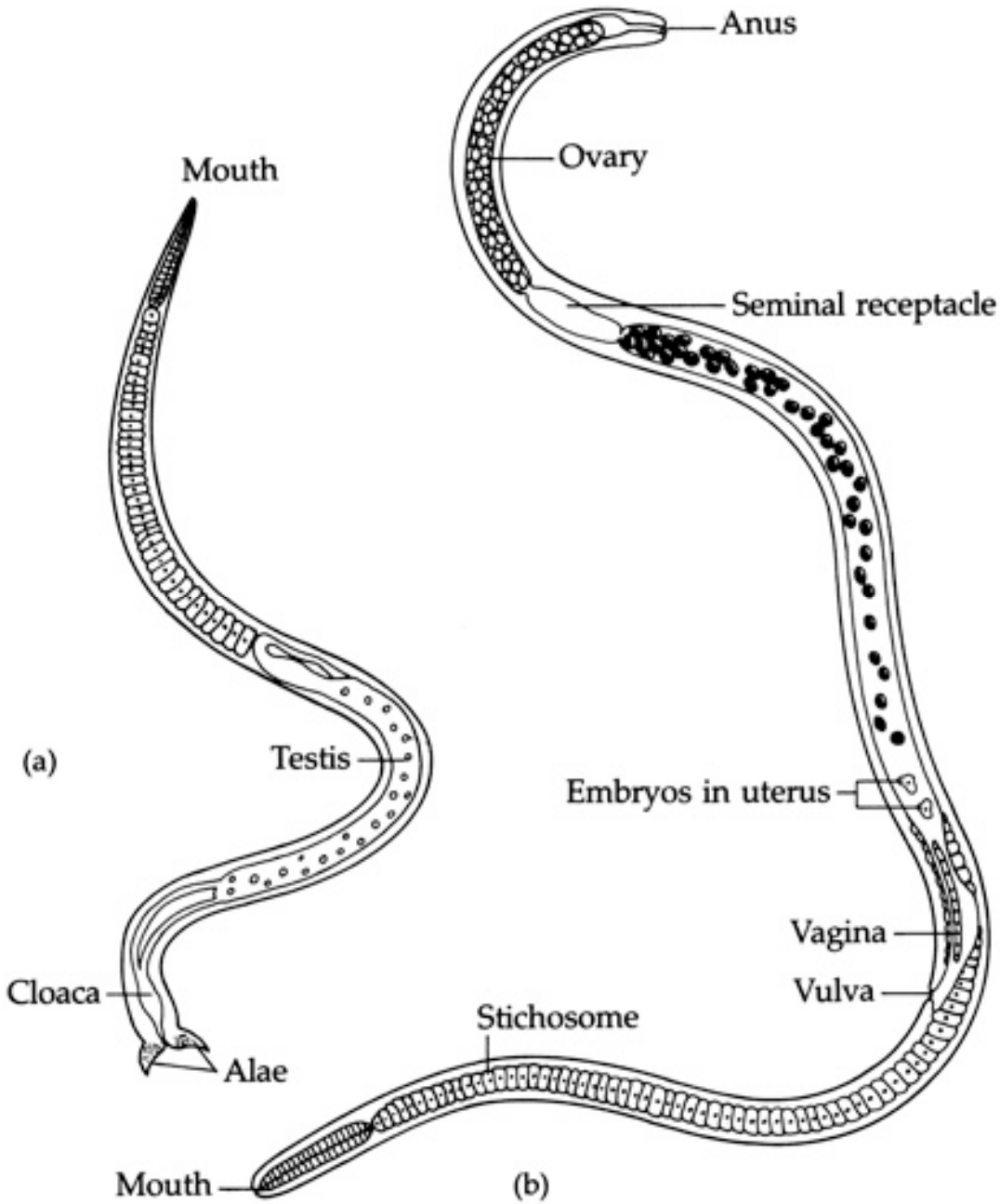
Other Nematodes – Other locations

- ***Trichinella spiralis***
- ***Dracunculus medinensis***
- ***Capillaria spp.***
- ***Angiostrongylus spp.***
- ***Thelazia spp.***
- ***Gnathostoma spinigerum***

Trichinellosis *Trichinella spiralis*

- **Man is not the normal host (in domestic and wild animals)**
- **Worldwide. Not particularly prevalent in tropical countries**
- **11 million people infected**
- **Not soil-transmitted**
- **Two forms: adult and cystic**
- **White worm : ♀ 3-4 x 0. 06 mm**
  ♂ 1.6 x 0.04 mm

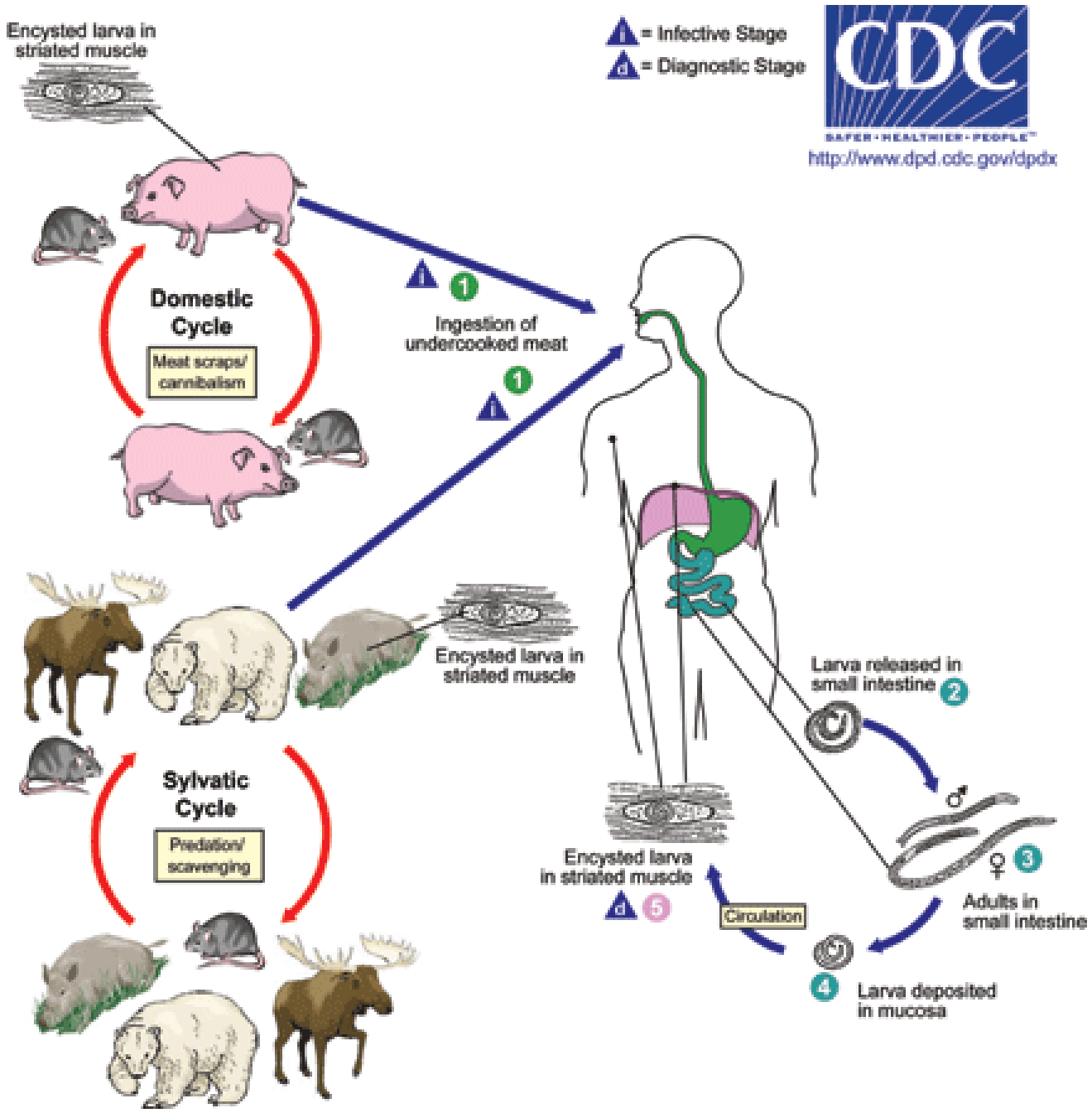


Trichinella spiralis
(a) male (b) female

Trichinella spiralis – Life cycle

- **Female lives for 30 days and is viviparous**
- **Eggs (20µm) are in upper uterus. Larvae (100 x 6µm) break out and live free in uterine cavity of the female (1500 larvae/female)**
- **Larvae emerge 4-7 days after infection, enter lymphatics & blood circulation and reach right heart, lungs ⟶ arterial circulation ⟶ striated muscles and encyst**
- **Ellipsoidal cysts with blunt ends (larvae + capsule of host tissue) lie in parallel to axis of muscle fibers and feed on amino acids of the host. They can remain alive for years**

Larvae of *Trichinella* liberated from bear meat



***Trichinella* spp. life cycle**

Trichinella spiralis – Life cycle

- **Usually calcification after 6 months with death of larva**
- **When eaten by carnivores, cysts pass stomach and larvae hatch in duodenal/jejunal mucosa**

  ⟶ enter columnar epithelium ⟶ adults in 36h
- **From infection to encysting in muscles: 17-21 days**
- **Transmission by mouth from eating undercooked meat**

3 sub-species can infect man:

- ***T. spiralis spiralis*** **: pigs , foxes**
  temperate regions
- ***T. spiralis nativa*** **: polar bears**
  Arctic regions
- ***T. spiralis nelsoni*** **: wild pigs, lions, cheetah**
  Africa, South Europe

Trichinella spiralis – Pathology

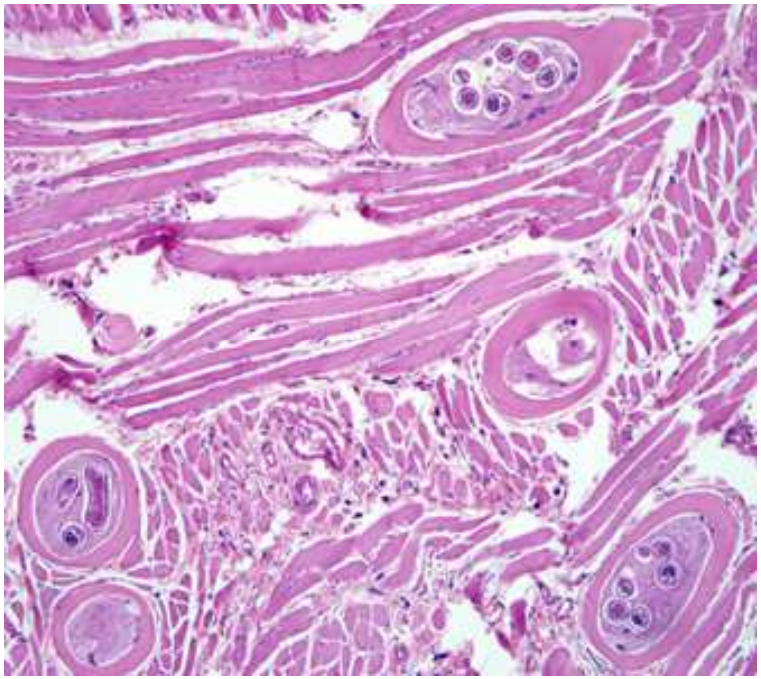
Pathology is related to 3 phases:

- **Enteric phase:**
  - **Larva in duodenal/jejunal mucosa**
  - **Pathology depends on number of larvae**

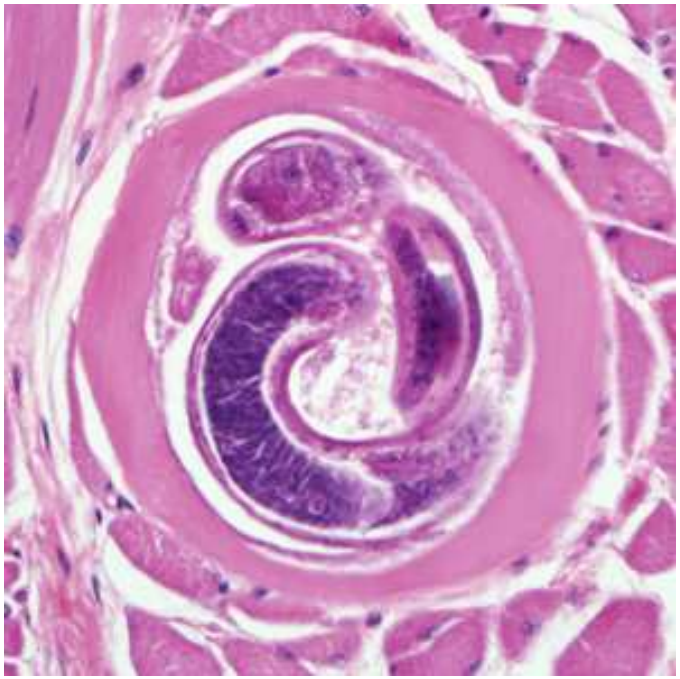
- **Migratory/invasive phase:**
  - **After 5-7 days, females lay larvae in tissues**

- **Encystment phase:**
  - **Larvae encyst in striated muscles but can travel to brain and heart muscle where they cannot encyst**

Encysted larvae of *Trichinella* sp. in muscle tissue, stained with hematoxylin and eosin

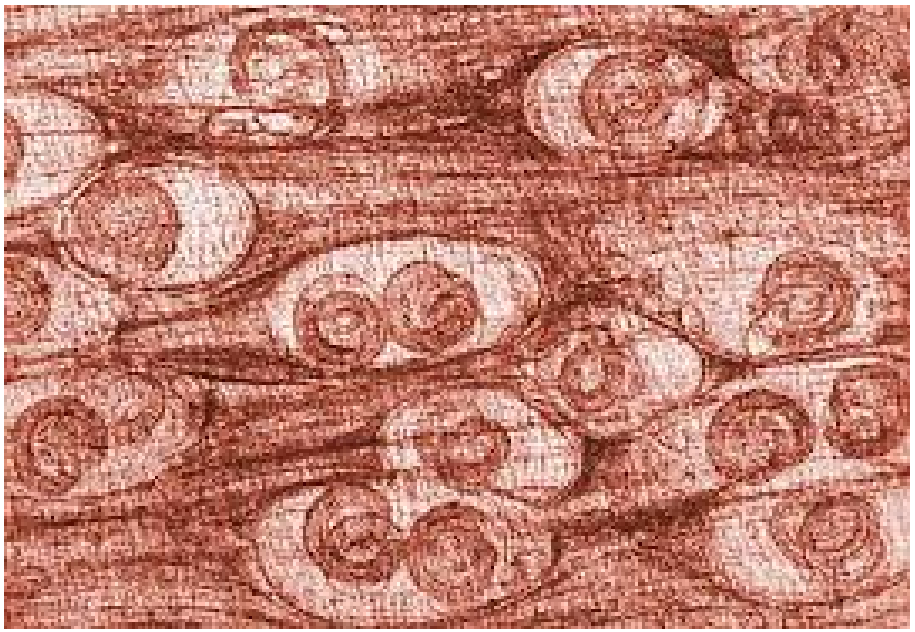


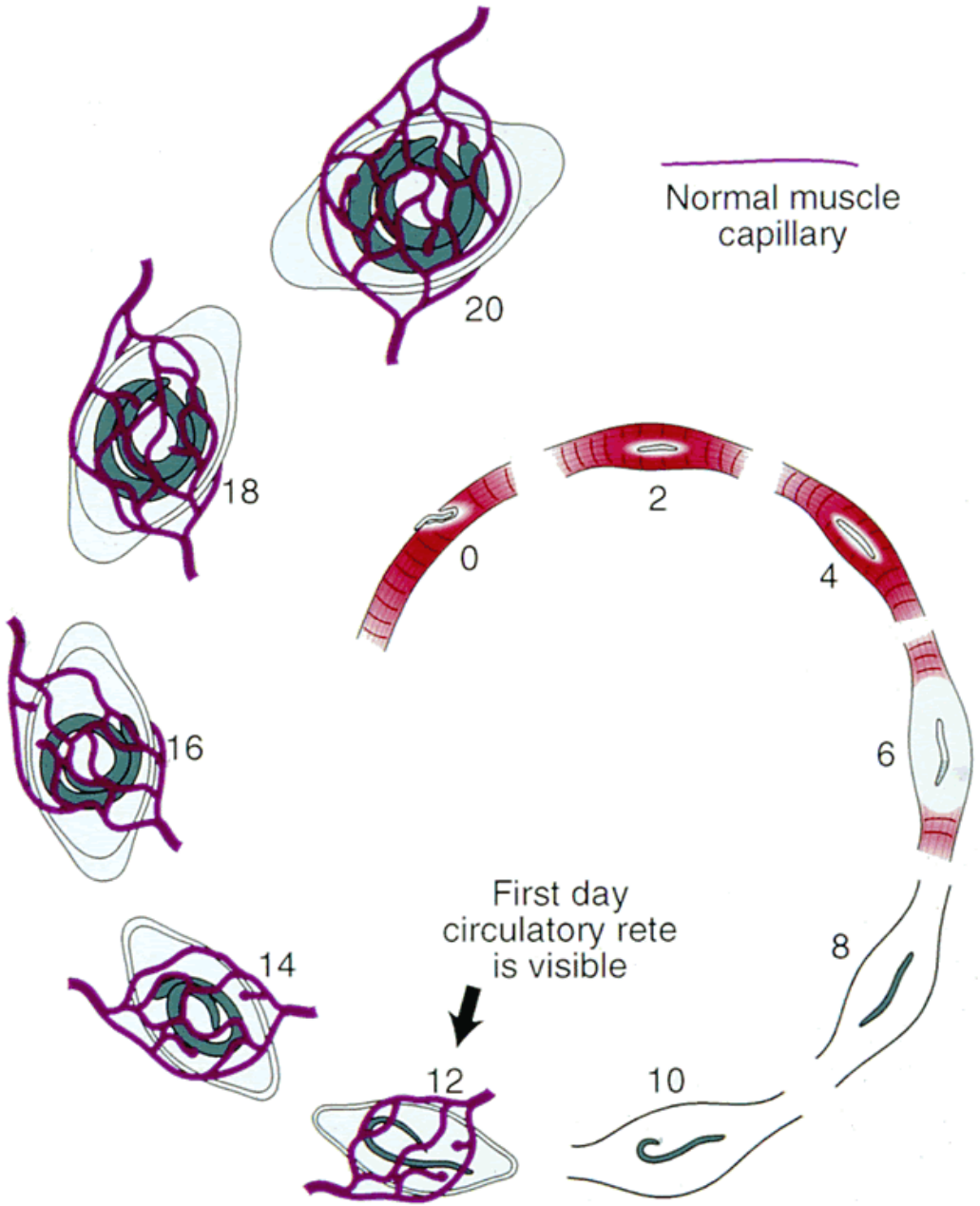
300 x



400 x

Encysted larvae of *Trichinella* sp. in muscle tissue





Trichinella spiralis

Changes in muscle capillaries

Trichinella spiralis – Clinical features

- **If light : asymptomatic, self-limiting 2-3 w, low mortality**
  - **Light : ≤ 10 Larvae/g of muscle**
  - **Moderate: 50-500 L/g**
  - **Severe: ≥ 1000 L/g**
- **Incubation period (from ingestion to enteric phase: 7 days; form ingestion to migratory phase: 7-21 days)**
- **Symptoms depend on no. of larvae/g of muscle**

Trichinella spiralis – Clinical features

- **Enteric phase:**
  - **Irritation & inflammation of duodenum/jejunum**
    - **Nausea, vomiting, colic, sweating**
    - **Skin rash**
    - **In third of cases, pneumonitis for 5 days**

- **Encystment phase:**
  - **Cachexia, oedema, extreme dehydration**
  - **After 2 months, tenderness of muscles decreases**
  - **Congestive heart failure & damage to brain possible**
  - **Jacksonian epilepsy has been described**
  - **Gram- septicemia & permanent hemiplegia have occurred**

Trichinella spiralis – Clinical features

- **Migratory phase:**
  - **Severe myalgia, periorbital oedema & eosinophilia**
  - **Difficulty in mastication, breathing & swallowing**
  - **High remittent fever with typhoidal symptoms**
  - **Splinter hemorrhages under nails & in conjunctivae**
  - **Blood & albumin in urine**
  - **Hyper-eosinophilia from day 14, then decreases**
  - **In severe cases, sub-pleural, gastric & intestinal hemorrhages**
  - **Rare, myocardial complications**
  - **In 10-20% of cases, neurological complications**

Striated muscle	Brain	Heart
Basophilic degeneration of fibres	Leptomeningitis	Considerable damage
Formation of capsule around L with inflammatory infiltration of lymphocytes & eosinophils	Granulomatous nodules in basal ganglia, medulla & cerebellum	Cell infiltration
Deposit of fat at poles of capsule	Cuffing in cortex	Necrosis
After 6 months, calcification and death of larva		Fibrosis of myocardial tissue

Trichinella spiralis – Immunity

- **Good immunity to re-infection but only if the cycle has gone to adult stage**

- **Mainly cell-mediated immunity but also some humoral**

Trichinella spiralis – Differential diagnosis

- **Trichinellosis resembles many other conditions:**
  - **Typhoid, encephalitis, myositis, tetanus, collagen disorders (e.g. rheumatoid arthritis)**
- **Also resembles tissue stages of schistosomes, hookworms, Strongyloides, etc.**

Trichinella spiralis – Diagnosis

By demonstration of L by immunological or molecular methods

- **Trichinoscopy: when encystment has started**
  - **Samples of deltoid, biceps, pectoralis major are digested with 15% pespsin + 1% HCL for hours at 37°c, then filtered, centrifuged ⟹ No L/g of muscle**
  - **Muscle pressed between two slides**
  - **Antigens detection by:**
    - **direct immuno-fluorescence**
    - **microfluorescence**
    - **ELISA**
    - **Western Blot test**
    - **DELFIA (Dissociated Enhanced Lanthemide FluoroImmunoAssay)**

Trichinella spiralis – Management

- **Mebendazole (10days)**
- **Thiabendazole , less well tolerated**
- **In severe infections: prednisolone to control immunological response (Inflammation)**

Trichinella spiralis – Epidemiology/Prevention

- **Man is not the normal host and is infected only when eating raw/undercooked meat**

PREVENTION:

- **Cook meat thoroughly**
- **Meat inspection**
- **Meat refrigeration to destroy cysts**
- **Cooking garbage fed to pigs**

Keep your pigs healthy and happy!

Other Nematodes – Other locations

- *Trichinella spiralis*
- ***Dracunculus medinensis***
- *Capillaria spp*
- *Angiostrongylus spp*
- *Thelazia spp*
- *Gnathostoma spinigerum*

Dracunculiasis *Dracunculus medinensis*

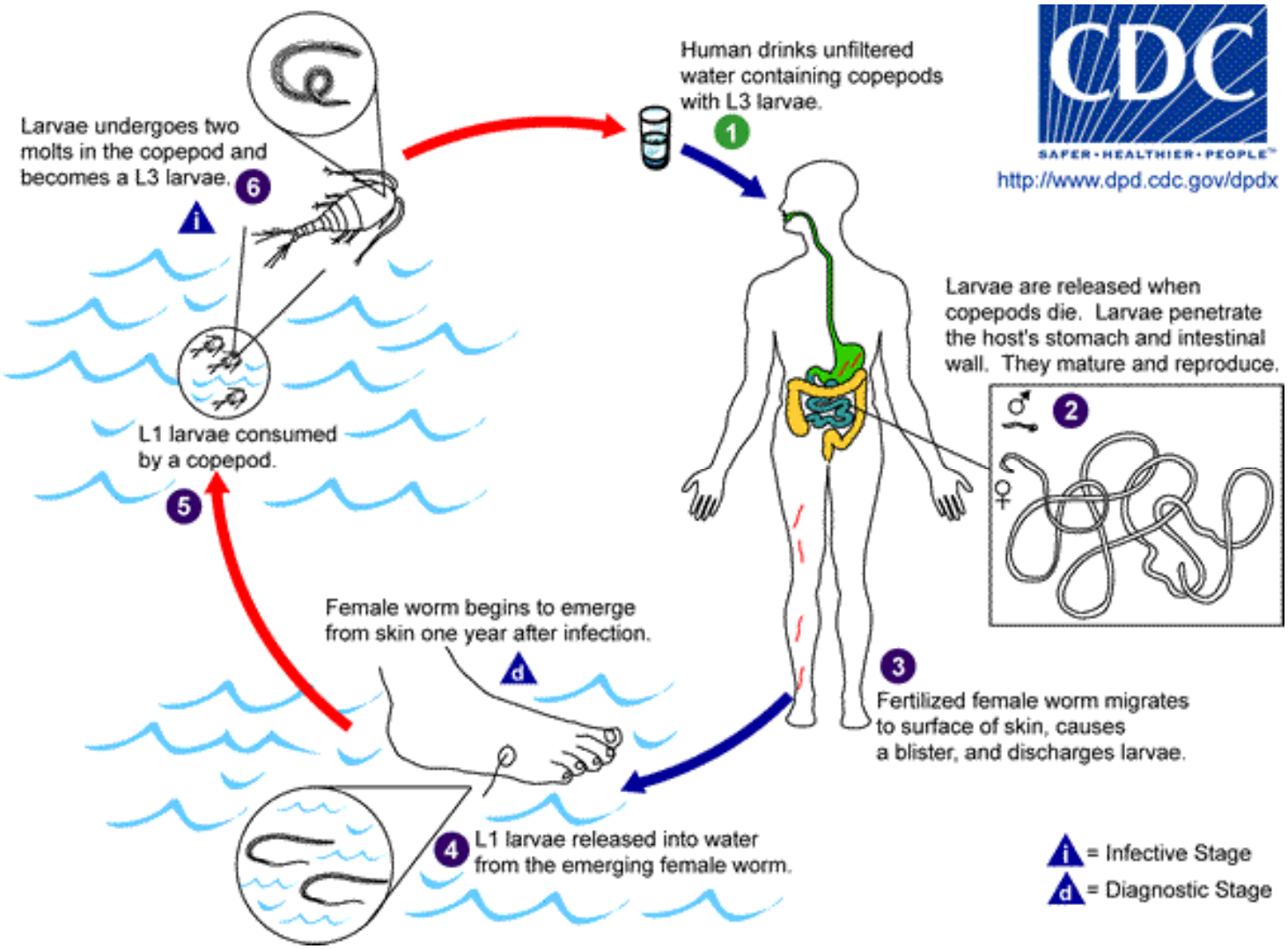
- **Also called Guinea worm. Related to filarial worms but not a "true" filaria as the vector is not a Dipteran**
- **The vector is a water flea (cyclopoid copepod) in freshwater**
- **Infection by drinking water with vectors containing the worm's larvae**
- **Adult female can reach 60-80 cm long and about 2 mm Φ**

Dracunculiasis Epidemiology

- Distribution limited to Sub-Sahel/Sahel Africa
- Use of small sources of water in semi-arid countries
- Seasonal transmission related to rainfall
- Incidence now low (mainly in 15-40 year old)
- No reports in Tanzania but in dogs

Dracunculus medinensis – Vector

Dracunculus medinensis - Life cycle



Dracunculus medinensis – Transmission

Dracunculus - Cycle

- **Adult female (60-80 cm L x 2mm Φ) lives in subcutaneous connective tissues of humans**
- **Can live everywhere in body but mostly in legs/ feet, especially in late stages. Female worm is full of L_1**
- **The female comes close to surface and a blister forms in host's skin. The blister bursts when in contact with water**
- **Female protrudes anterior end & discharges L_1 (600x20 µm) into water (this goes on for 2-6 weeks) then the female dies**
- **L_1 is infective in water for 5-6 days. They must be swallowed by the copepod**

Dracunculus - Cycle

- **In the copepod, the larvae penetrate the gut wall and mould twice in the haemocoele (body cavity) to become infective L_3 (450x14 µm) in 2 weeks**
- **The infected vectors sink at the bottom of the pond and can be ingested with water.**
- **In the stomach, the L_3 is liberated, passes to the intestine, connective tissues and becomes an adult.**
- **Male and female mate about 3 weeks after ingestion. The male dies and the female moves in connective tissues to lower extremities (8-10 months after infection)**
- **The female begin producing eggs**

Dracunculus – Clinical features

- First signs a few days before female pierces the skin
- Blister develops with burning & itching
- On exposure to water, blister ruptures and discharges larvae
- If the worm lives close to joints ⟶ arthritis
- Calcification of worms ⟶ joints of legs/feet become stiff ⟶ crippling
- In 50% cases, ulcer becomes infected with bacteria ⟶ spreading cellulitis
- Tetanus infection might be a complication

Dracunculus medinensis Clinical features

Dracunculus – Clinical features

- Inflammation makes the worm difficult to extract
- When worm is extricated → the ulcer heals
- If the worm breaks → severe inflammatory reaction
- Usually, one worm per year in one patient but can be up to 20 worms
- Some females fail to emerge and die in the body. They are encysted and calcify. This may lead to sterile subcutaneous abscess
- Migration to vital organs (rare) → serious pathology

Dracunculus – Diagnosis

- **Cannot be diagnosed for 8-10 months of infection**
- **When female appears ⟶ see & palpate**
- **Blister is visible, itching & burning**
- **Larvae can be obtained by immersing female in water container ⟶ microscope**
- **Serology is not useful**
- **Radiology of dead calcified worms is possible**
- **No evidence of acquired immunity**

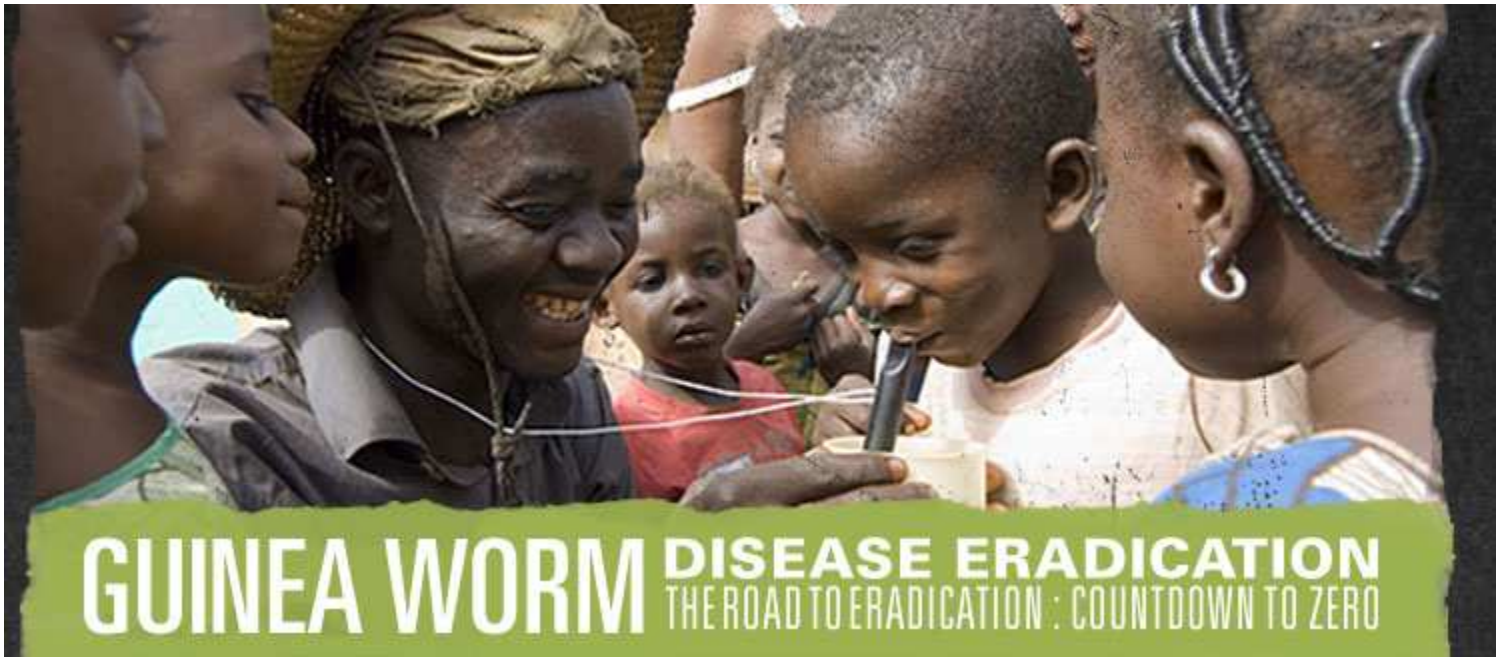
Calcified dead worms visible at radiography

Dracunculus – Management

- **Slow extraction of female worm by rolling it on a small stick (pull slowly each day)**
- **Be careful not to break the worm!**
- **Antibiotics & dressing ulcers to avoid bacterial infection**
- **Tetanus vaccination is recommended**
- **Surgical removal possible**
- **No specific drug available**
- **Niridazole (12.5 mg/kg daily) reduces inflammation and makes extraction easier**

Dracunculus Control

- **Can be eliminated (simple cycle, no animal reservoir)**
- **Improve quality of drinking water (boreholes)**
- **Health education (filtering, boiling water)**
- **Chemical water control**
- **Temephos (Abate) is used as insecticide (kills the copepod)**
- **Surveillance of infected villages**


GUINEA WORM DISEASE ERADICATION
THE ROAD TO ERADICATION : COUNTDOWN TO ZERO

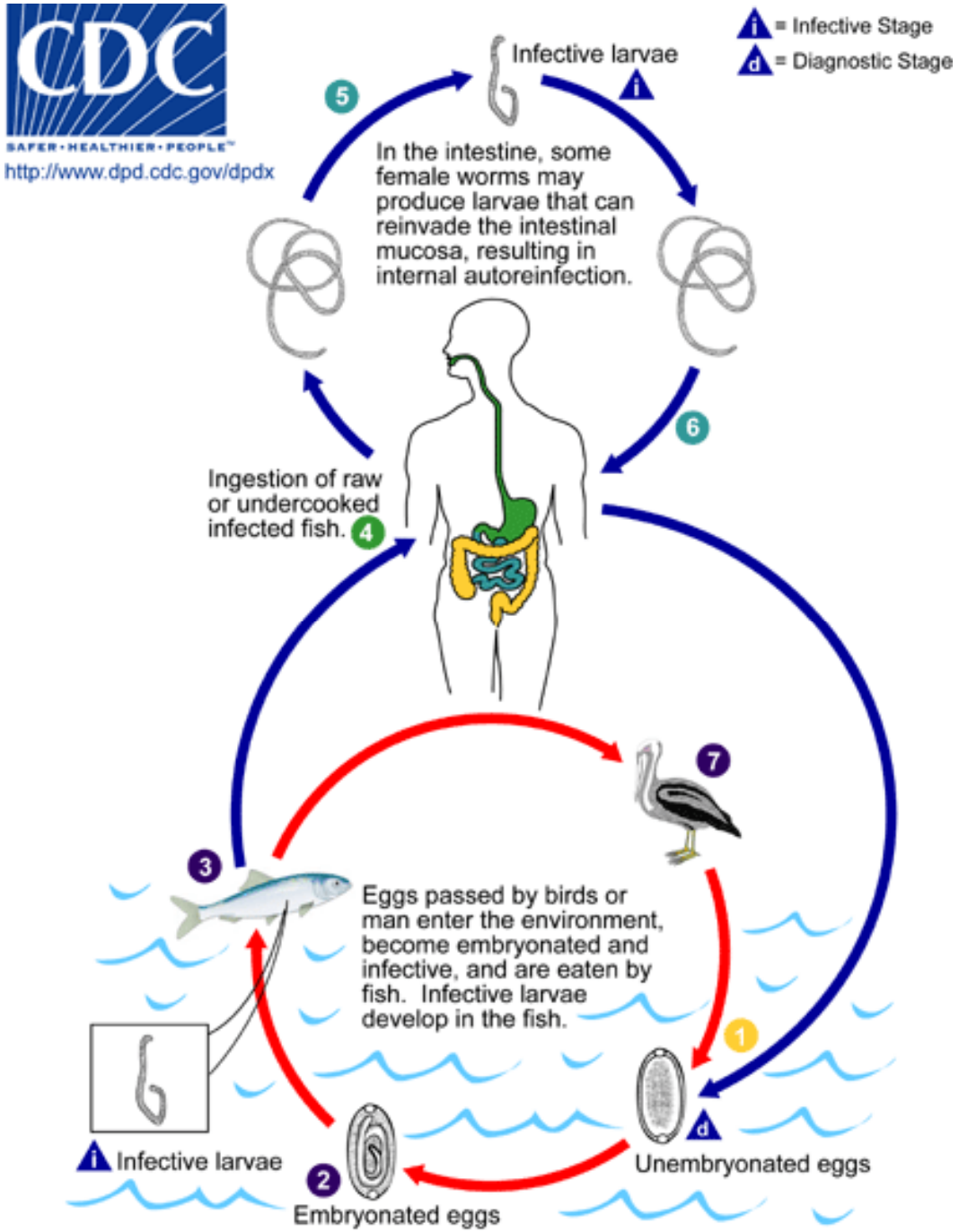
The end....for now!

Other Nematodes – Other locations

- *Trichinella spiralis*
- *Dracunculus medinensis*
- ***Capillaria spp***
- *Angiostrongylus spp*
- *Thelazia spp*
- *Gnathostoma spinigerum*

Capillaria spp: 3 species with potential pathology for man

C. philippinensis	*C. hepatica*	*C. aerophila*
Philippines Thailand	Few cases reported	Few cases reported
Infection by ingestion of undercooked, raw fish	Ingestion of embryonated eggs in stool, contaminated food, water, soil	Unsure. Similar to *C. hepatica ?*
Human intestinal capillariasis	Human hepatic capillariasis Animal parasite, rare in humans	Human pulmonary capillariasis Animal parasite, rare in humans
Abdominal pain, diarrhea, protein-losing enteropathy, cachexia, death	Acute, sub-acute hepatitis, eosinophilia, possible dissemination to other organs. May be fatal	Fever, cough, asthma, pneumonia. May be fatal
Diagnosed by adults, eggs, L in stool or intestinal biopsies In severe infection, eggs, L, adults in faeces	Diagnosed by adults/eggs in liver tissue biopsy	Diagnosed by eggs in lung biopsy

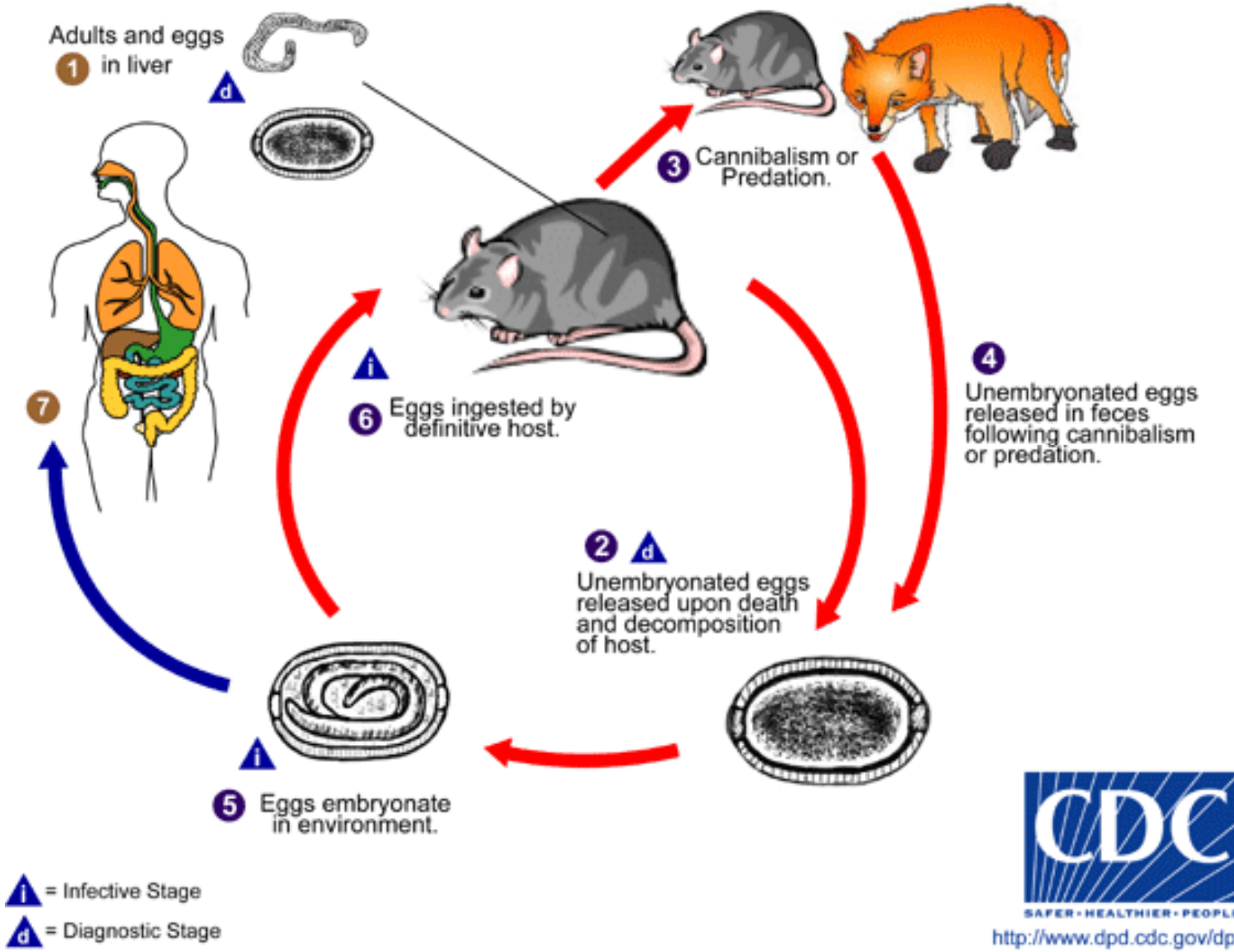


Capillaria philippinensis

-

Life cycle

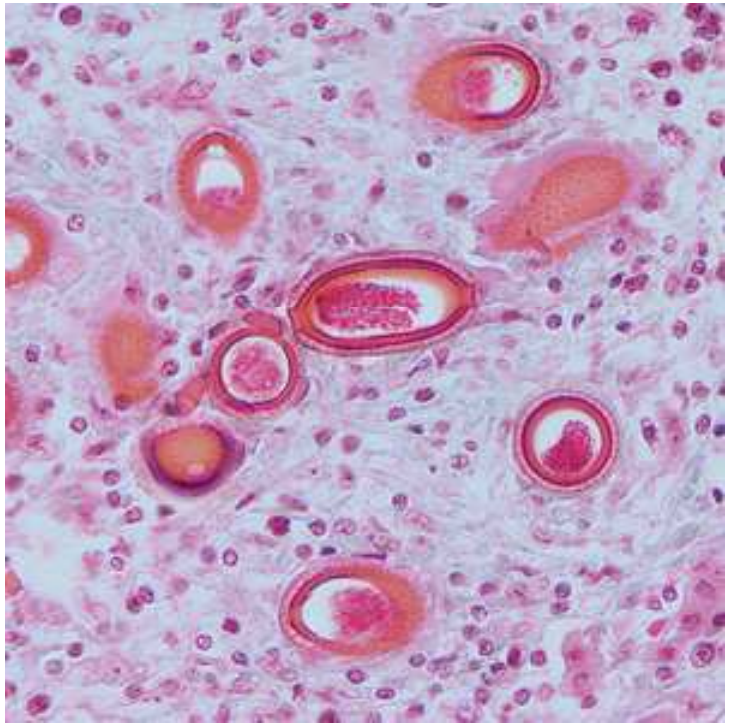
Capillaria hepatica - Cycle



Capillaria spp

C. philippinensis egg

C. hepatica in liver



Capillariasis - Management

- **Mebendazole**
- **Albendazole**

Other Nematodes – Other locations

- *Trichinella spiralis*
- *Dracunculus medinensis*
- *Capillaria spp*
- ***Angiostrongylus spp***
- *Thelazia spp*
- *Gnathostoma spinigerum*

Angiostrongyliasis *Angiostrongylus spp*

- **The nematode (roundworm) *Angiostrongylus cantonensis*, the rat lungworm, is the most common cause of human eosinophilic meningitis.**
- **In addition, *Angiostrongylus (Parastrongylus) costaricensis* is the causal agent of abdominal, or intestinal, angiostrongyliasis**

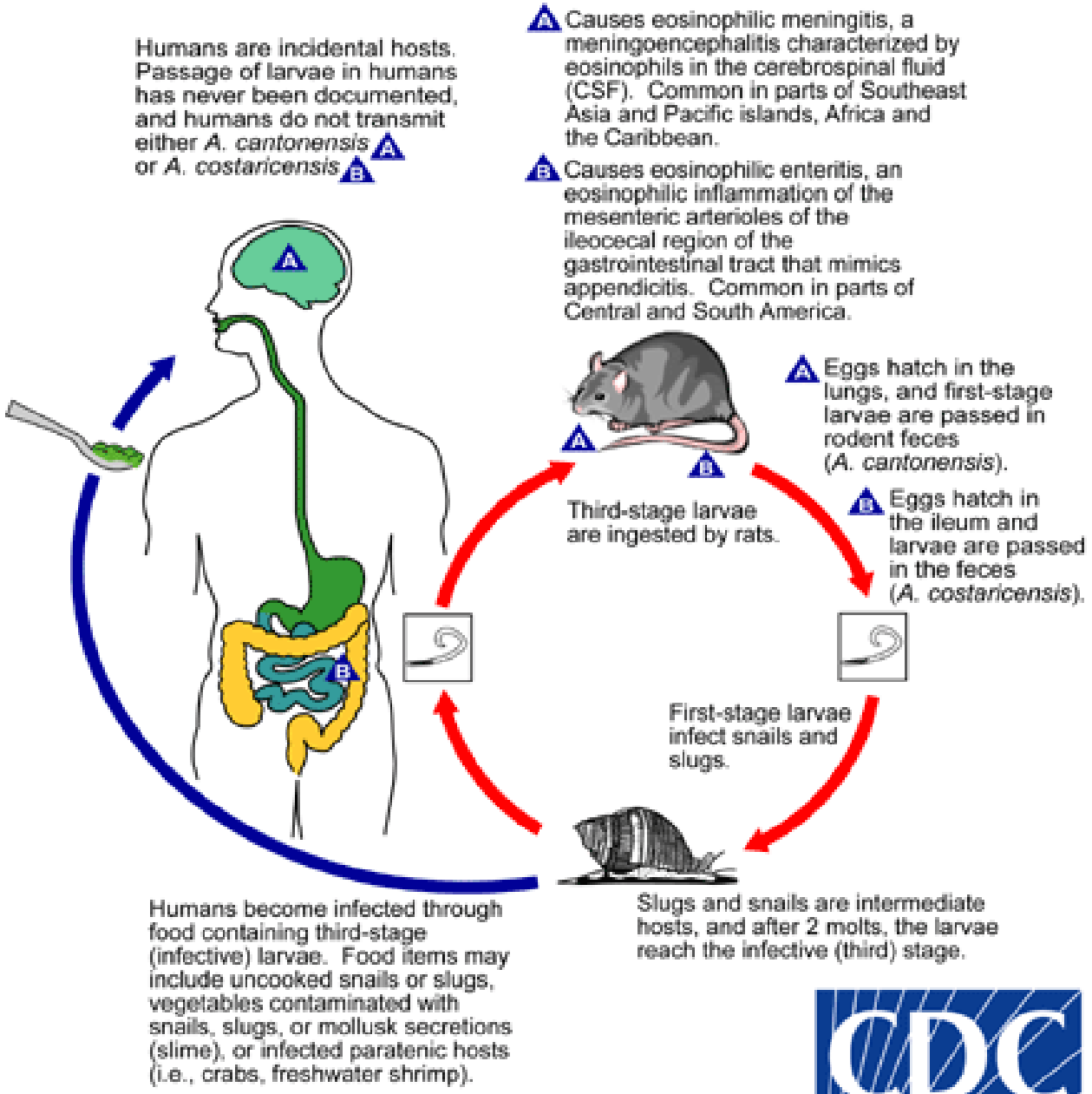
Distribution:

A. cantonensis: **South-East Asia & Pacific Basin but spreading elsewhere, including Africa**

A. costaricensis: **Costa Rica, Young children**

Angiostrongylus cantonensis - Cycle

- **Adults in pulmonary arteries of rats**
- **Eggs discharged into bloodstream and lodge as emboli in smaller vessels**
- **L_1 break through respiratory tract, migrate up the trachea and passes out in faeces**
- **L_1 enter molluscs (*Acatina, Agriolimax*, etc.) intermediate hosts**
- **Two moulds occur around 17^{th} day**
- **When molluscs are eaten by rats, larvae are freed in stomach**
- **They pass to ileum, where they enter bloodstream and congregate in the CNS**
- **The anterior part of cerebrum is the favourite site where third mould takes place (6-7^{th} day) and 4^{th} mould on days 11-13**
- **Young adults emerge from day 12-14 and spread during the next 2 weeks on the arachnoid surface.**
- **From day 28-31,, they migrate to lungs via venous system and settle in pulmonary arteries**



CDC
SAFER·HEALTHIER·PEOPLE™

http://www.dpd.cdc.gov/dpdx

Angiostrongylus spp
Life cycle

Angiostrongylus cantonensis – Clinical features

- **Humans can acquire the infection by eating raw or undercooked snails or slugs infected with the parasite**
- **They may also acquire the infection by eating raw produce that contains a small snail or slug, or part of one**
- **The disease can also be acquired by ingestion of contaminated or infected paratenic animals (crabs, freshwater shrimps)**
- **Clinical symptoms of eosinophilic meningitis are caused by the presence of larvae in the brain and by local host reactions.**
- **Symptoms : severe headaches, nausea, vomiting, neck stiffness, seizures, and neurologic abnormalities.**
- **Occasionally, ocular invasion occurs.**
- **Eosinophilia is present in most of cases.**
- **Most patients recover fully.**
- **Abdominal angiostrongyliasis mimics appendicitis, with eosinophilia**.

Angiostrongylus cantonensis – Diagnosis

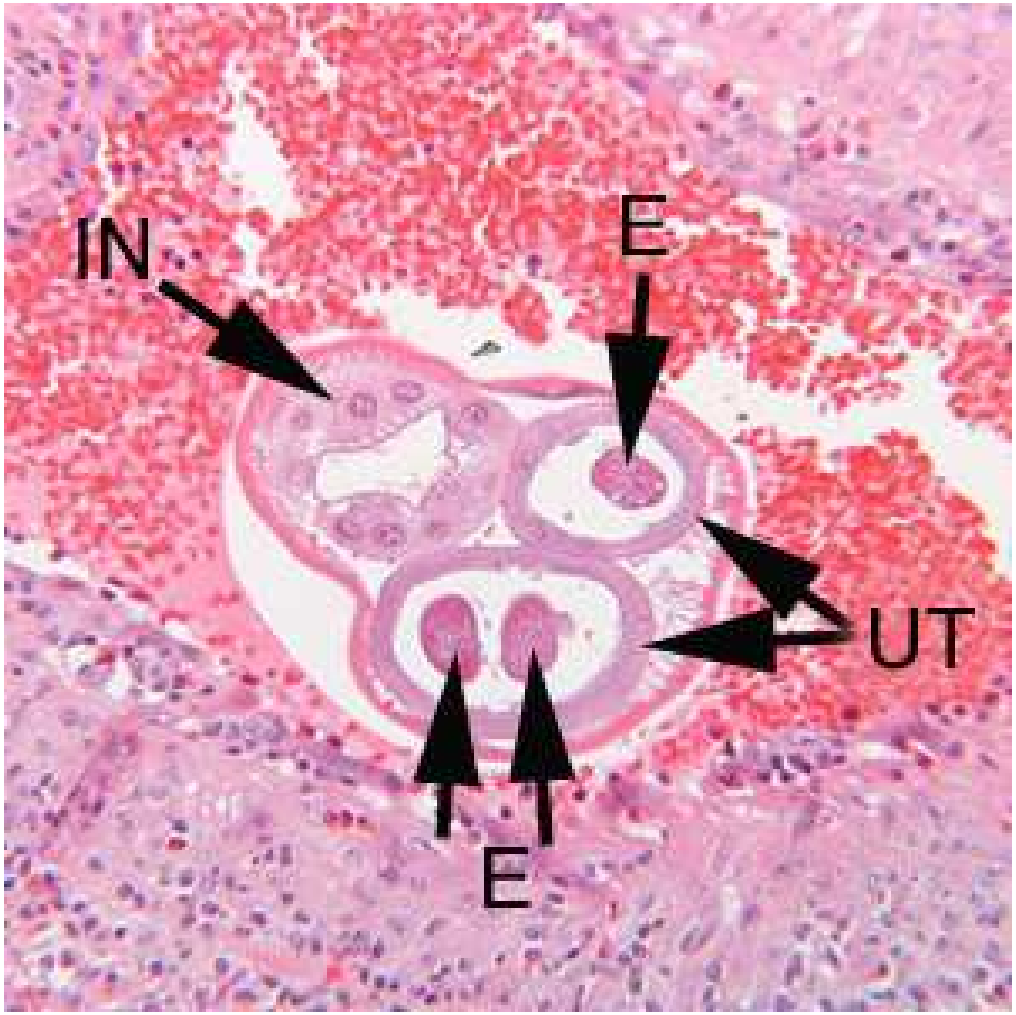
- **In eosinophilic meningitis the cerebrospinal fluid (CSF) is abnormal (elevated pressure, proteins, and leukocytes; eosinophilia)**
- **On rare occasions, larvae have been found in the CSF**
- **In abdominal angiostrongyliasis, eggs and larvae can be identified in the tissues removed at surgery (larvae similar to S. *stercoralis* but smaller)**

Angiostrongylus cantonensis – Management

- **No drug with proven efficacy**
- **Relief of symptoms by analgesics, corticosteroids, removal of CSF at frequent intervals**

***Angiostrongylus* worm. This specimen is approximately 4.25 mm in length and was recovered from vitreous humor of a human patient**

Female *Angiostrongylus costaricensis* in an appendix biopsy, stained with hematoxylin and eosin



IN : Intestine

UT: Uterus

E: Eggs

Other Nematodes – Other locations

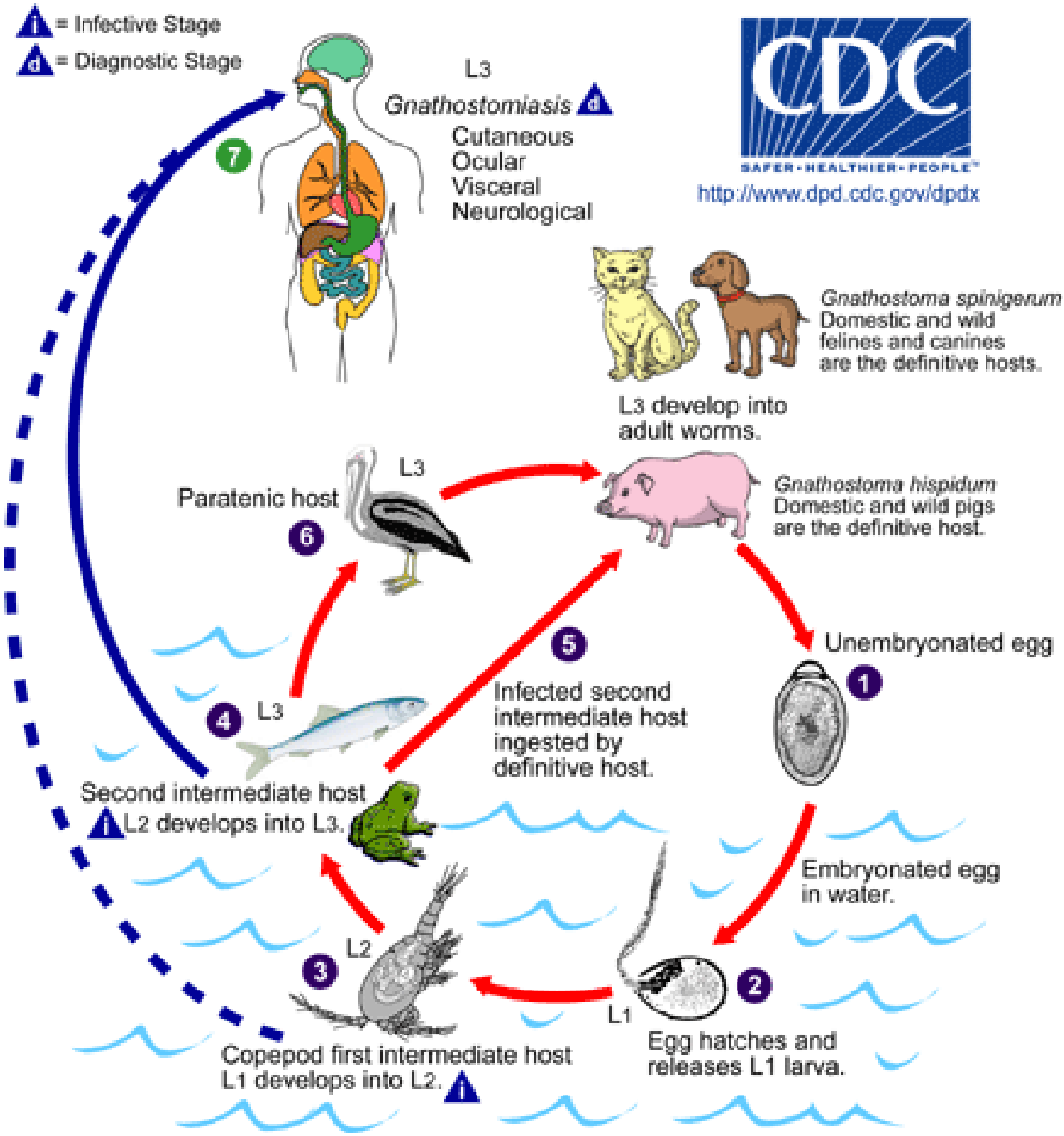
- *Trichinella spiralis*
- *Dracunculus medinensis*
- *Capillaria spp*
- *Angiostrongylus spp*
- ***Gnathostoma spinigerum***
- *Thelazia spp*

Gnathostoma spinigerum

- **Adult worms are parasites of both wild & domestic felines and canines**
- **Widespread in Oriental, Palaeartic, Nearctic regions. Recently imported to Mexico**
- **Adults live in tumours in the stomach wall of cats & dogs**
- **Eggs are extruded form lesions and evacuated in faeces to water, where they hatch**
- ***G. spinigerum* and *G. hispidum* are found in vertebrates**
- **Human gnathostomiasis is due to migrating immature worms**

Gnathostoma – Cycle

- **In the natural definitive host (pigs, cats, dogs, wild animals), the adult worms reside in a tumor which they induce in the gastric wall**
- **Un-embryonated eggs deposited when passed in the faeces**
- **Eggs become embryonated in water and release first-stage larvae(L_1)**
- **If ingested by a small crustacean (*Cyclops*, first intermediate host), L_1 develop into L_2**
- **Following ingestion of the *Cyclops* by a fish, frog, or snake (second intermediate host), L_2 migrate into the flesh and develop into L_3**
- **When the second intermediate host is ingested by a definitive host, L_3 develop into adult parasites in the stomach wall**
- **<u>Or</u>, the second intermediate host may be ingested by paratenic host (such as birds, snakes, and frogs) in which the L_3 do not develop further but remain infective to the next predator**
- **Humans are infected by eating undercooked fish/poultry containing L_3 , or by drinking water containing infective L_2 in *Cyclops***



Gnathostoma spp
Cycle

Gnathostoma – Clinical features

- **The clinical manifestations in human gnathostomiasis are caused by migration of the immature worms (L3s).**
- **Migration in the subcutaneous tissues causes intermittent, migratory, painful, pruritic swellings (cutaneous larva migrans).**
- **Migration to other tissues (visceral larva migrans) can result in cough, hematuria, and ocular involvement, with the most serious manifestations eosinophilic meningitis with myeloencephalitis.**
- **High eosinophilia is present**.

Gnathostoma – Diagnostic/Management

- **Removal and identification of the worm is both diagnostic and therapeutic.**
- **Identification of gnathostomiasis is achieved by serology or microscopic observation of the larval worms in tissue sections**

- **Surgical removal or treatment with Albendazole or Ivermectin is recommended**.

Other Nematodes – Other locations

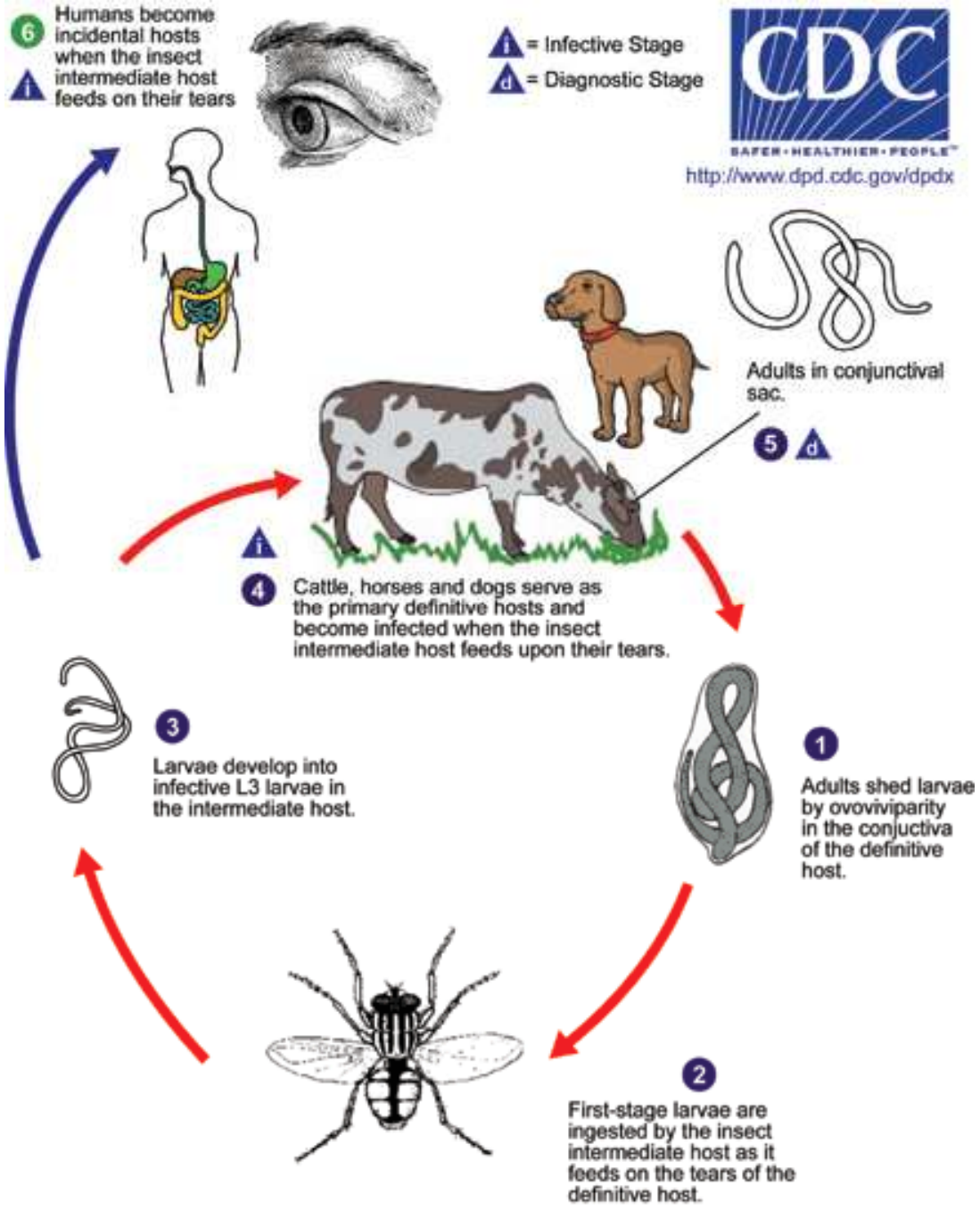
- *Trichinella spiralis*
- *Dracunculus medinensis*
- *Capillaria spp*
- *Angiostrongylus spp*
- *Gnathostoma spinigerum*
- ***Thelazia spp***

Thelaziasis – Aetiology/Distribution

- **Spirurid nematodes of the genus *Thelazia***
- **Two species that have been implicated in human infection:**
  - ***T. callipaeda* (the Oriental eye worm)**
  - ***T. californiensis* (the California eye worm)**
- **Worldwide. Human infections have been recorded from the United States, China, Russia, India, Japan, and Thailand**
- **Dogs and other canids, cattle, and horses are the usual definitive hosts for *Thelazia* spp., although other mammals, including cats, lagomorphs, cervids and humans, can also become infected**
- **Dipteran flies are utilized as intermediate hosts.**

Thelaziasis – Aetiology/Distribution

- **Adults measure up to 2.0 cm in length. The cuticle has coarse striations, often giving the worms a serrate appearance in profile. The mouth is without lips and the esophagus is short. The tail of the male is recurved and without caudal alae; the tail of the female is bluntly rounded.**



Thelazia spp - Cycle

Thelaziasis - transmission

Thelaziasis

Thelaziasis – Clinical features /diagnosis/Treatment

- **Clinical Features**
  - Adults in the eye cause varying degrees of inflammation and lacrimation.
  - In heavier infections, photophobia, edema, conjunctivitis, and blindness may occur.
- **Laboratory diagnosis**
  - Identification is made by finding of adult worms in the conjunctival sac
- **Treatment**
  - Treatment is usually limited to the complete removal of the adult worms from the eye