12 mm by 4 to 6 mm). The worms can also reach other organs and tissues, such as the brain and striated muscles, respectively. However, when this takes place completion of the life cycles is not achieved, because the eggs laid cannot exit these sites. Time from infection to oviposition is 65 to 90 days. Infections may persist for 20 years in humans. Animals such as pigs, dogs, and a variety of feline species can also harbor *P. westermani*.

Symptoms

The fluke provokes the development of a fibrous tissue capsule with bloody purulent material containing eggs. There is inflammatory infiltrate around the capsule. The symptoms include a dry cough, followed by production of blood stained rusty brown sputum. Pulmonary pain and pleurisy may develop. Worms may migrate to the brain where they lay eggs and cause a granulomatous abscess resulting in symptoms similar to epilepsy.

Diagnosis

Diagnosis is based on history and symptoms. Eggs are found in rust colored sputum, often being examined for tuberculosis.

Treatment and control

Praziquantel taken orally is quite effective. Adequate cooking of crustaceans is a preventive measure. Improved sanitary conditions have lowered the infection rate in endemic areas.

Summary

Organism	Transmission	Symptoms	Diagnosis	Treatment
S. mansoni ***S. japonicum***	skin penetration by cercaria	Dermatitis, abdominal pain, bloody stool, peri-portal fibrosis, hepato-splenomegaly, ascites, CNS	Eggs in stool	Praziquantel
Schistosoma hematobium	skin penetration by cercaria	Dermatitis, urogenital cystitis, urethritis and bladder carcinoma	Eggs in urine	Praziquantel
Fasciolopsis buski	Metacercaria on water chestnut	Epigastric pain, nausea, diarrhea, edema, ascites	Eggs in stool	Praziquantel,
C. sinensis ***O. felinus*** ***O. viverini***	Cysts in fish	Inflammation and deformation of bile duct, hepatitis, anemia and edema	Eggs in stool	Praziquantel
Paragonimus westermani	Cyst in crab meat	Cough (dry / rusty brown sputum), pulmonary pain, pleurisy, tuberculosis-like	Eggs in sputum	Praziquantel

CESTODES (TAPE WORMS)

TENIA SOLIUM T. SAGINATA (TENIASIS)

Epidemiology

These cestodes have a worldwide distribution but incidence is higher in developing countries. Infection rate is as low as 1 per 1000 in most of North America and as high as 10% in the third world. Pork tapeworm shows a higher incidence but this is dependent on dietary habits.

Morphology

T. saginata can be up to 4 to 6 meters long and 12 mm broad; it has a pear-shaped head (**scolex**) with four suckers but no hooks or neck. It has a long flat body with several hundred segments (**proglottids**). Each segment is about 18 x 6 mm with a branched uterus (15-30 branches). The egg is 35 x 45 micrometers, roundish and yellow-brown. It has peripheral radial striations and contains an embryo with 3 hooklets (figure 3).

T. solium is slightly smaller than ***T. saginata***. It has a globular **scolex** with four suckers and a circular row of hooks (rostellum) that gives it a solar appearance. There is a **neck** and it has a long flat body (0.1 meter in length). The **proglottids** are 5 x 10 mm with a 7-12 branch uterus. The eggs of *T. solium* and *T. saginata* are indistinguishable (figure 3).

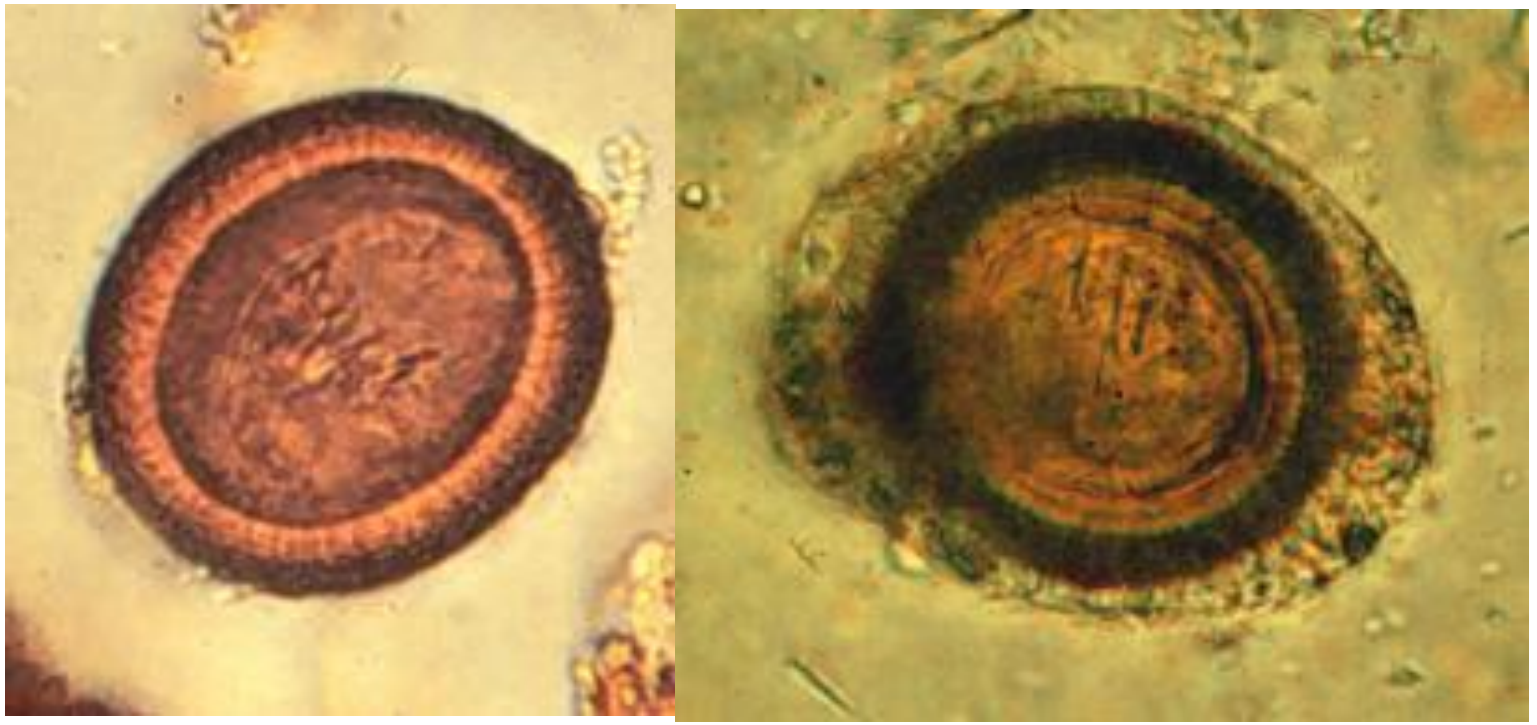


Figure 3:**Taeniid eggs**. The eggs of ***Taenia saginata*** and ***T. solium*** are undistinguishable morphologically (morphologic species identification will have to rely on the proglottids or scolices). The eggs are rounded or subspherical, diameter 31 - 43 µm, with a thick radially striated brown shell. Inside each shell is an **embryonated oncosphere** with 6 hooks. The egg in B still has the primary membrane that surrounds eggs in the proglottids. CDC

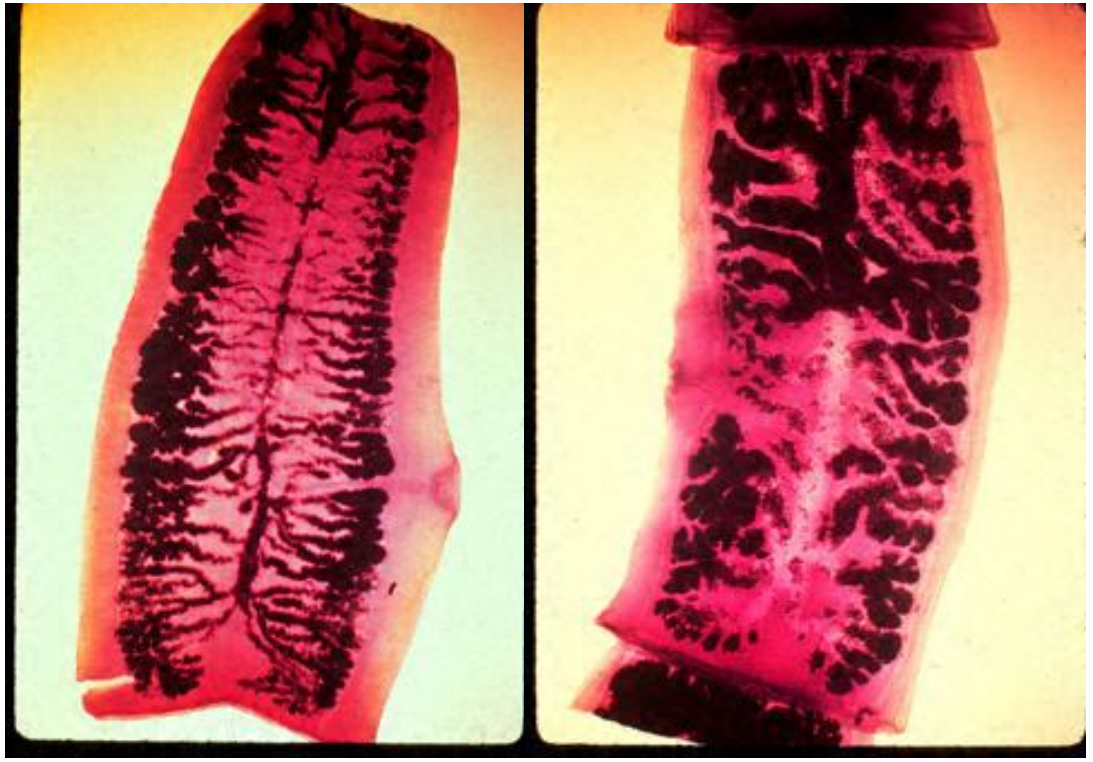
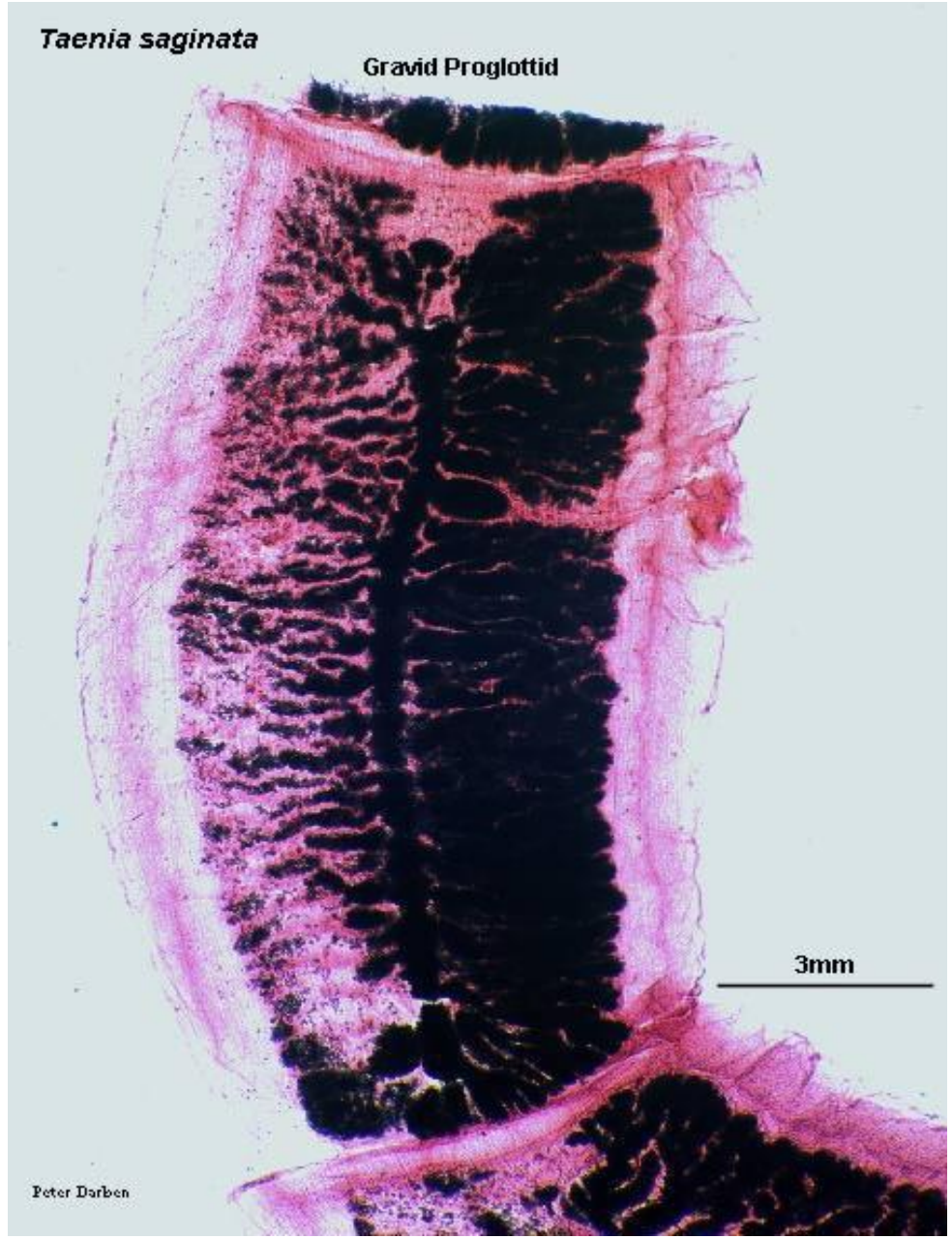


Figure 4: Gravid proglottids of (left) ***Taenia saginata*** and (right) ***T. solium***. Injection of India ink in the uterus allows visualization of the primary lateral branches. Their number allows differentiation between the two species: ***T. saginata*** **has 15 - 20 branches** on each side, while ***T. solium*** **has 7 - 13**. Note the genital pores in mid-lateral position. CDC


Taenia saginata
Gravid Proglottid
3mm
Peter Darben

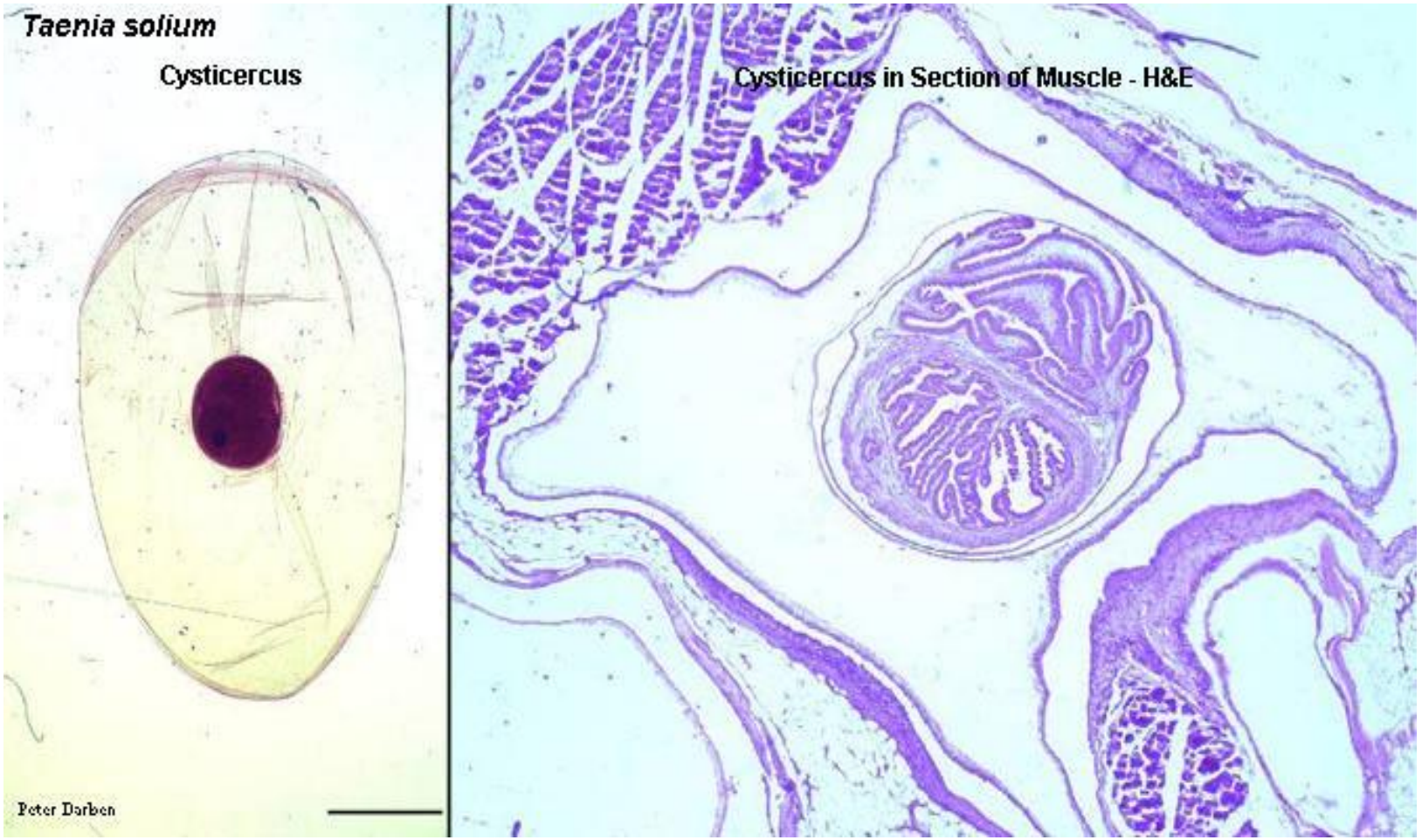


***Figure 5: Taenia solium* cysticercus**, whole and in section of muscle (H&E)

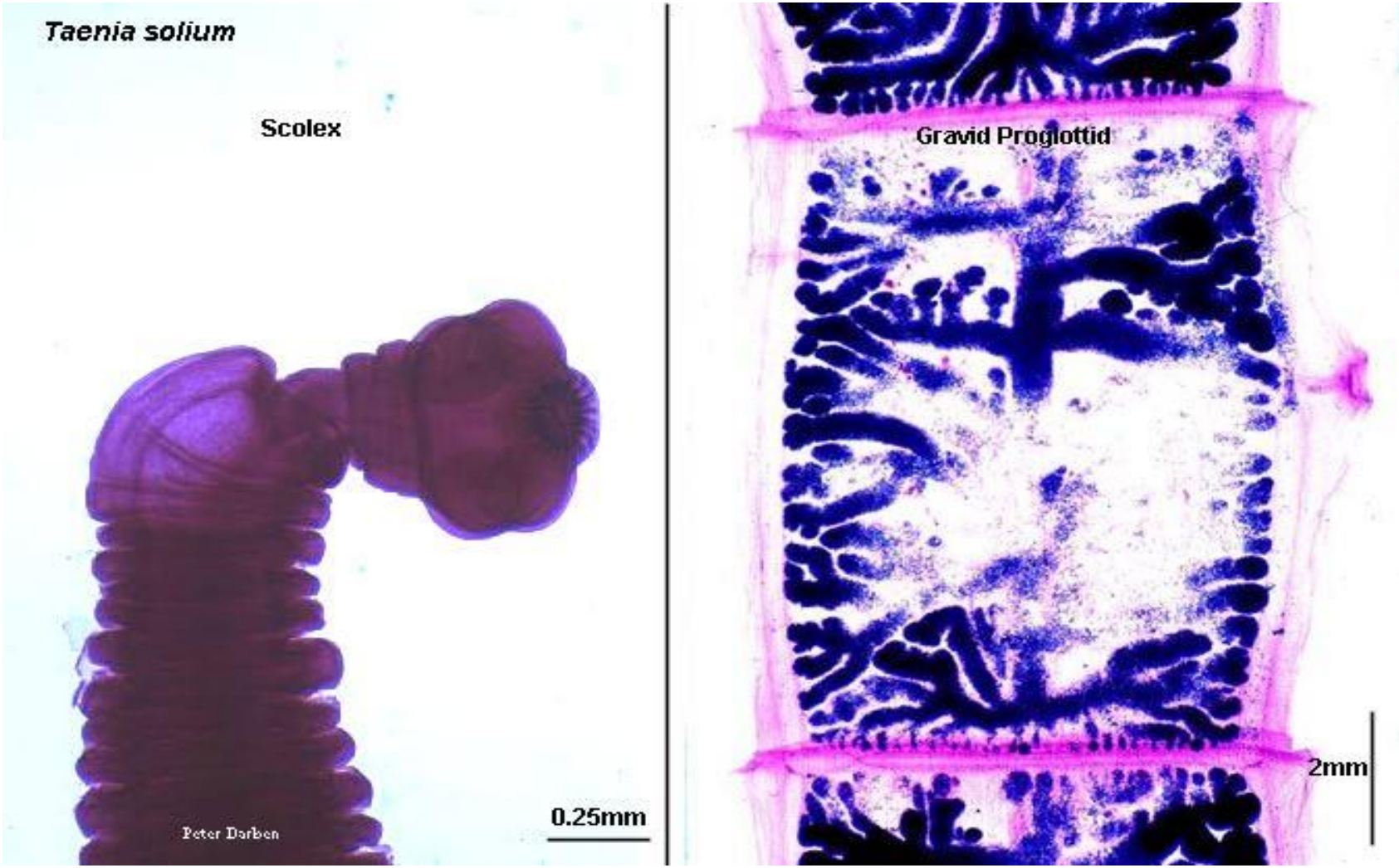


Figure 6: Taenia solium scolex and gravid proglottid

Life cycle

A tapeworm larval cyst (cysticercus) is ingested with poorly cooked infected meat; the larva escapes the cyst and passes to the small intestine where it attaches to the mucosa by the scolex suckers. The proglottids develop as the worm matures in 3 to 4 months. The adult may live in the small intestine as long as 25 years and pass gravid proglottids with the feces. Eggs extruded from the proglottid contaminate and persist on vegetation for several days and are consumed by cattle or pigs in which they hatch and form cysticerci (Figure 7).

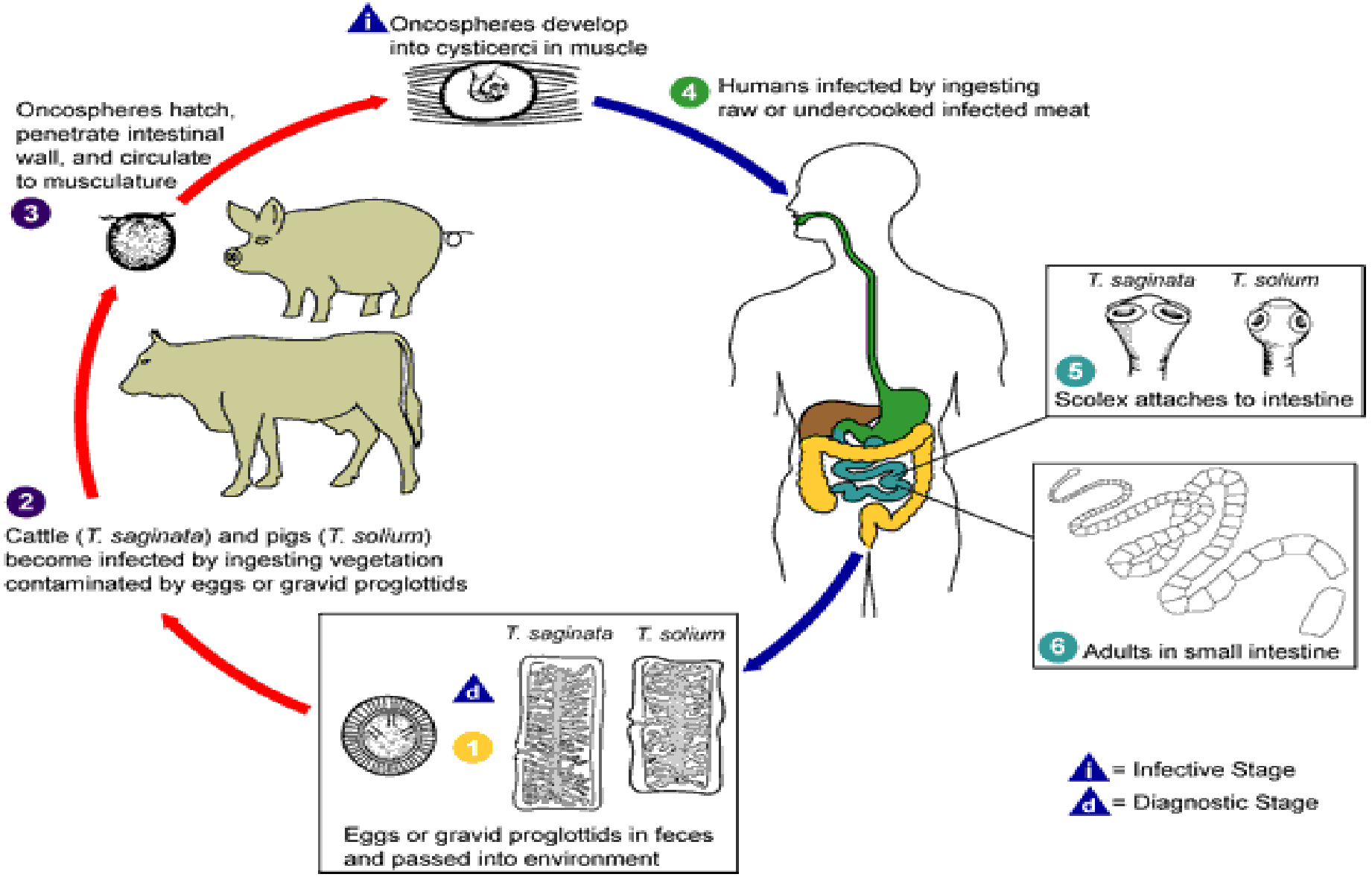


Figure 7: Life cycle of ***Taenia saginata*** and ***Taenia solium***

Humans are the only definitive hosts for ***Taenia saginata*** and ***Taenia solium***. **Eggs** or **gravid proglottids** are passed with feces **1**; the **eggs** can survive for days to months in the environment. **Cattle** (***T. saginata***) and **pigs** (***T. solium***) become infected by ingesting vegetation contaminated with eggs or gravid proglottids **2.** In the animal's intestine, the **oncospheres** hatch **3,** invade the intestinal wall, and migrate to the striated muscles, where they develop into **cysticerci**. A **cysticercus** can survive for several years in the animal. Humans become infected by ingesting raw or undercooked infected meat **4.** In the human intestine, the **cysticercus** develops over 2 months into an **adult tapeworm**, which can survive for years. The **adult tapeworms** attach to the small intestine by their **scolex 5** and reside in the small intestine 6. Length of adult worms is usually **5 m or less** for ***T. saginata*** (however it may reach up to 25 m) and **2 to 7 m** for ***T. solium*.**

The adults produce **proglottids** which mature, become gravid, detach from the tapeworm, and migrate to the anus or are passed in the stool (approximately 6 per day). ***T. saginata* adults usually have 1,000 to 2,000 proglottids**, while ***T. solium* adults have an average of 1,000 proglottids**. The **eggs** contained in the **gravid proglottids** are released after the **proglottids** are passed with the feces. ***T. saginata* may produce up to 100,000** and ***T. solium* may produce 50,000 eggs per proglottid** respectively.

Symptoms

Light infections remain asymptomatic, but heavier infections may produce abdominal discomfort, epigastric pain, vomiting and diarrhea.

Cysticercosis

***T. solium* eggs** can also infect humans and cause **cysticercosis** (**larval cysts in lung, liver, eye and brain**) resulting in blindness and neurological disorders. The incidence of **cerebral cysticercosis** can be as high 1 per 1000 population and may account for up to 20% of neurological case in some countries (e.g., Mexico); **cysticercosis ocular** involvement occurs in about 2.5% of patients and **muscular involvement** is as high as 10% (India).

Pathology and Immunology

Gastrointestinal symptoms are due to the presence of the tape worm. Cysticercosis symptoms are a result of inflammatory/immune responses. Antibodies are produced in cysticercosis and are useful epidemiological tools.

Diagnosis

Diagnosis is based on the recovery of eggs or proglottids in stool or from the perianal area. Cysticercosis is confirmed by the presence of antibodies.

Treatment and control

Praziquantel is the drug of choice. Expulsion of scolex must be assured to assume a satisfactory treatment. A thorough inspection of beef and pork, adequate cooking or freezing of meat are effective precautions, since cysticerci do not survive temperatures below -10^{o} C and above 50^{o} C.

Parasitology
Helminthes/ Cestoda
Diphyllobothrium latum
Echinococcus granulosus
E. multiloculari

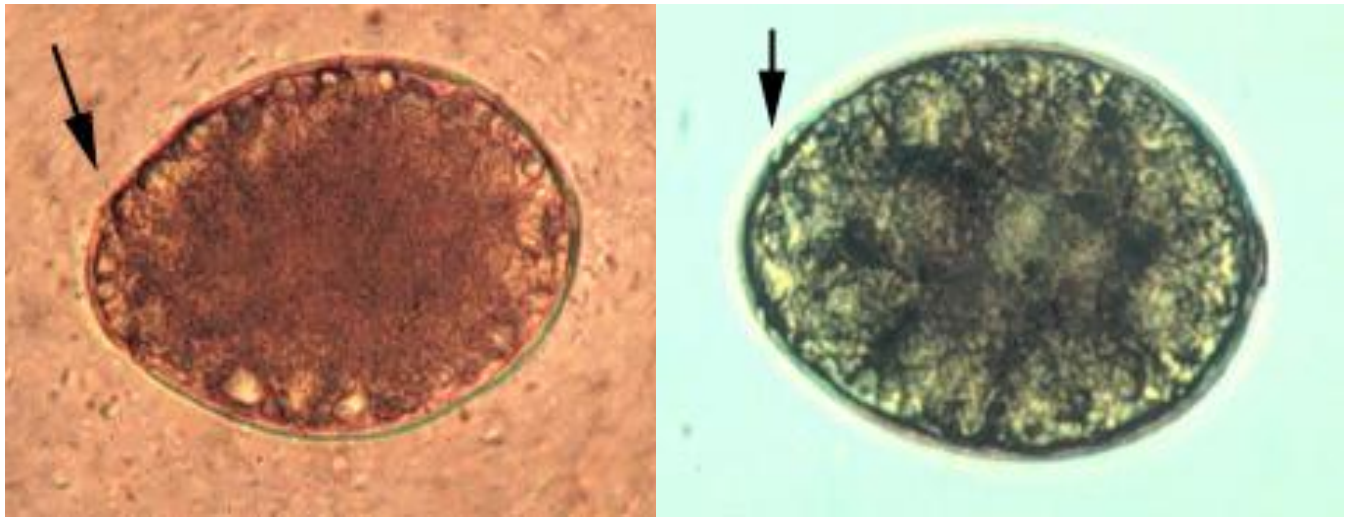
DIPHYLLOBOTHRIUM LATUM (FISH OR BROAD TAPEWORM)

Epidemiology

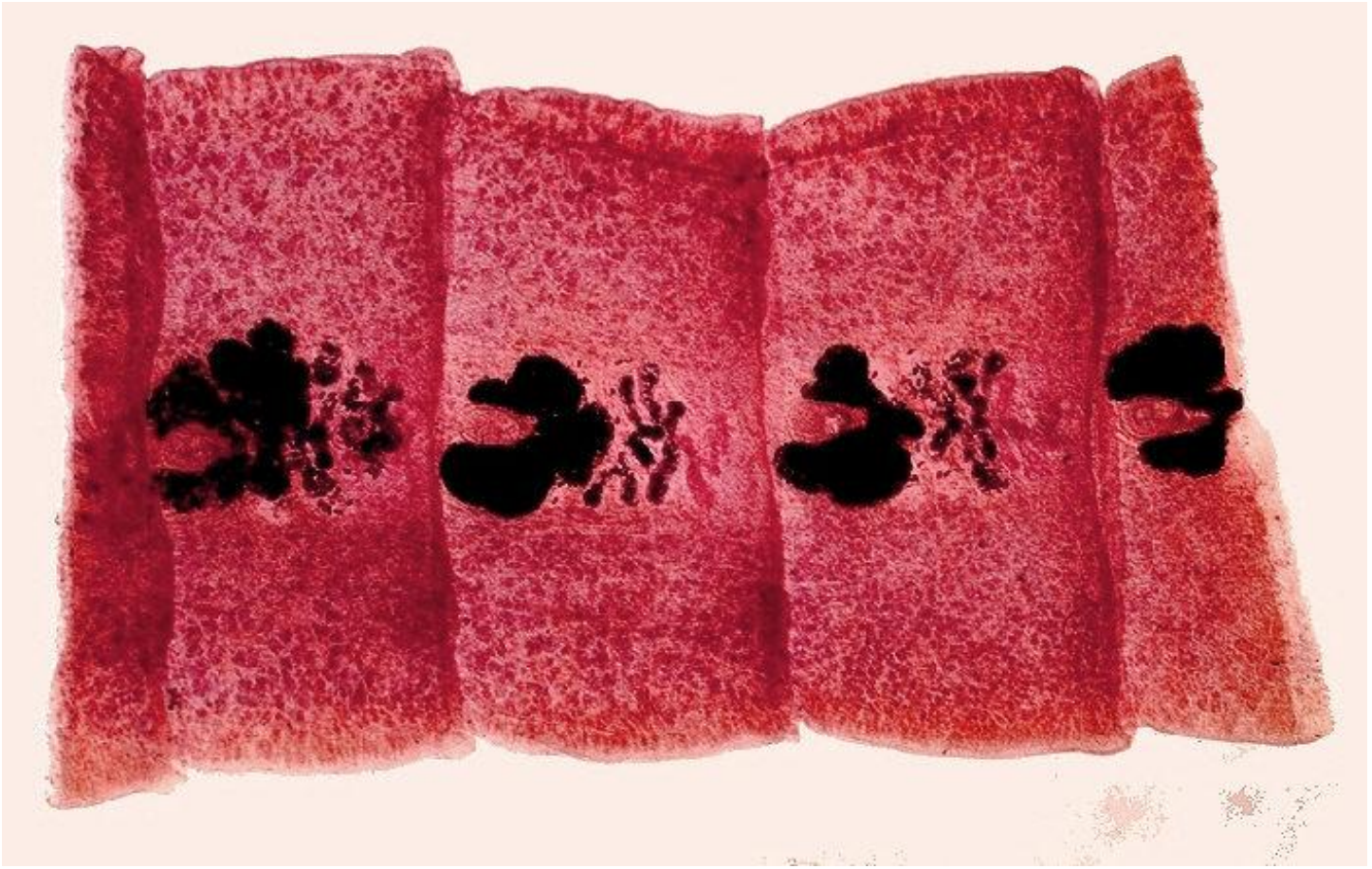
Fish tapeworm infection is distributed worldwide, in the subarctic and temperate regions; it is associated with eating of raw or improperly cooked fresh water fish.

Morphology

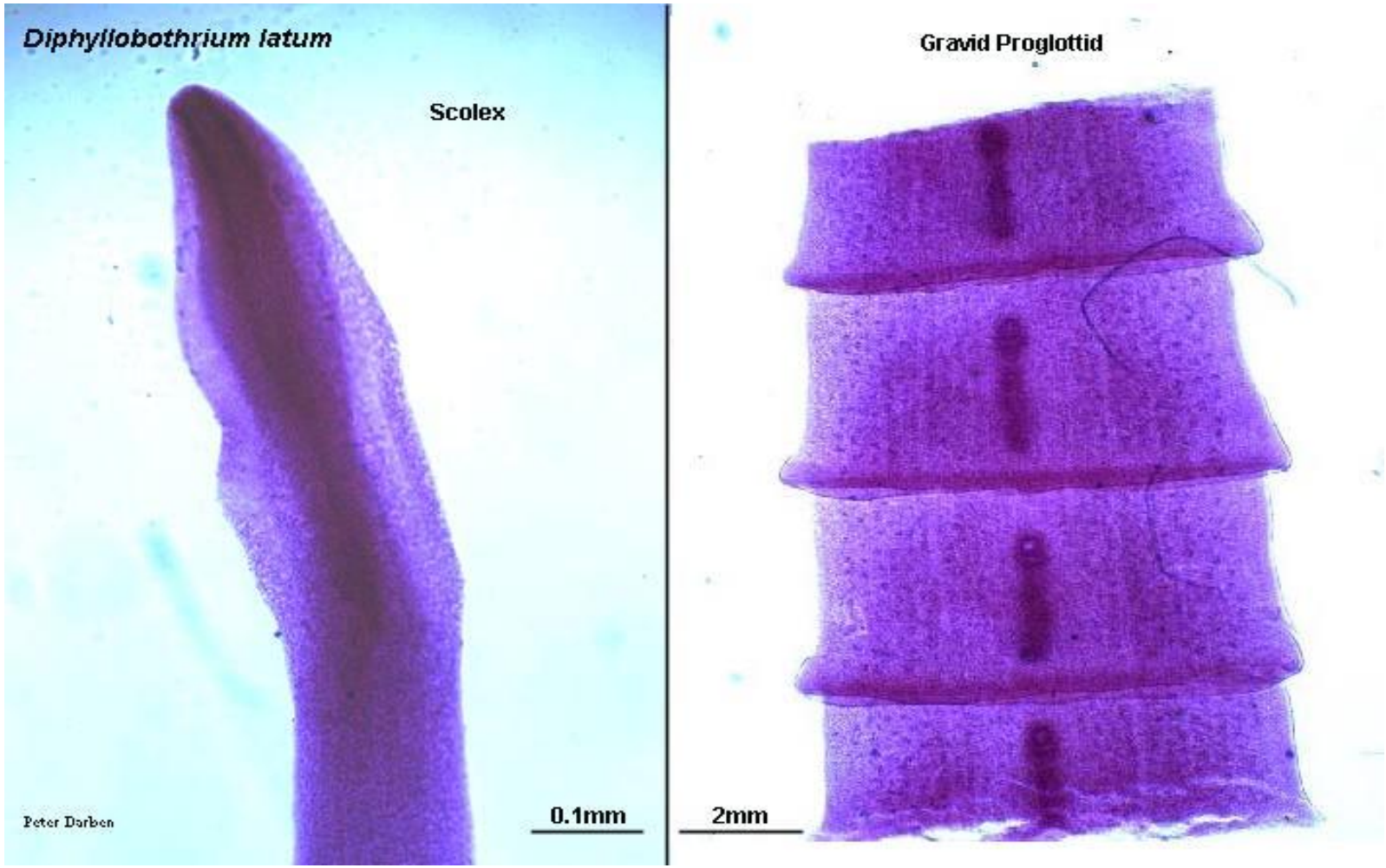
This is the longest tapeworm found in man, ranging from 3-10 meters with more than 3000 proglottids. The scolex resembles two almond-shaped leaves and the proglottids are broader than they are long, a morphology reflected in the organism's name. Eggs are 30 x 50 micrometers in size and contain an embryo with 3 pairs of hooklets.



Eggs of *Diphyllobothrium latum*. These eggs are oval or ellipsoidal, with at one end an operculum (arrows) that can be inconspicuous (right). At the opposite (abopercular) end is a small knob that can be barely discernible (left). The eggs are passed in the stool unembryonated. Size range: 58 to 76 µm by 40 to 51 µm. CDC.



Gravid proglottids of *Diphyllobothrium latum*. CDC

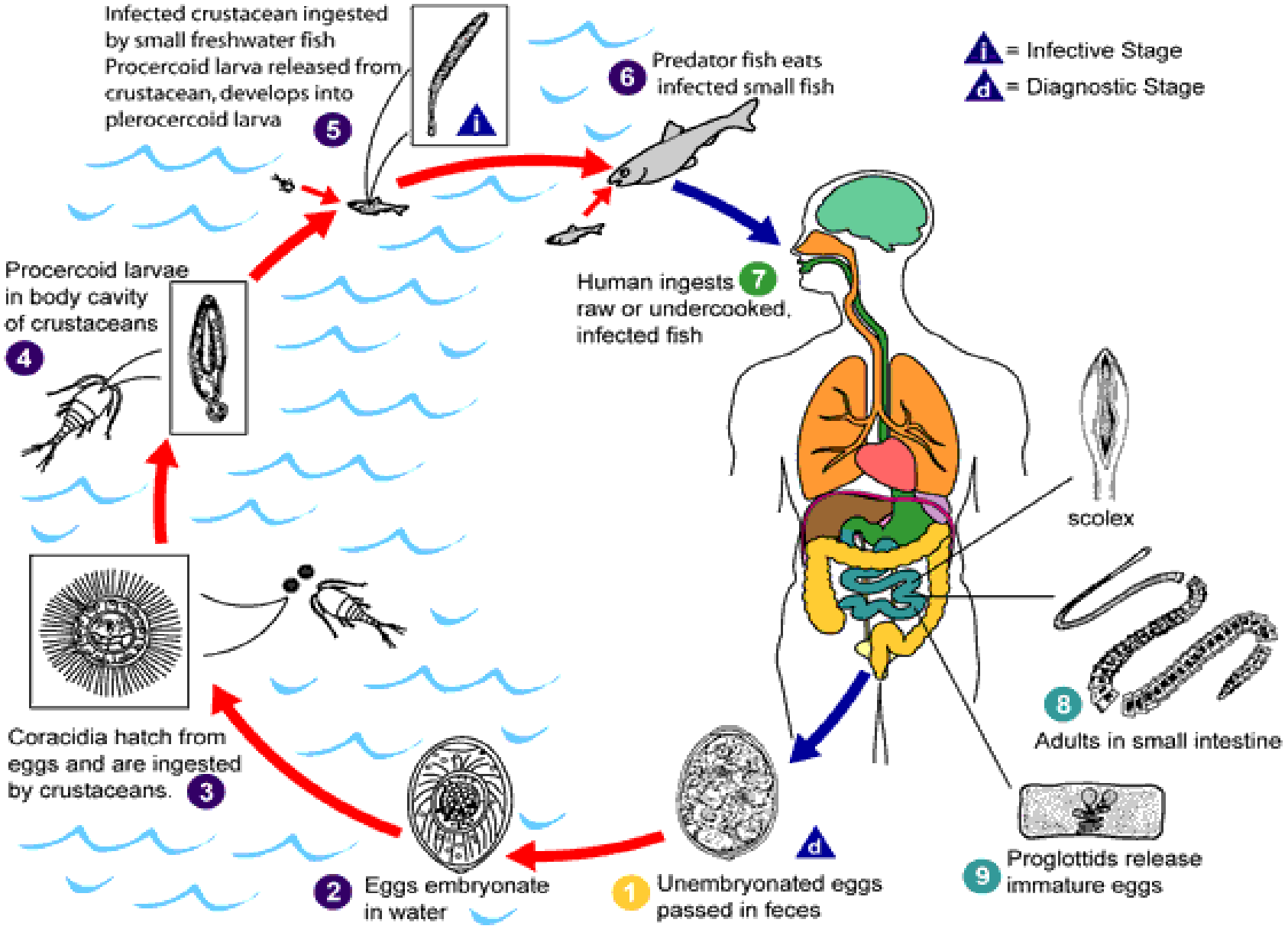


***Diphyllobothrium latum* scolex and gravid proglottids**

Life cycle

Man and other animals are infected by **eating uncooked fish** that contains **plerocercoid larvae** (15 x 2 mm) which attach to **the small intestinal wall** and mature into **adult worms** in 3 to 5 weeks. **Eggs** discharged from **gravid proglottids** in the **small intestine** are passed in the **feces**. The **egg** hatches in fresh water to produce a ciliated **coracidium** which needs to be ingested by a **water flea (Cyclops)** where it develops into a **procercoid larva**.

When **infected Cyclops** are ingested by **the freshwater fish**, the **procercoid** larva penetrates the intestinal wall and develops into **a plerocercoid larva**, infectious to man.



Life cycle of *Diphyllobothrium latum*

Immature **eggs** are passed in feces **1**. Under appropriate conditions, the **eggs** mature (approximately 18 to 20 days) **2**. and yield **oncospheres** which develop into a **coracidia 3**. After ingestion by a suitable **freshwater crustacean** (**the copepod first intermediate host**) the **coracidia** develop into **procercoid larvae 4.** Following ingestion of the **copepod** by a suitable **second intermediate host**, typically **minnows and other small freshwater fish**, the **procercoid** larvae are released from the **crustacean** and migrate into **the fish flesh** where they develop into a **plerocercoid larvae (sparganum) 5.** The **plerocercoid larvae** are the **infective stage for humans.** Because **humans** do not generally eat undercooked minnows and similar small freshwater fish, these do not represent an important source of infection. Nevertheless, these **small second intermediate hosts** can be eaten by larger **predator species, e.g., trout, perch, walleyed pike 6.**

In this case, the **sparganum** can migrate to the **musculature of the larger predator fish** and humans can acquire the disease by eating these later intermediate infected host **fish raw or undercooked 7.** After ingestion of the infected fish, the **plerocercoid** develop into **immature adults** and then into **mature adult tapeworms** which will reside in the **small intestine**. **The adults of *D. latum* attach to the intestinal mucosa** by means of the two **bilateral groves (bothria) of their scolex 8.** The adults can reach more than 10 m in length, with more than **3,000 proglottids**. **Immature eggs** are discharged from the **proglottids (up to 1,000,000 eggs per day per worm) 9.** and are passed in the **feces 1. Eggs** appear in the feces 5 to 6 weeks after infection. In addition to humans, many other mammals can also serve as definitive hosts for ***D. latum*.** CDC DPDx Parasite Image Library

Symptoms

Clinical symptoms may be mild, depending on the number of worms. They include abdominal discomfort, loss of weight, loss of appetite and some malnutrition. Anemia and neurological problems associated with vitamin B12 deficiency are seen in heavily infected individuals.

Diagnosis

Diagnosis is based on finding many typical eggs and empty proglottids in feces. A history of raw fish consumption and residence in an endemic locality is helpful.

Treatment and control

Praziquantel is the drug of choice. Freezing for 24 hours, thorough cooking or pickling of fish kills the larvae. Fish reservoirs should be kept free of raw sewage.

ECHINOCOCCOSIS (HYDATID)

Echinococcus granulosus and *E. multilocularis* are causative agents of hydatid cysts.

ECHINOCOCCUS GRANULOSUS

Epidemiology

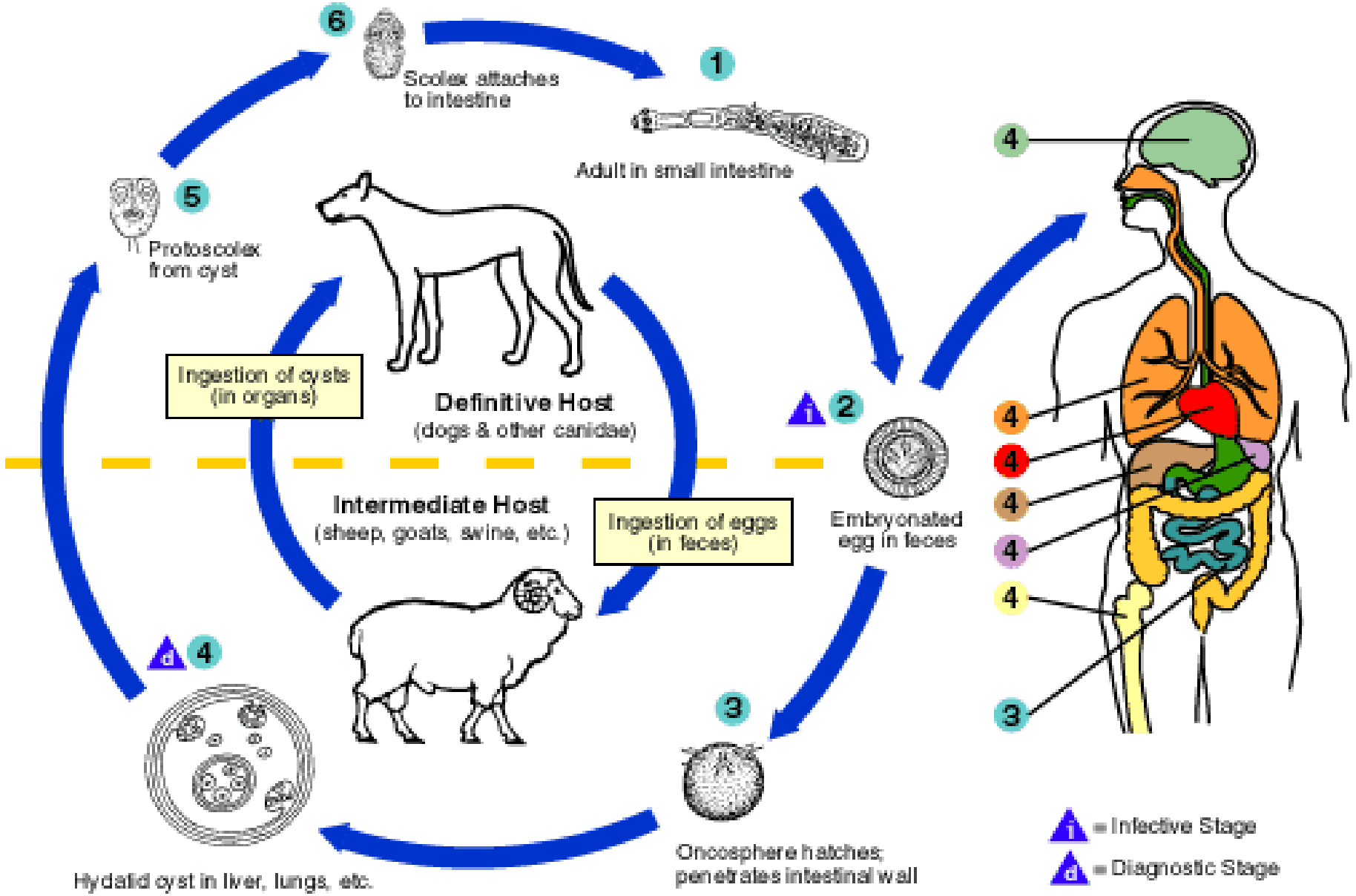
The organism is common in Asia, Australia, Eastern Africa, southern Spain, southern parts of South America and northern parts of North America. The incidence of human infection about 1 to 2 per 1000 population and may be higher in rural areas of affected regions.

Morphology

This is the smallest of all tapeworms (3 to 9 mm long) with only 3 proglottids.

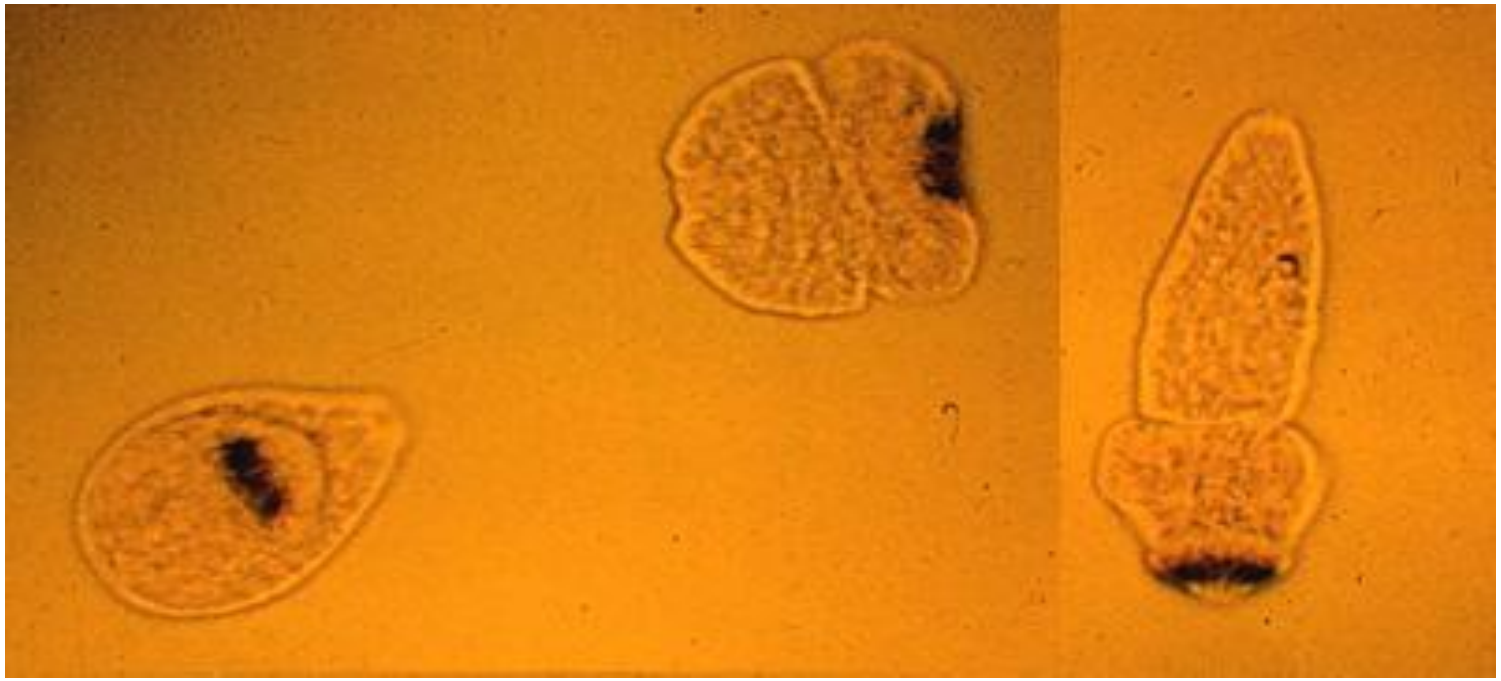
Life cycle

The adult worm lives in **domestic and wild carnivorous animals**. **Eggs**, passed by infected animals, are ingested by the **grazing farm animals or man**, localize in different organs and develop into **hydatid cysts** containing many larvae (**proto-scolices or hydatid sand**). When other animals consume infected organs of these animals, **proto-scolices** escape the cyst, enter the **small intestine** and develop into **adult worms**. **Echinococcus eggs**, when swallowed by **man**, produce **embryos** that penetrate the small intestine, enter the circulation and form **cysts in liver, lung, bones, and sometimes, brain**. The **cyst** is round and measures 1 to 7 cm in diameter, although it may grow to be 30 cm. The **cyst** consists of an **outer unclear hyaline cuticle and an inner nucleated germinal layer containing clear yellow fluid**. **Daughter cysts attach to the germinal layer**, although some cysts, known as **brood cysts**, may have only larvae (**hydatid sand**). **Man is a dead end host.**

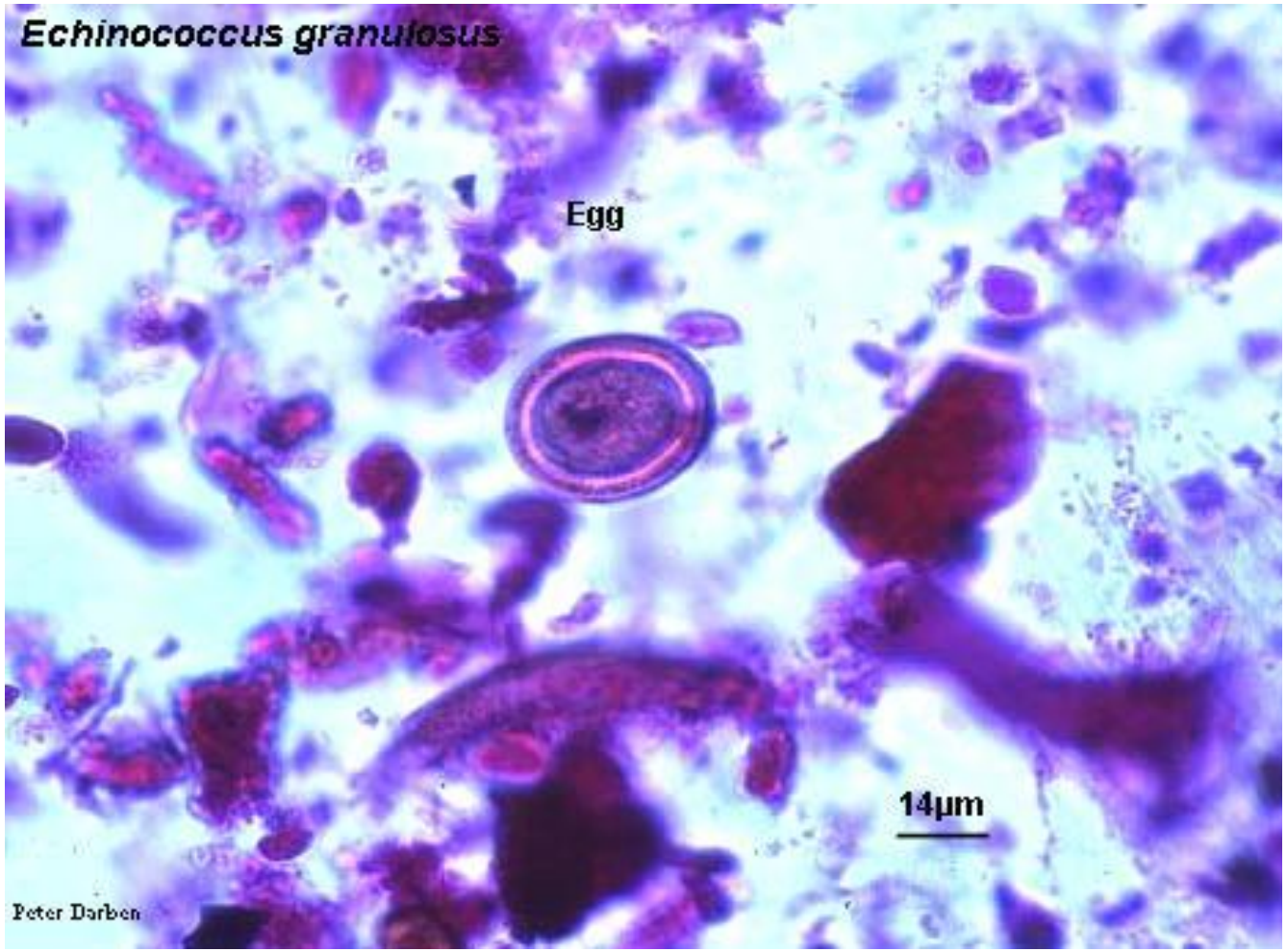


Life cycle of *Echinococcus granulosus*

The adult **Echinococcus granulosus** (3 to 6 mm long) **(1)** resides in **the small bowel of the definitive hosts**, **dogs or other canids. Gravid proglottids** release **eggs (2)** that are passed in the **feces**. After ingestion by a suitable **intermediate host** (under natural conditions: sheep, goat, swine, cattle, horses, camel), the **egg** hatches in the **small bowel** and releases an **oncosphere (3)** that penetrates the **intestinal wall** and migrates through the **circulatory system** into various organs, **especially the liver and lungs**. In these organs, the **oncosphere** develops into a **cyst (4)** that enlarges gradually, producing **protoscolices and daughter cysts** that fill the cyst interior. **The definitive host** becomes infected by ingesting the **cyst-containing organs of the infected intermediate host**. After ingestion, **the protoscolices (5) evaginate**, attach to the **intestinal mucosa (6)**, and develop into **adult stages (1)** in 32 to 80 days. The same life cycle occurs with ***E. multilocularis*** (1.2 to 3.7 mm), with certain differences.

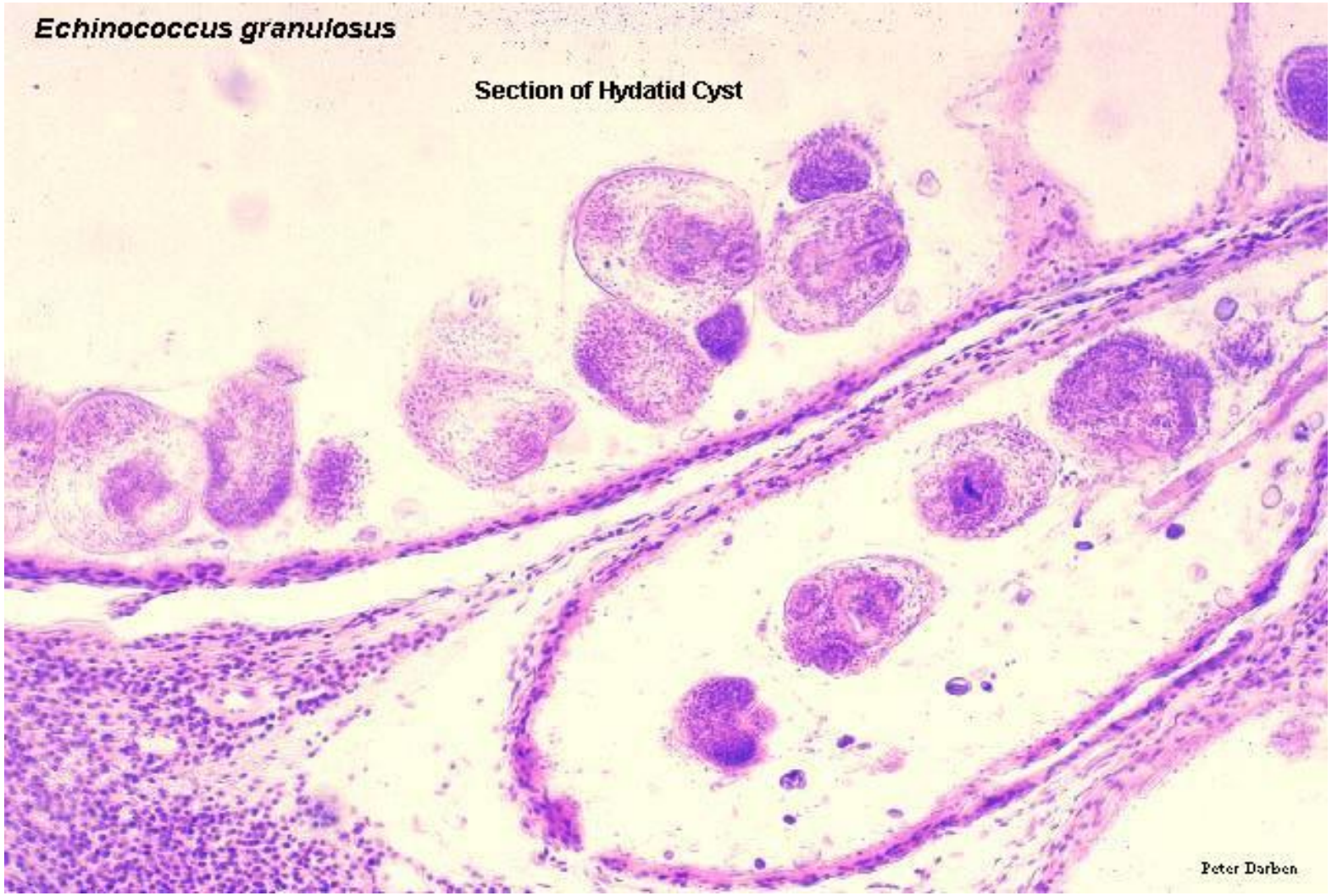


Hydatid sand". Fluid aspirated from a hydatid cyst will shows multiple **protoscolices** (size approximately 100 µm), each of which has typical **hooklets**. The protoscolices are normally invaginated (left), and evaginate (middle, then right) when put in saline.

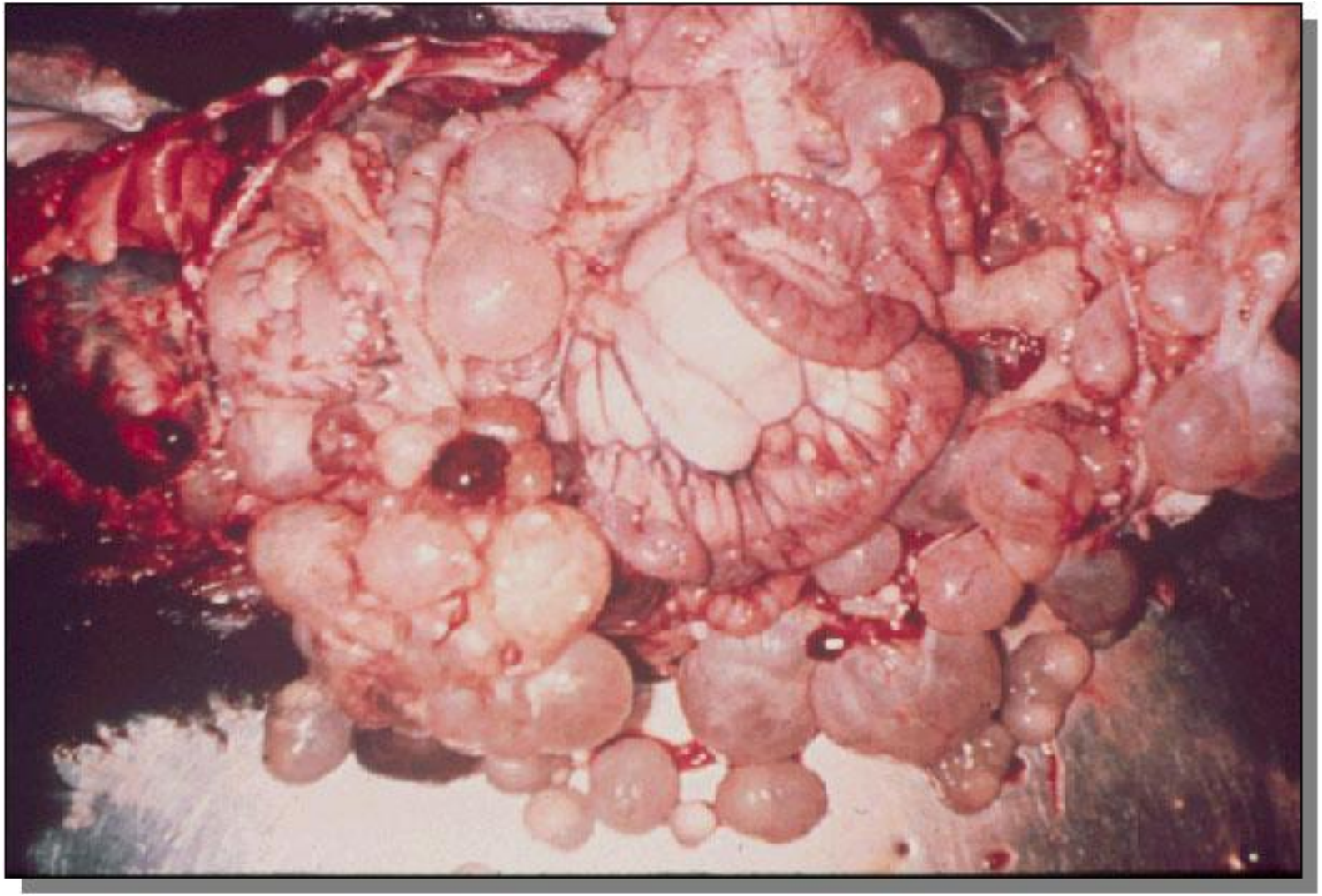


***Echinococcus granulosus* egg.**

***Echinococcus granulosus* adult.**



***Echinococcus granulosus* hydatid cysts in section of lung (H&E).**



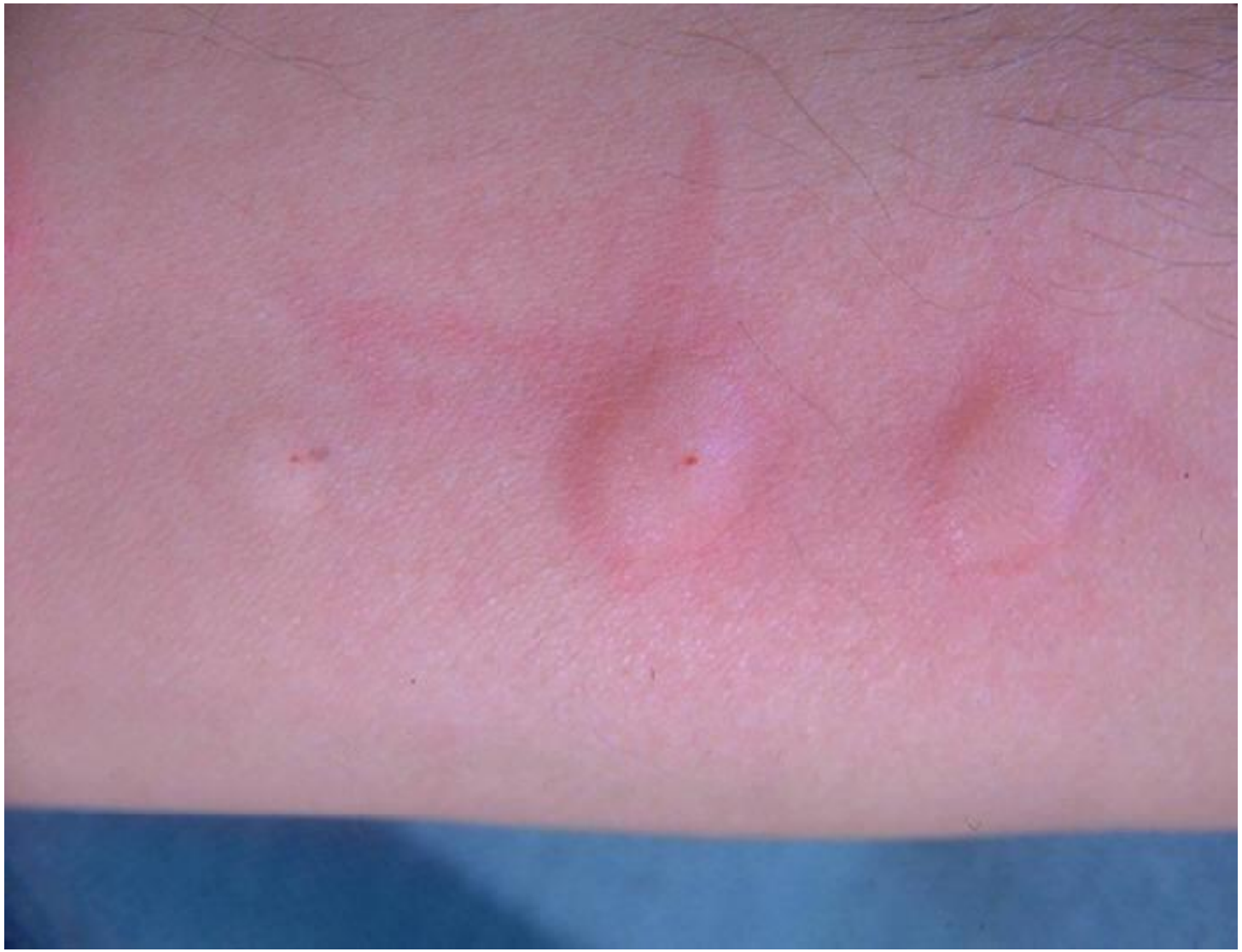
Hydatid cysts

Symptoms

The symptoms, comparable to those of a slowly growing tumor, depend upon the location of the cyst. Large abdominal cysts produce increasing discomfort. Liver cysts cause obstructive jaundice. Peribronchial cysts may produce pulmonary abscesses. Brain cysts produce intracranial pressure and Jacksonian epilepsy. Kidney cysts cause renal dysfunction. The contents of a cyst may produce anaphylactic responses.

Diagnosis

Clinical symptoms of a slow-growing tumor accompanied by eosinophilia are suggestive. Intradermal (Casoni) test with hydatid fluid is useful. Pulmonary cysts and calcified cysts can be visualized using x-rays. Antibodies against hydatid fluid antigens have been detected in a sizable population of infected individuals by ELISA or indirect hemagglutination test.



Man's arm showing positive skin test for hydatid disease (echinococcosis) CDC

Treatment and control

Treatment involves surgical removal of cyst or inactivation of hydatid sand by injecting the cyst with 10% formalin and its removal within few (4-5) minutes. Prazequantel has been shown to be effective in many cases. Albendazole, in high doses, is an alternative. Preventive measures involve avoiding contact with infected dogs and cats and elimination of their infection.

ECHINOCOCCUS MULTILOCULARIS

This is a tapeworm, similar to *E. granulosus*, that also causes hydatid in northern parts of Asia and North America. It has a very similar morphology and life cycle except that **rodents are its intermediate host**. **Humans,** when infected with this worm, also **develop hydatid cysts** which produce symptoms similar to those caused by ***E. granulosus*.** However, **the cysts are multilocular (many chambers)**. The organism is **resistant to praziquantel;** high doses of **Albendazole has some anti-parasitic effect. Surgery** is the means of removing the cyst. **Rodent control** is the means of prevention.

Summary				
Organism	Transmission	Symptoms	Diagnosis	Treatment
Tenia saginata	Cyst in beef	Epigastric pain, vomiting, diarrhea	Proglottids or eggs in stool or perianal area	Praziquantel
Tenia solium	Cyst in pork	Epigastric pain, vomiting, diarrhea	Proglottids or eggs in stool or perianal area	Praziquantel
T. solium Cysticercosis	Oro-fecal	Muscle pain and weakness, ocular and neurologic problems	Roentgenography, anti-cysticercal antibody (EIA)	Praziquantel
D. latum	Cyst in fish	Abdominal pain, loss of weight, anorexia, malnutrition and B12 deficiency problems	Proglottids or eggs in stool or perianal area	Praziquantel
E. granulosus	Oro-fecal	Large cysts produce various symptoms depending on the location of the organism.	Roentgenography, anti-hydatid fluid antibody (EIA), Casoni skin test	Surgery, formalin injection and drainage, Praziquantel
E. multiloculoris	Oro-fecal	As above	As above	Surgery, Albendazole

Parasitology
Helminthes/NEMATODES
Intestinal and Tissue Nematodes

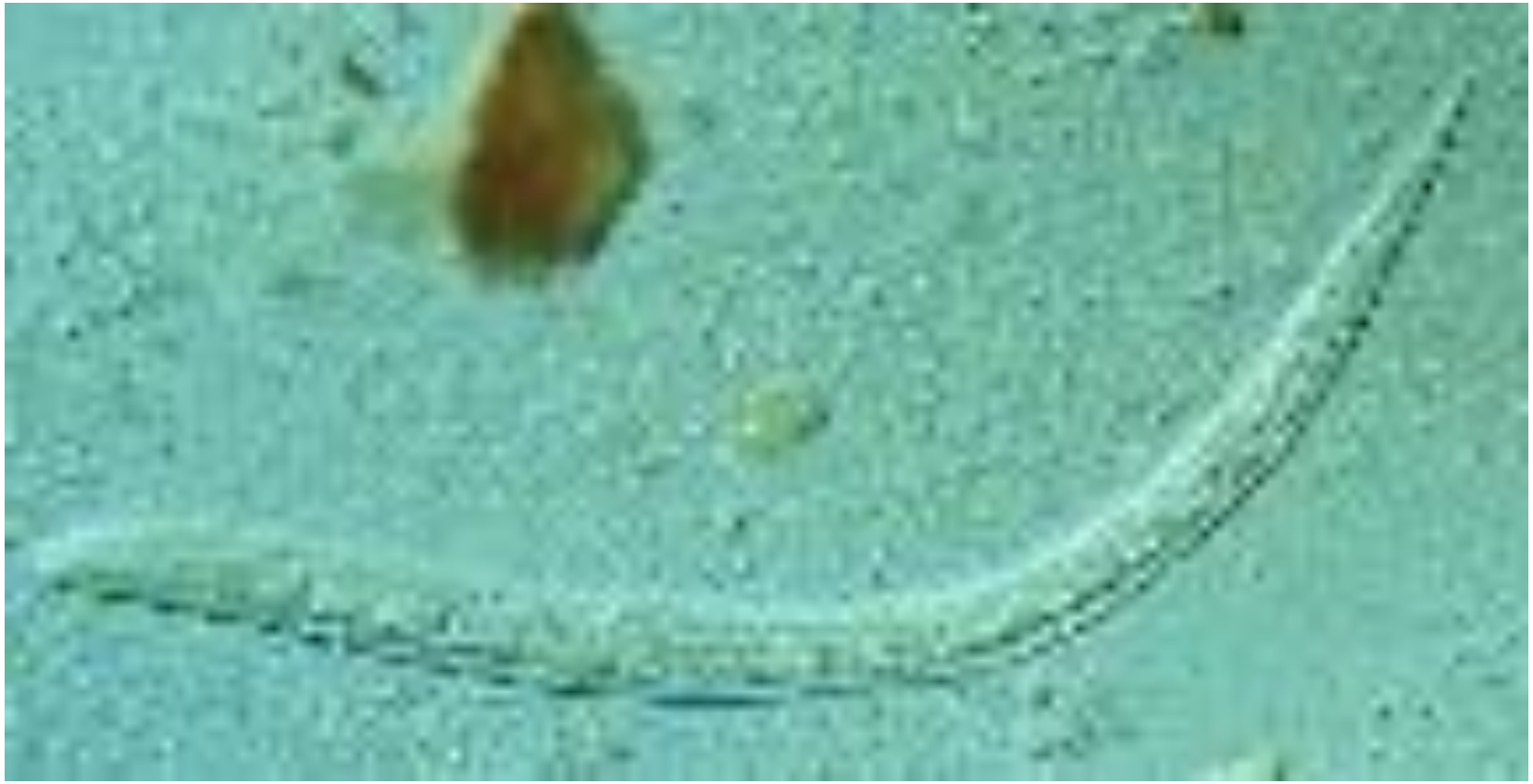
Strongyloides stercoralis (Threadworm)

Epidemiology

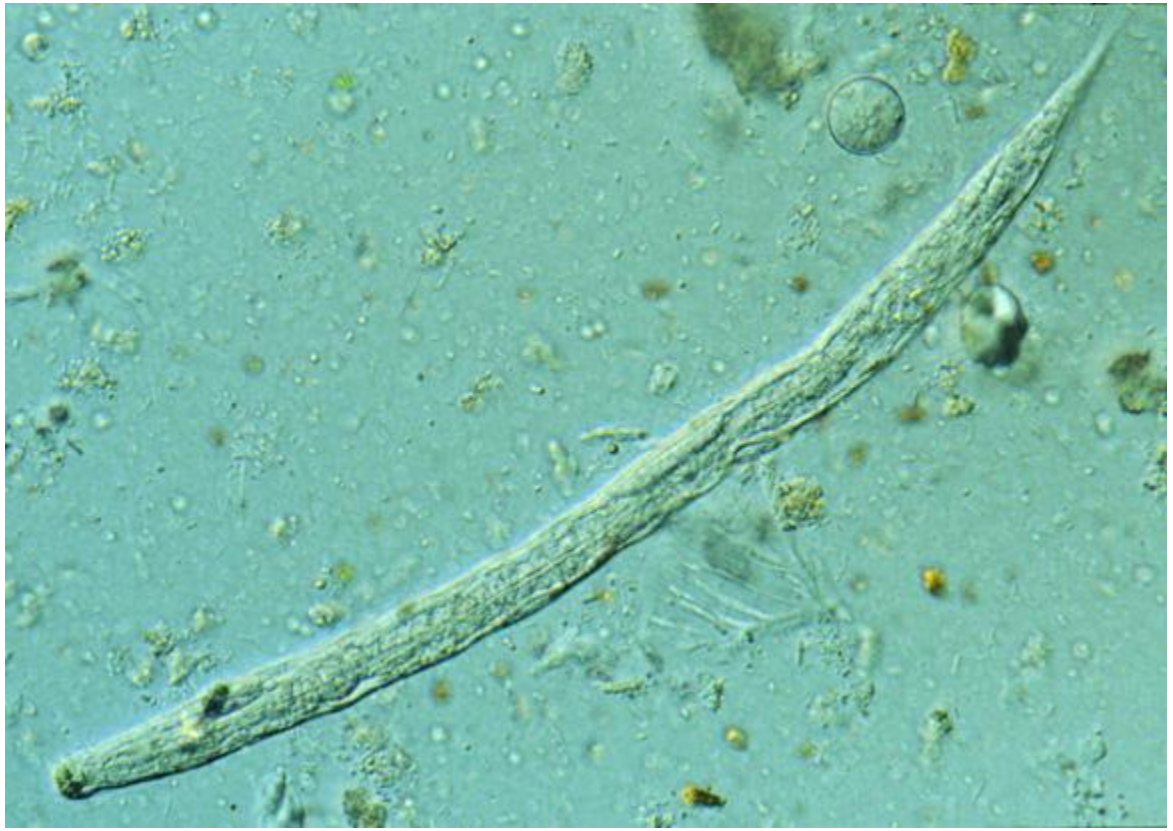
Threadworm infection, also known as **Cochin-China diarrhea,** estimated at 50 to 100 million cases worldwide, is an infection of the tropical and subtropical areas with poor sanitation. In the United States, it is prevalent in the South and among Puerto Ricans.

Morphology

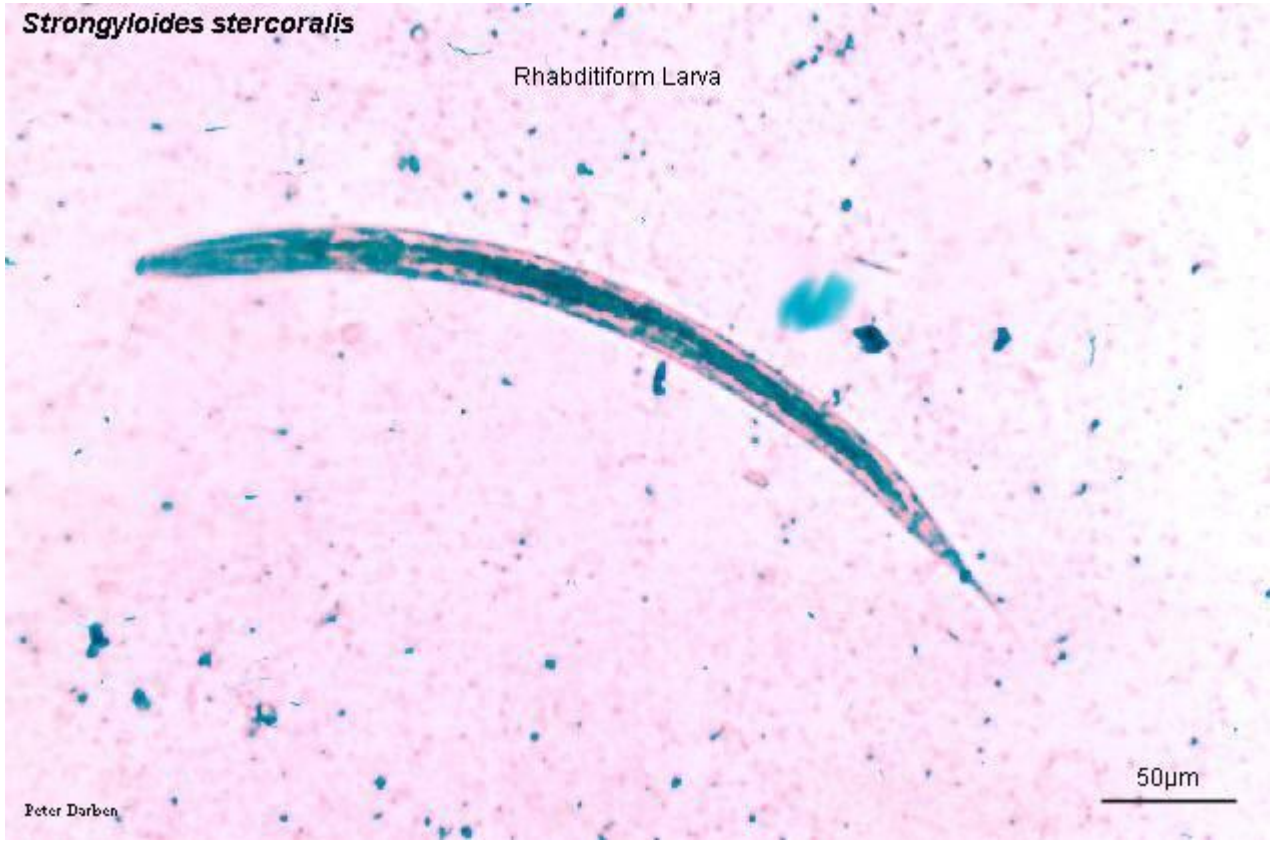
The size and shape of threadworm varies depending on whether it is parasitic or free-living. The parasitic female is larger (2.2 mm x 45 micrometers) than the free-living worm (1 mm x 60 micrometers). The eggs, when laid are 55 micrometers by 30 micrometers.



Strongyloides stercoralis The esophageal structure is clearly visible in this larva; it consists of a club-shaped anterior portion; a post-median constriction; and a posterior bulbus



Strongyloides stercoralis Note the prominent genital primordium in the mid-section of the larva; note also the Entamoeba coli cyst near the tail of the larva.



Strongyloides stercoralis rhabditiform larva

Life cycle

The **infective larvae** of ***S. stercoralis*** penetrate the **skin** of man, enter the **venous circulation** and pass through the **right heart** to **lungs**, where they penetrate into the **alveoli**. From there, the adolescent parasites ascend to the **glottis**, are swallowed, and reach the **upper part** of the **small intestine**, where they develop into **adults**.

Ovipositing females develop in 28 days from infection.

The **eggs** in the **intestinal mucosa**, hatch and develop into rhabditiform larvae in man.

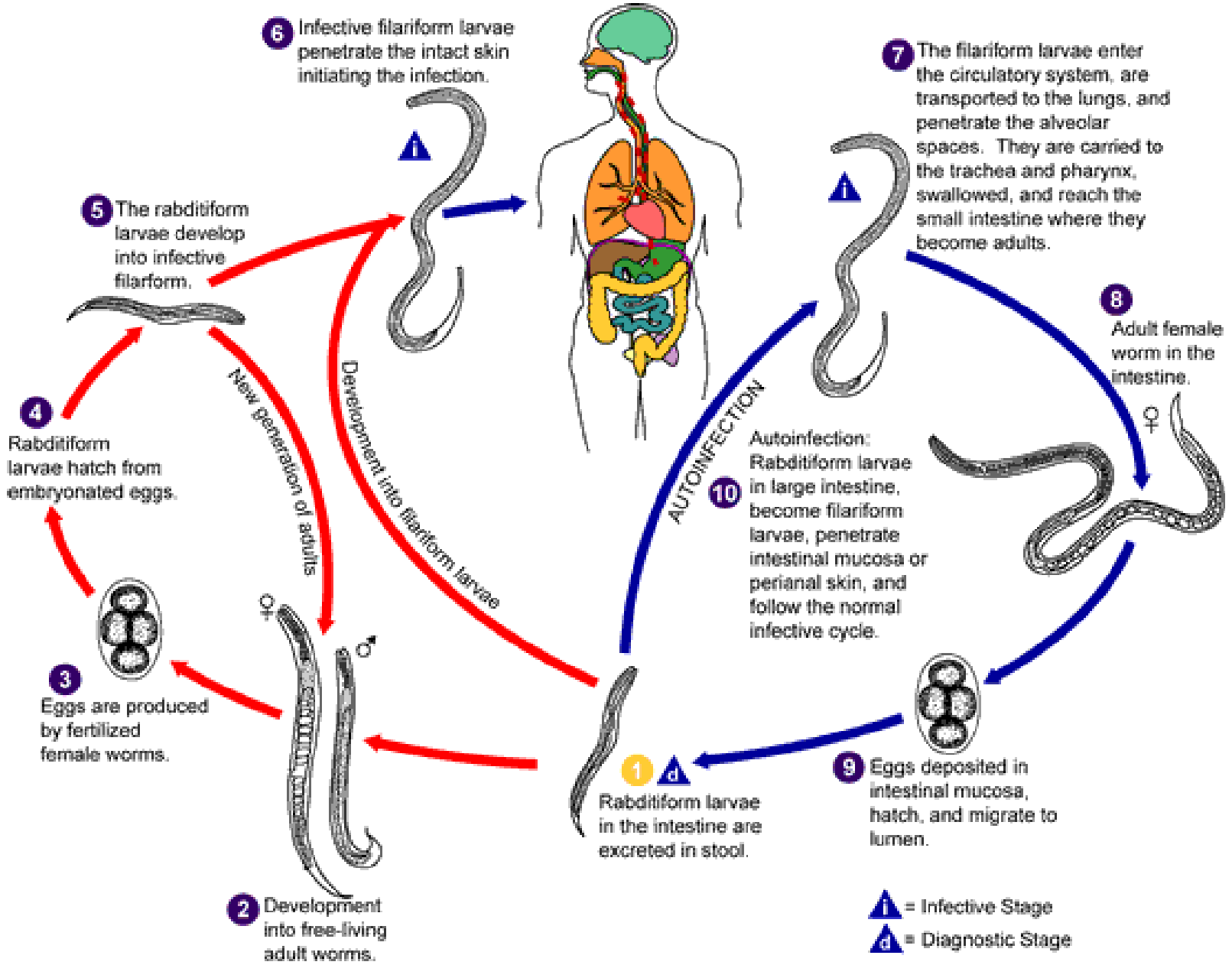
These larvae can penetrate through the **mucosa** and cycle back into the **blood circulation**, **lung**, **glottis** and **duodenum** and **jejunum**; thus they continue the auto infection cycle.

Alternatively, they are passed in the **feces**, develop into **infective filariform larvae** and enter another host

to complete the **direct cycle.**

If no suitable host is found, the **larvae** mature into **free-living worm** and lay **eggs** in the **soil**.

The **eggs** hatch in the soil and produce **rhabditiform larvae** which develop into **infective filariform larvae** and enter a new host **(indirect cycle),** or mature into **adult worms** to **repeat** the **free-living cycle.**


6 Infective filariform larvae penetrate the intact skin initiating the infection.
7 The filariform larvae enter the circulatory system, are transported to the lungs, and penetrate the alveolar spaces. They are carried to the trachea and pharynx, swallowed, and reach the small intestine where they become adults.
8 Adult female worm in the intestine.
♀
5 The rabditiform larvae develop into infective filarform.
4 Rabditiform larvae hatch from embryonated eggs.
New generation of adults
Development into filariform larvae
AUTOINFECTION
10 Autoinfection: Rabditiform larvae in large intestine, become filariform larvae, penetrate intestinal mucosa or perianal skin, and follow the normal infective cycle.
3 Eggs are produced by fertilized female worms.
♀
♂
9 Eggs deposited in intestinal mucosa, hatch, and migrate to lumen.
1 d Rabditiform larvae in the intestine are excreted in stool.
2 Development into free-living adult worms.
i = Infective Stage
d = Diagnostic Stage

The ***Strongyloides*** **life cycle** is complex among helminths with its alternation between free-living and parasitic cycles, and its potential for autoinfection and multiplication within the host. **Two types of cycles** exist:

Free-living cycle: The **rhabditiform larvae** passed in the stool **(1)** (see "Parasitic cycle" below) can either molt twice and become infective **filariform larvae** (**direct development**) **(6)** or **molt four times** and become free living **adult males and females (2)** that mate and produce **eggs (3)** from which **rhabditiform larvae** hatch **(4).** The latter in turn can either develop **(5)** into a new generation of free-living **adults** (as represented in **(2)**), or into infective **filariform larvae (6)**. The **filariform larvae** penetrate the human host skin to initiate the parasitic cycle **(6).**

Parasitic cycle: Filariform larvae in **contaminated soil** penetrate the **human skin (6),** and are transported to the **lungs** where they penetrate the **alveolar spaces**; they are **carried** through **the bronchial tree** to the **pharynx**, are **swallowed** and then reach **the small intestine (7).**

In the **small intestine** they **molt twice** and become **adult female worms (8).**

The **females** live threaded in the **epithelium of the small intestine** and by **parthenogenesis** produce **eggs (9),** which yield **rhabditiform larvae.** The **rhabditiform larvae** can either be passed in the **stool (1)** (**see "Free-living cycle" above**), or can cause **autoinfection (10).**

In autoinfection, the **rhabditiform larvae** become infective **filariform larvae**, which can penetrate either the **intestinal mucosa** (**internal autoinfection**) or the **skin of the perianal area** (**external autoinfection**); in either case, the **filariform larvae** may follow the previously described route, being carried successively to the **lungs**, the **bronchial tree**, the **pharynx**, and the **small intestine** where they mature into **adults**; or they may disseminate widely in the body.

To date, occurrence of autoinfection in humans with helminthic infections is recognized only in ***Strongyloides stercoralis*** and ***Capillaria philippinensis*** infections.

In the case of ***Strongyloides***, **autoinfection** may explain the possibility of persistent infections for many years in persons who have not been in an endemic area and of **hyperinfections** in immunodepressed individuals.

Symptoms

Light infections are asymptomatic. Skin penetration causes **itching** and **red blotches**. During migration, the organisms cause **bronchial verminous pneumonia** and, in the **duodenum**, they cause a **burning mid-epigastric pain** and **tenderness** accompanied by **nausea** and **vomiting**. **Diarrhea** and **constipation** may alternate. **Heavy**, **chronic** infections result in **anemia**, **weight loss** and **chronic bloody dysentery**. Secondary **bacterial infection** of **damaged mucosa** may produce **serious complications**.

Diagnosis

The presence of free **rhabditiform larvae** in the **feces** is diagnostic. **Culture of stool** for 24 hours will produce **filariform larvae**.

Treatment and control

Ivermectin or thiabendozole can be used effectively. Direct and indirect infections are controlled by **improved hygiene** and **auto-infection is controlled by chemotherapy.**

Dracunculus medinensis (Guinea worm; Fiery serpent)

Dracunculiasis comes from the Latin: *affliction with little dragons*. The common name **"Guinea worm"** results from the first observation of this parasite by Europeans in the Guinea coast of West Africa in the 17th century. Infection causes a burning, painful sensation leading to the disease being called ***the fiery serpent.***

Epidemiology

There have been dramatic efforts to eradicate *Dracunculus*. CDC estimated that in 1986 there were 3.5 million cases worldwide. However, at the end of 2007, there were fewer than 10,000 reported cases in five nations in Africa: Sudan, Ghana, Nigeria, Niger, and Mali, and as of June 2008, cases had been reduced by more than 50

percent compared to the same period of 2007. Guinea worm disease is expected to be the next disease after smallpox to be eradicated and in 2016 there were probably as few as 25 cases worldwide. There are three counties in which the disease is still found: Chad, Ethiopia and South Sudan.

Morphology

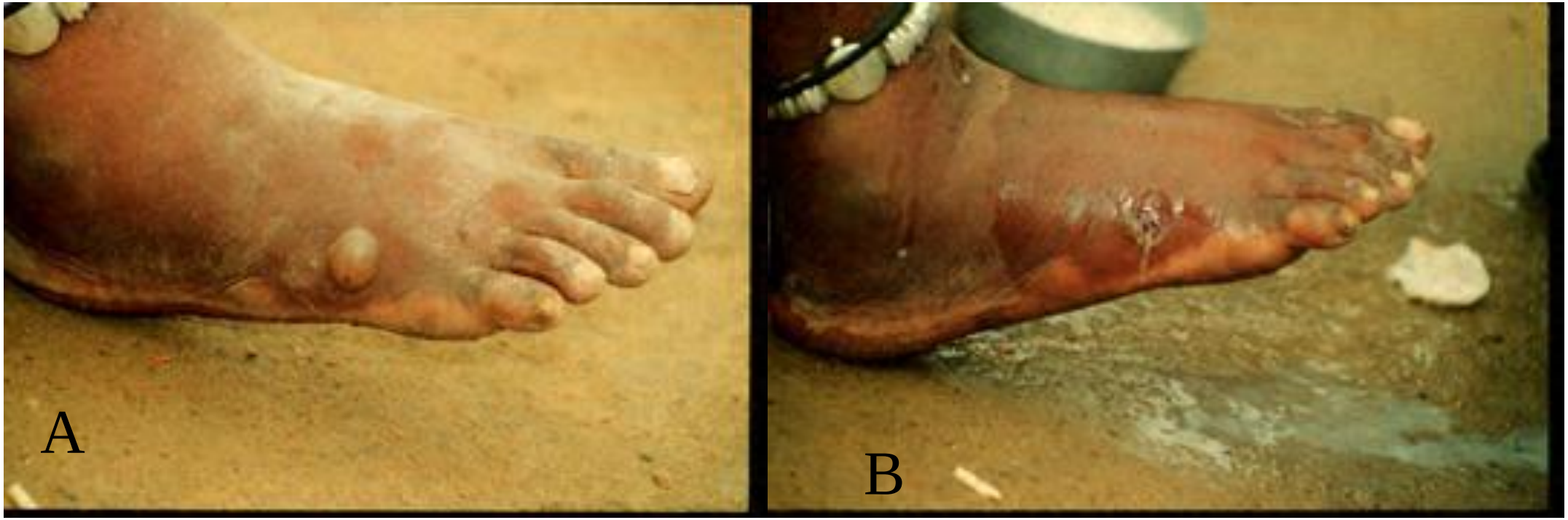
The adult female worm measures 50-120 cm by 1 mm and the male is half that size.

Life cycle

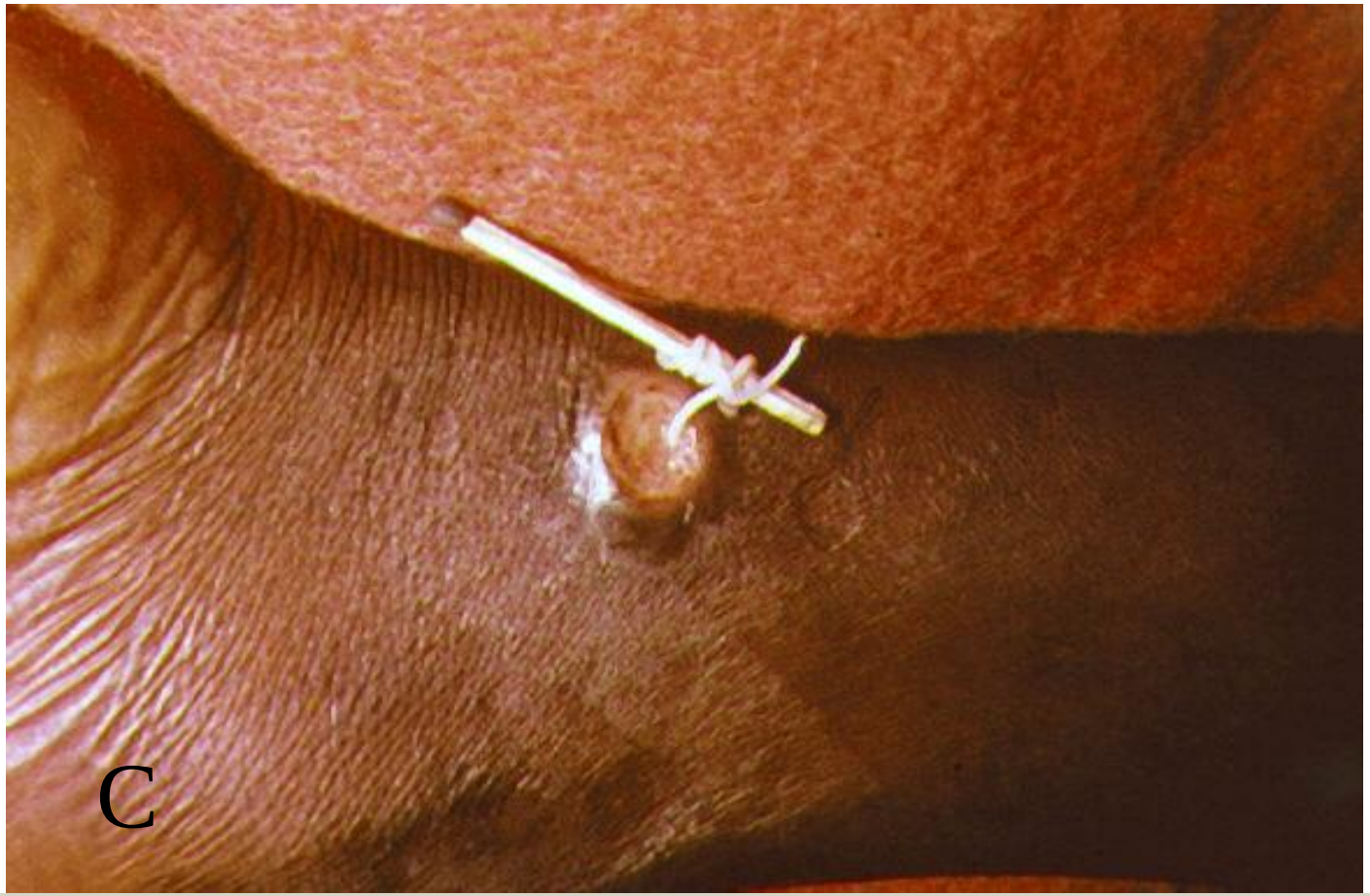
The infection is caused by ingestion of **water** contaminated with **water fleas (Cyclops)** infected with **larvae.**

The **rhabtidiform larvae** penetrate the human **digestive tract wall**, lodge in the **loose connective tissues** and mature into the **adult form** in 10 to 12 weeks.

In about a year, the **gravid female** migrates to the **subcutaneous tissue** of organs that normally come **in contact with water** and discharges its **larvae** into the **water**. The larvae are picked up by **Cyclops**, in which they develop into **infective form** in 2 to 3 weeks.



female guinea worm induces a painful blister (A); after rupture of the blister, the worm emerges as a whitish filament (B) in the center of a painful ulcer which is often secondarily infected. (Images contributed by Global 2000/The Carter Center, Atlanta, Georgia).
CDC



Dracunculus medinensis worm wound around matchstick. This helminth is gradually withdrawn from the body by winding the stick
CDC

Life cycle

The infection is caused by ingestion of water contaminated with **water fleas (Cyclops)** infected with larvae. The **rhabtidiform larvae** penetrate the human **digestive tract wall**, lodge in the **loose connective tissues** and mature into the **adult form** in 10 to 12 weeks.

In about a year, the **gravid female** migrates to the **subcutaneous tissue** of organs that normally come in contact with water and discharges its **larvae** into the water.

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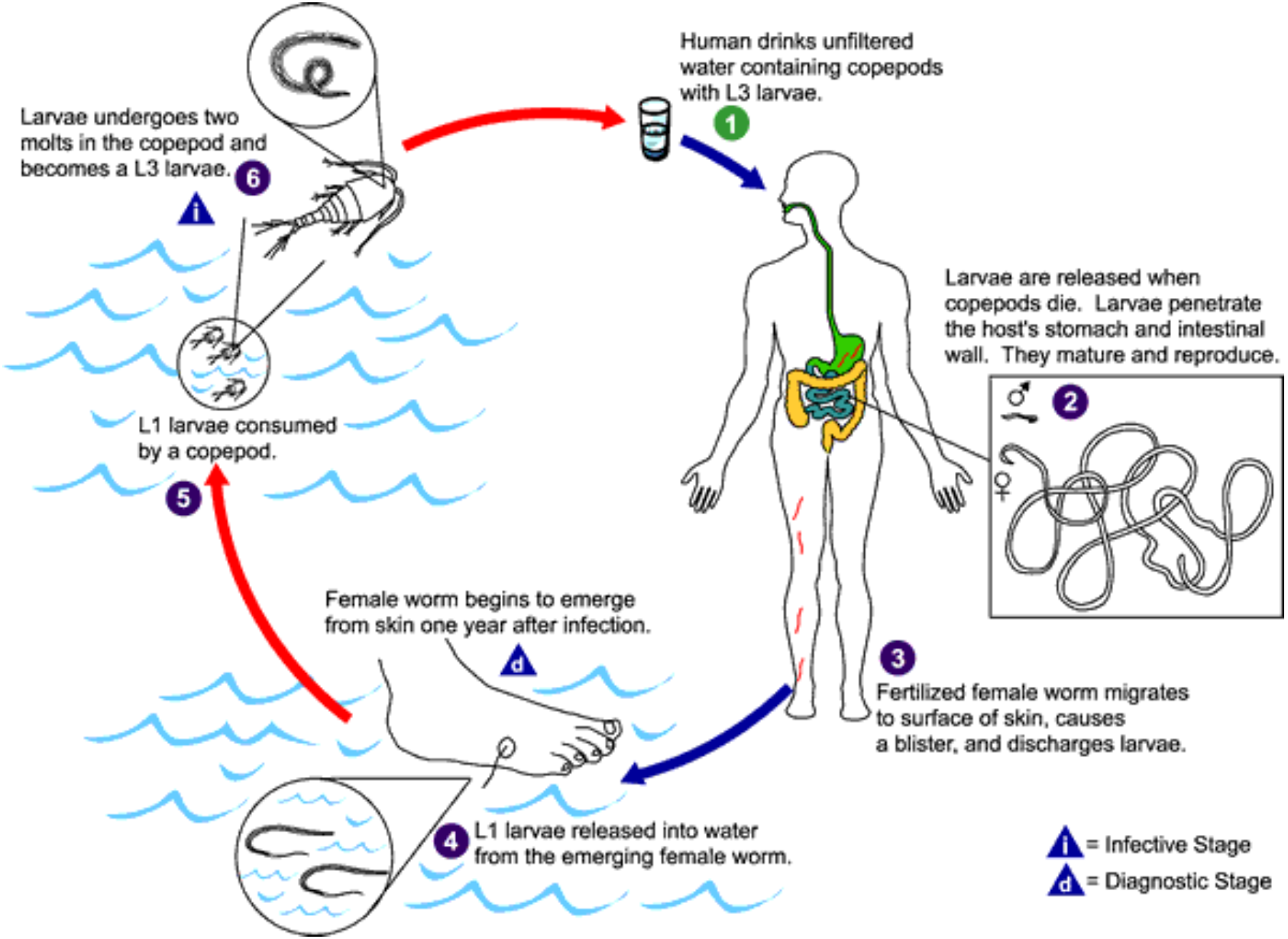

Human drinks unfiltered water containing copepods with L3 larvae.
1
Larvae are released when copepods die. Larvae penetrate the host's stomach and intestinal wall. They mature and reproduce.
2
Fertilized female worm migrates to surface of skin, causes a blister, and discharges larvae.
3
L1 larvae released into water from the emerging female worm.
4
L1 larvae consumed by a copepod.
5
Larvae undergoes two molts in the copepod and becomes a L3 larvae.
6
Female worm begins to emerge from skin one year after infection.
i = Infective Stage
d = Diagnostic Stage

Humans become infected by drinking unfiltered water containing **copepods (small crustaceans)** which are infected with **larvae of *D. medinensis* 1.** Following ingestion, the copepods die and release the **larvae**, which penetrate the host **stomach** and **intestinal wall** and enter the **abdominal cavity** and **retroperitoneal space 2.** After maturation into **adults** and copulation, the male worms die and the **females** (length: 70 to 120 cm) migrate in the **subcutaneous tissues** towards the **skin surface 3.** Approximately one year after infection, the **female** worm induces a **blister** on the **skin**, generally on the distal **lower extremity**, which ruptures. When this lesion comes into contact with water, a contact that the patient seeks to relieve the local discomfort, the **female worm** emerges and releases **larvae 4.**

The larvae are ingested by a copepod **5.** and after two weeks (and two molts) have developed into infective larvae **6.** Ingestion of the copepods closes the cycle **1.** CDC DPDx Parasite Image Library

Symptoms

If the worm does not reach the skin, it dies and causes little reaction. In superficial tissue, it liberates a toxic substance that produces a **local inflammatory reaction in** the form of a **sterile blister** with serous exudation. The worm lies in a **subcutaneous tunnel** with its posterior end **beneath the blister**, which contains **clear yellow fluid**. The course of the **tunnel** is marked with induration and **edema**. Contamination of the **blister** produces **abscesses**, cellulitis, **extensive ulceration** and **necrosis.**

Diagnosis

Diagnosis is made from the **local blister, worm or larvae.** The outline of the worm under the skin may be revealed by reflected light.

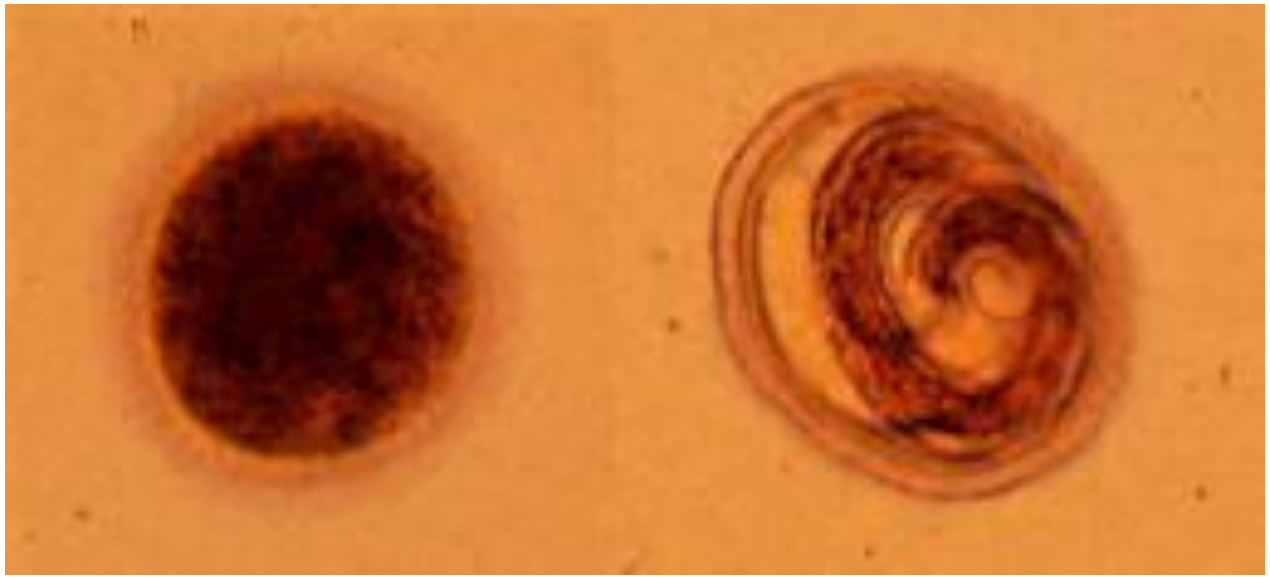
Treatment

Treatment includes the **extraction of the adult guinea worm** by rolling it a few centimeters per day or preferably by **multiple surgical incisions under local anaesthesia**. **No drug** is effective at killing the worm and there is **no vaccine**. Protection of drinking **water** from being **contaminated** with **Cyclops and larvae** are effective preventive measures and these have led to a dramatic decline in the incidence of guinea worm infections.

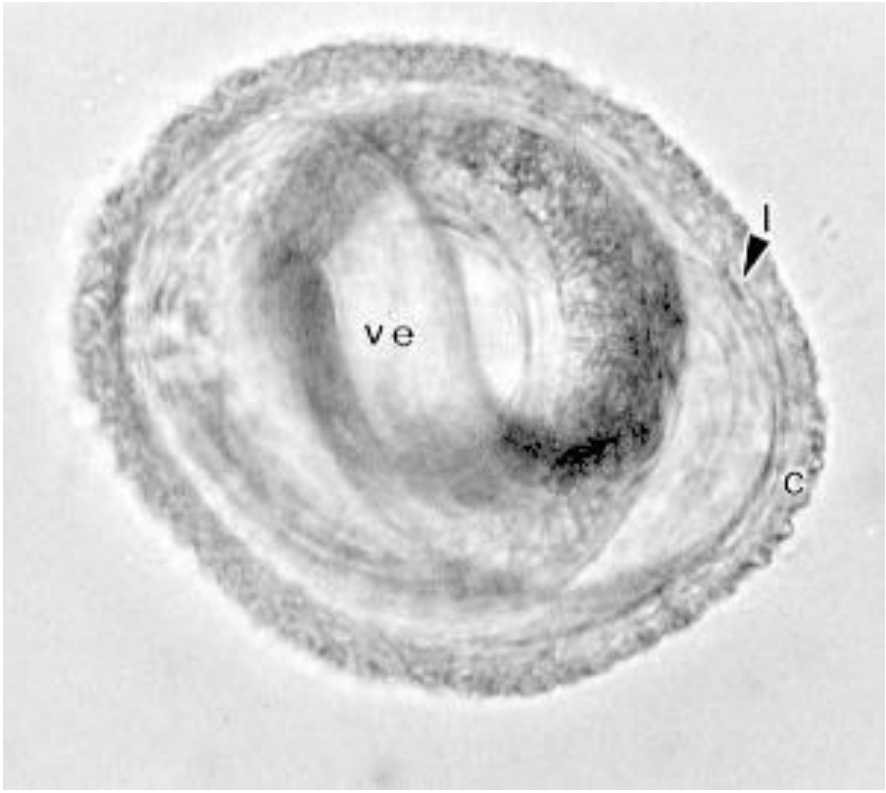
Toxocara canis and *T. catti* (visceral larva migrans)

These are **roundworms of dogs** and **cats** but they can infect **humans** and cause damage of the **visceral organs**. **Eggs** from **feces** of infected animals are swallowed by man and **hatch** in the **intestine**. The **larvae** penetrate the **mucosa**, enter the **circulation** and are carried to **liver**, **lungs**, **eyes** and **other organs** where they cause **inflammatory necrosis**. Symptoms are due to the inflammatory reaction at the site of infection.

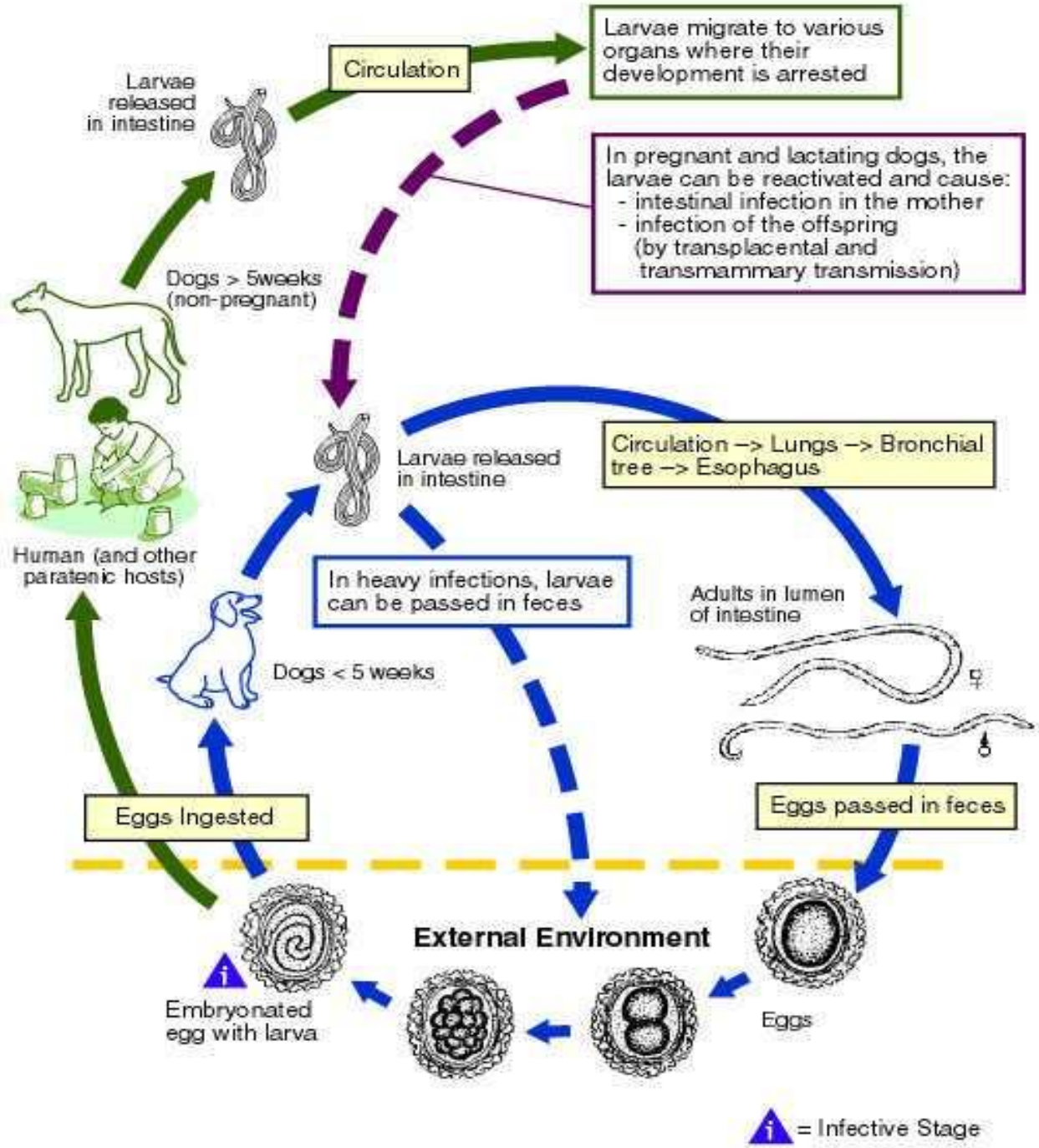
The most serious consequence of infection may be **loss of sight** if the worm localizes in the **eye**. Treatment includes **Mebendazole** to eliminate the **worm** and **prednisone** for **inflammatory symptoms**. Avoidance of infected dogs and cats is the best prevention.



Eggs of *Toxocara canis*. These **eggs** are passed in **dog feces**, especially **puppies' feces**. Humans do not produce or excrete eggs, and therefore **eggs** are not a diagnostic finding in human toxocariasis! The egg to the left is fertilized but not yet embryonated, while the egg to the right contains a well developed larva. The latter egg would be infective if ingested by a human (frequently, a child).



Toxocara canis (Dog Roundworm) egg, embryonated


Larvae migrate to various organs where their development is arrested
Circulation
Larvae released in intestine
In pregnant and lactating dogs, the larvae can be reactivated and cause:
- intestinal infection in the mother
- infection of the offspring (by transplacental and transmammary transmission)
Dogs > 5weeks (non-pregnant)
Larvae released in intestine
Circulation –> Lungs –> Bronchial tree –> Esophagus
Human (and other paratenic hosts)
In heavy infections, larvae can be passed in feces
Adults in lumen of intestine
Dogs < 5 weeks
Eggs Ingested
Eggs passed in feces
External Environment
Embryonated egg with larva
Eggs
= Infective Stage

Toxocara Life Cycle

Toxocara canis accomplishes its life cycle in **dogs**, with **humans** acquiring the infection as **accidental hosts**. Following ingestion by **dogs**, the infective **eggs** yield **larvae** that penetrate the **gut wall** and migrate into **various tissues**, where they **encyst** if the dog is older than 5 weeks. In younger dogs, the **larvae** migrate through the **lungs**, **bronchial tree**, and **esophagus**; **adult worms** develop and **oviposit** in the **small intestine**. In the older dogs, the **encysted stages** are **reactivated** during **pregnancy**, and infect by the **transplacental** and **transmammary** routes the puppies, in whose **small intestine adult worms** become established. Thus, **infective eggs** are excreted by lactating bitches and puppies.

Humans are **paratenic hosts** who become infected by **ingesting infective eggs in contaminated soil**. After ingestion, the **eggs** yield **larvae** that penetrate the **intestinal wall** and are carried by the **circulation** to a **wide variety of tissues** (**liver, heart, lungs, brain, muscle, eyes**). While the **larvae do not undergo any further development** in these sites, they can cause **severe local reactions** that are the basis of **toxocariasis.**