Helminths (worms)

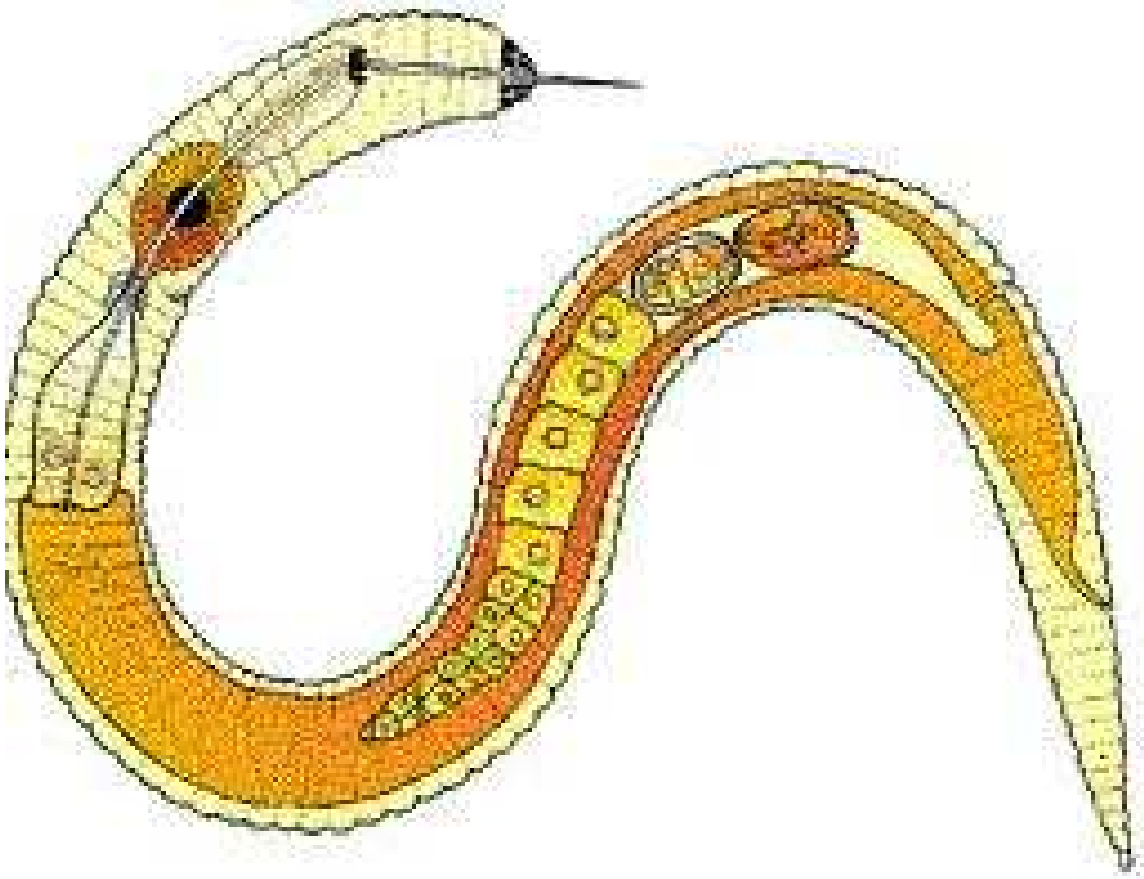
- **Nematodes**: **Round worms, “thread”, bisexual, intestinal, outside**

- **Cestodes**: **Tape worms, Flat worms, segmented, hermaphrodites, intestinal (larva extraintestinal)**

- **Trematodes**: **Flukes, “leaf-shaped”, suckers, hermaphrodites except blood flukes (bisexual). Snail as intermediate host**

Helminths

Nematoda - general morphology - female



Transmission
3 types according to life cycle

- **Type 1: DIRECT TRANSMISSION:**
  - **Embryonated eggs in stools hatch and re-infect within 2-3 hours by anus to mouth (DO NOT REACH SOIL). E.g. *Enterobius vermicularis* (threadworm)**

    ***Trichuris trichiura* (whipworm)**

- **Type 2: MODIFIED DIRECT**

- **Type 3: PENETRATION OF THE SKIN**

Transmission- Ctd

- **Type 2: MODIFIED DIRECT**
  - **Eggs in stools ⟶ develop in soil ⟶ ingestion ⟶ hatching ⟶ Larvae penetrate mucous membrane of stomach ⟶ circulation ⟶ lungs ⟶ esophagus ⟶ intestine where they become adults**

E.g. *Ascaris lumbricoïdes* (roundworm)

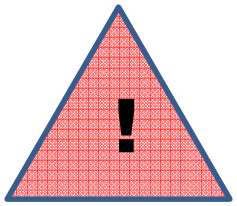
***Toxocara* spp.**

Transmission- Ctd

- **Type 3: PENETRATION OF THE SKIN**
  - **Eggs in stools → soil → hatching → larvae → penetrate the skin → circulation → lungs → esophagus → small intestine → adults**

E.g. *Ancylostoma* spp (hookworm)

Strongyloïdes stercoralis

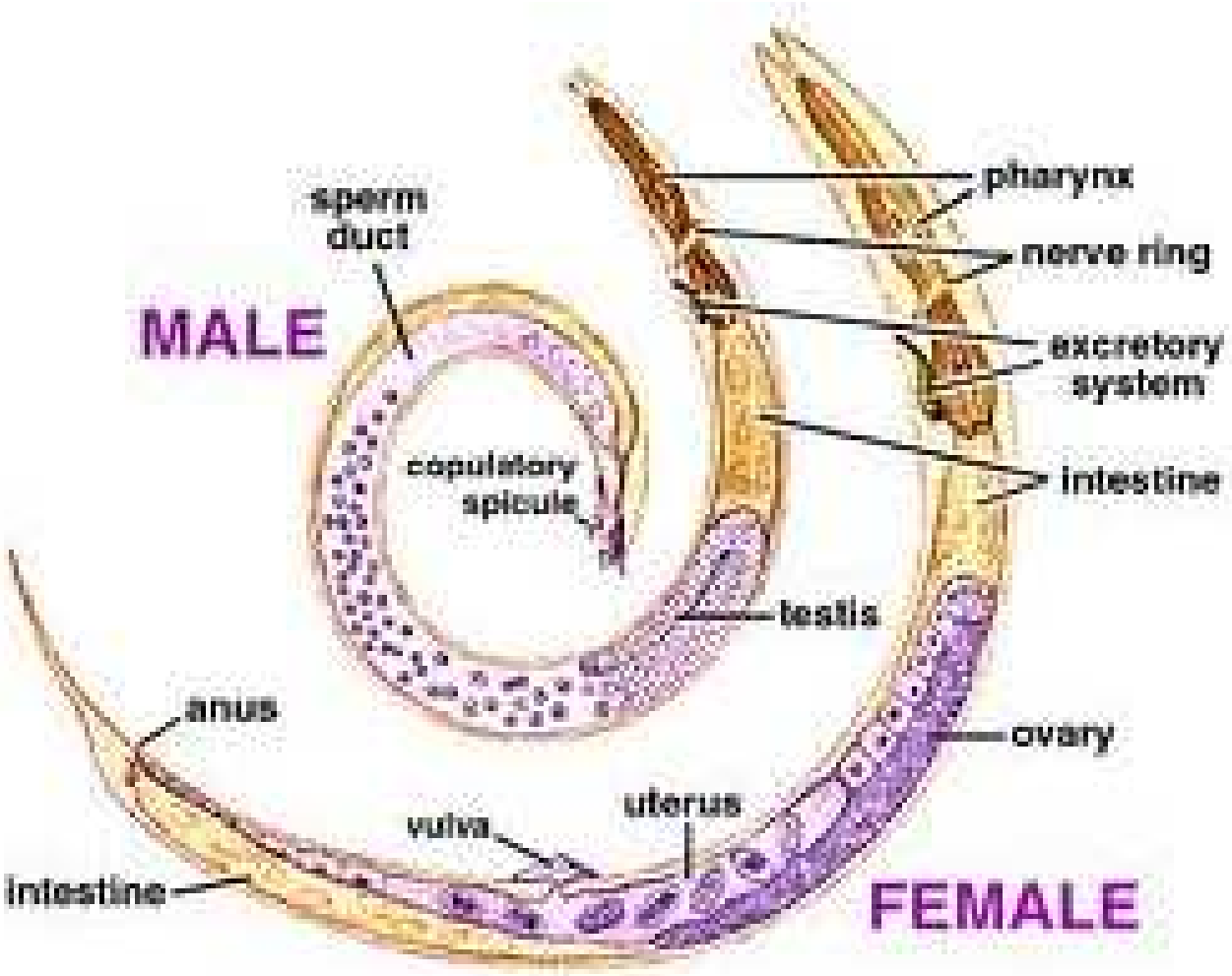


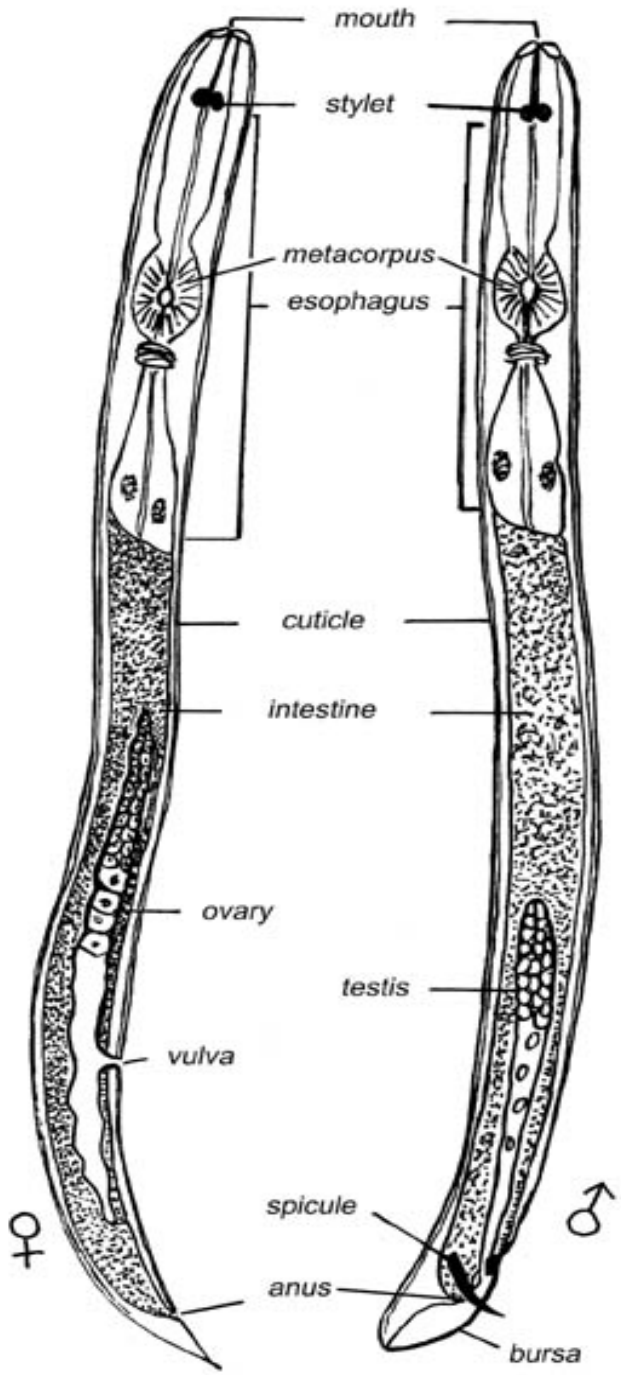
Autoinfection can also occur at the anal margin & can exist in soil without developing in humans!

Enterobius - adults

Enterobius - male

Enterobius – adults

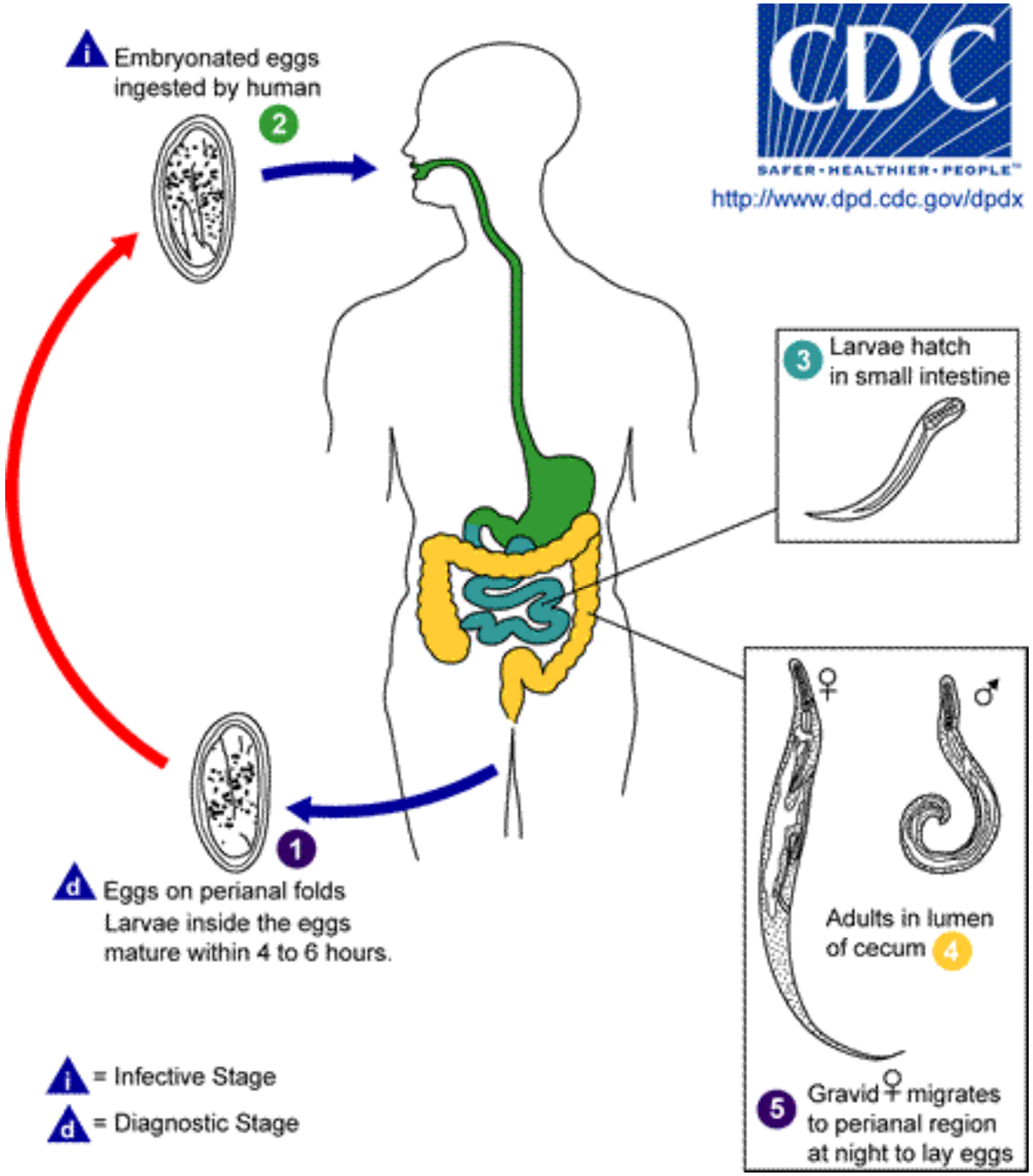




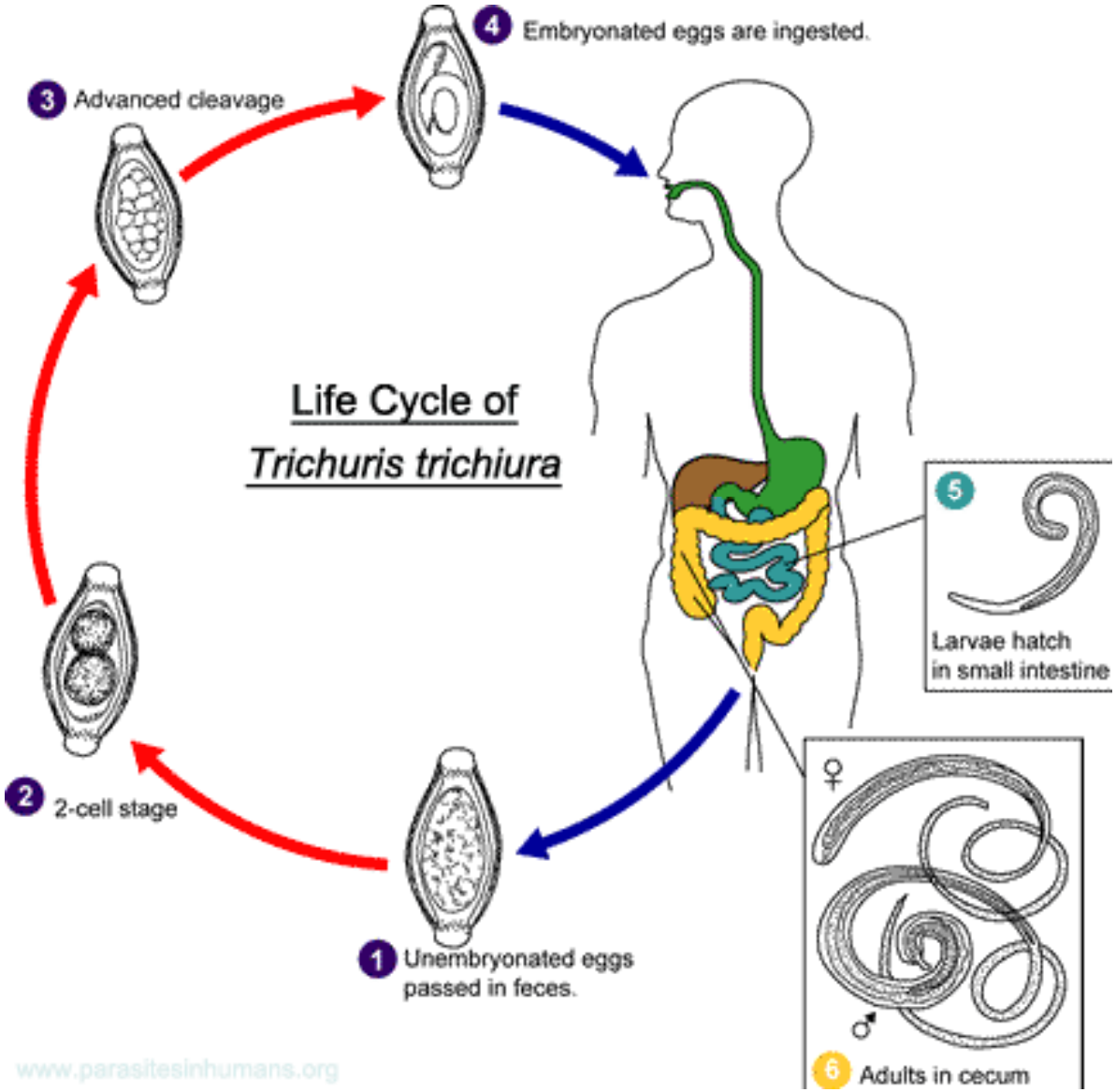
Enterobius - adults

Enterobius - egg

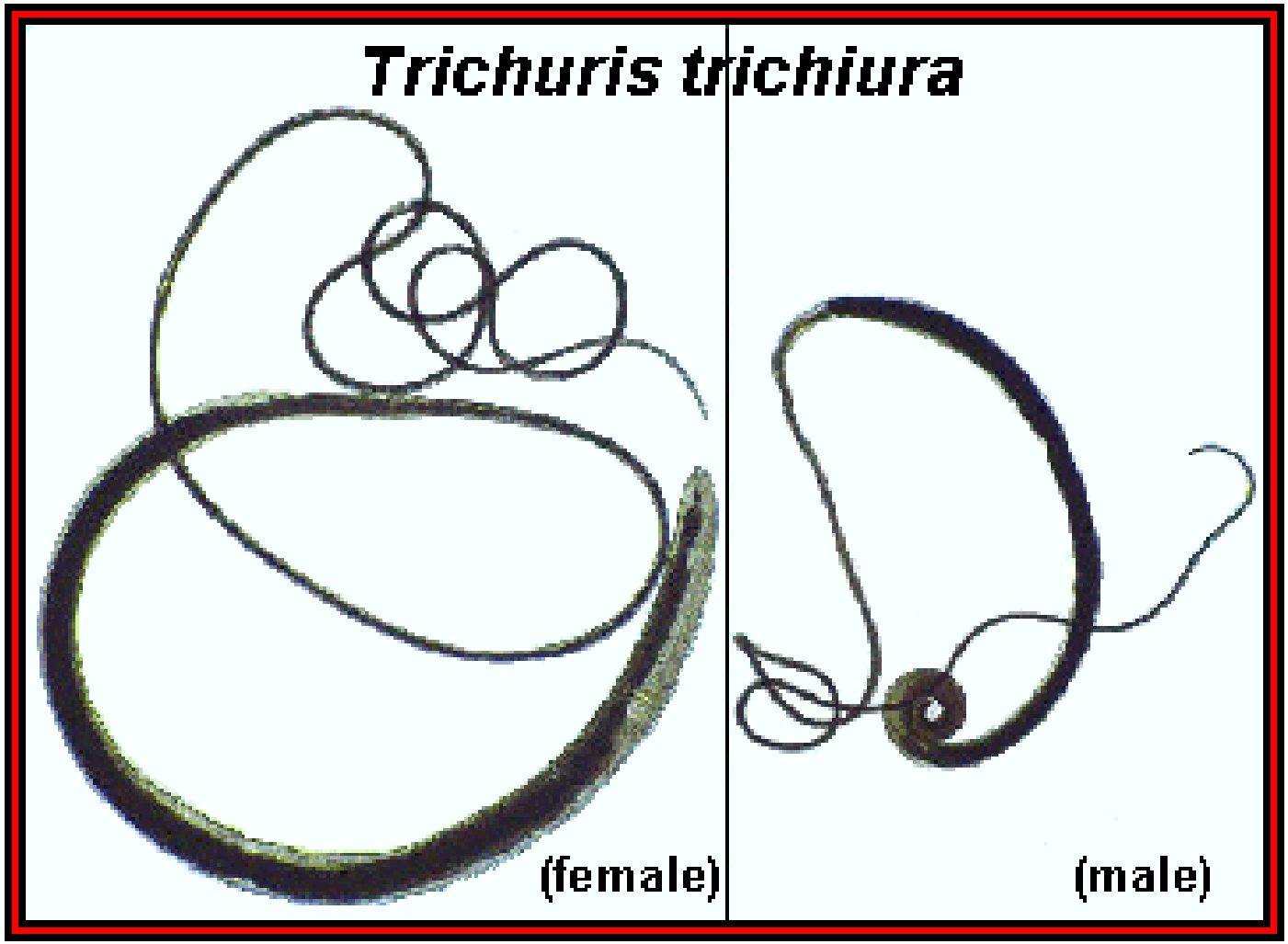
Enterobius – Cycle



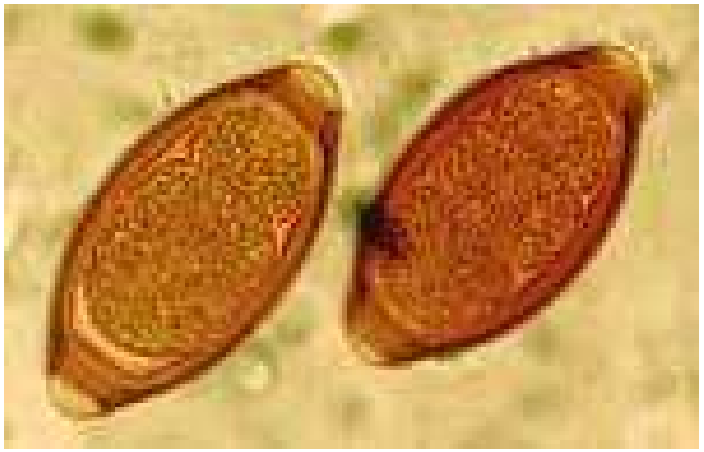
Trichuris - cycle



Trichuris - adults

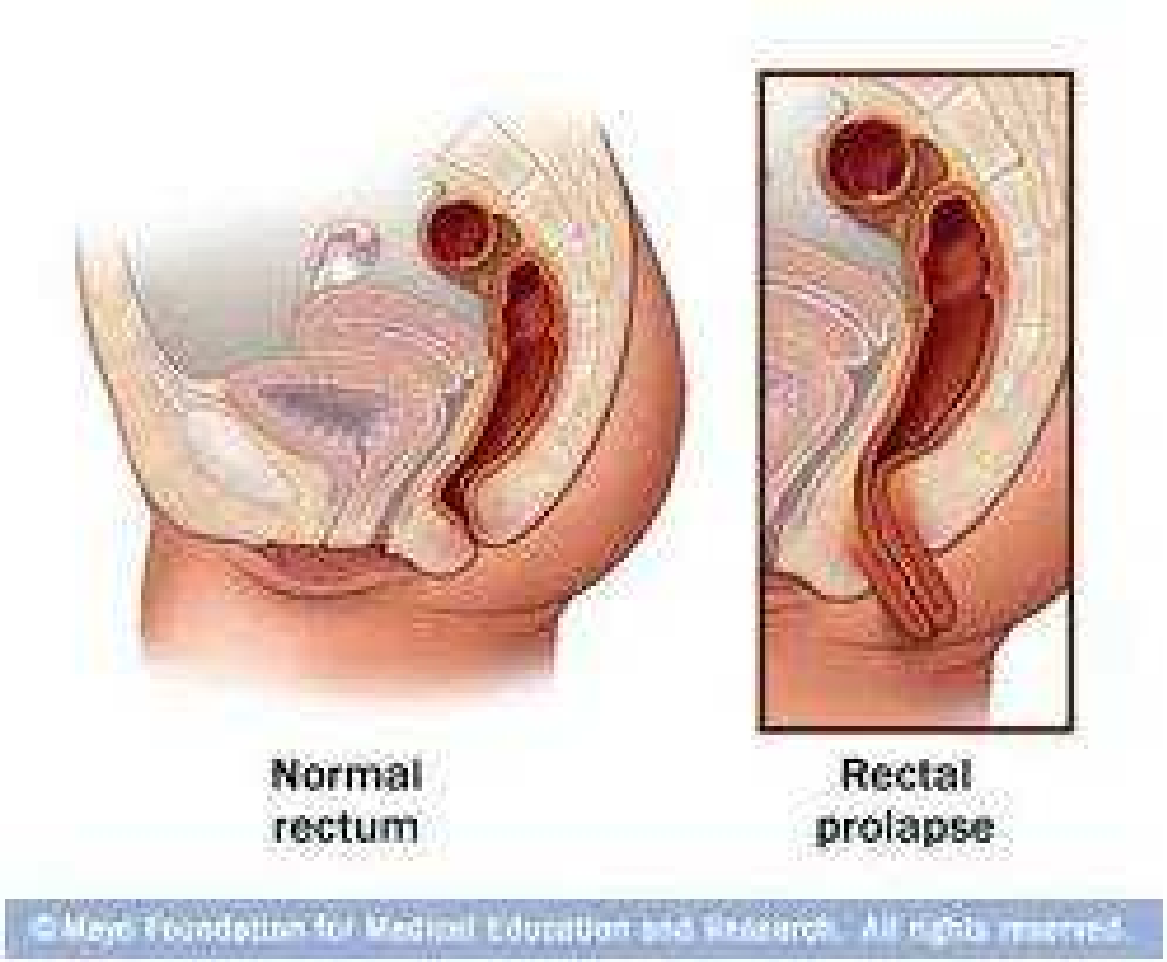


Trichuris



Trichuris – prolapse of rectum

Trichuris – prolapse of rectum



Trichuris – prolapse of rectum

Clubbing of fingers

Transmission
3 types according to life cycle

- Type 1: DIRECT TRANSMISSION
- **Type 2: MODIFIED DIRECT**
- Type 3: PENETRATION OF THE SKIN

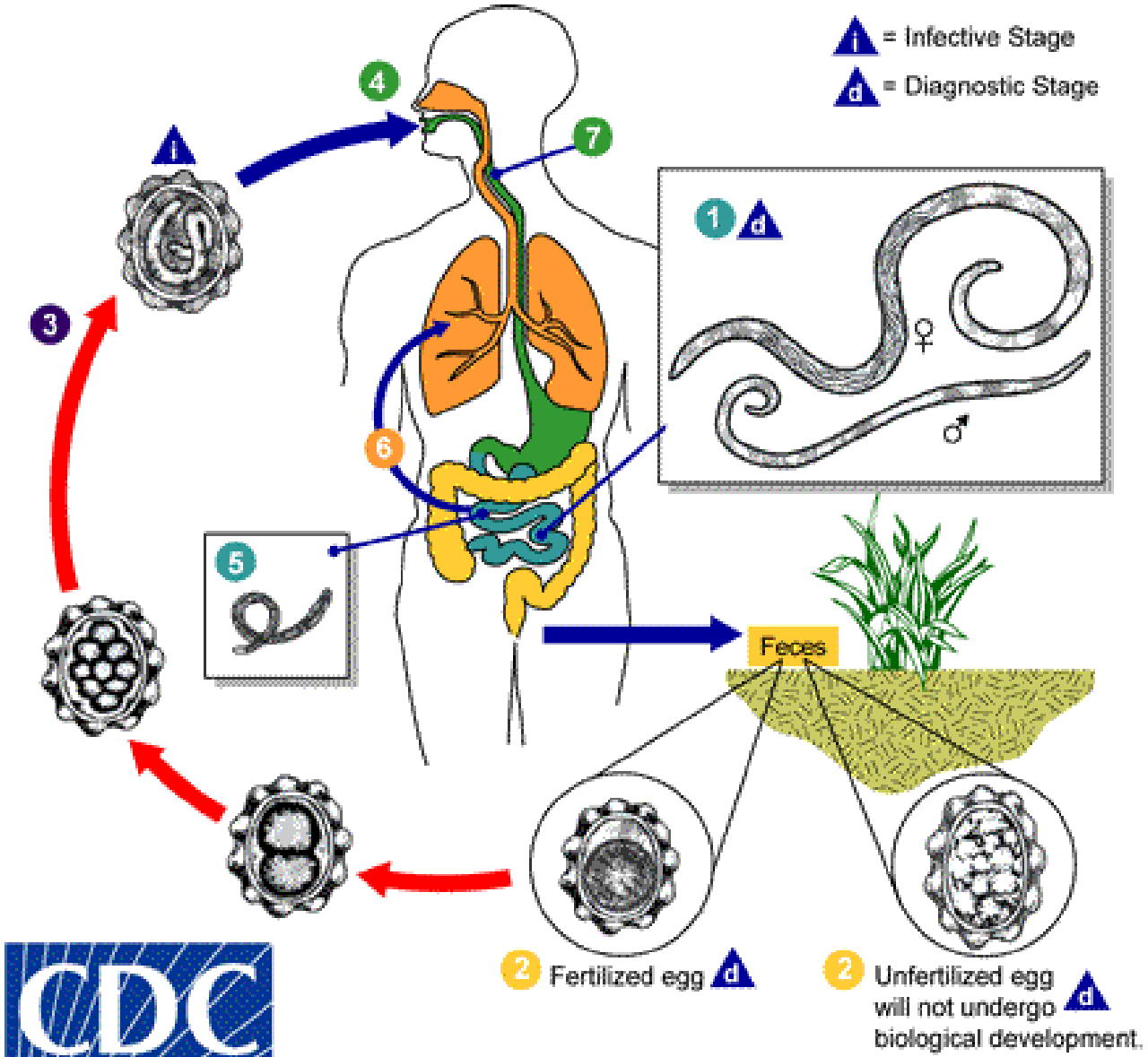
Type 2: MODIFIED DIRECT TRANSMISSION

Ascariasis (*Ascaris lumbricoides)*

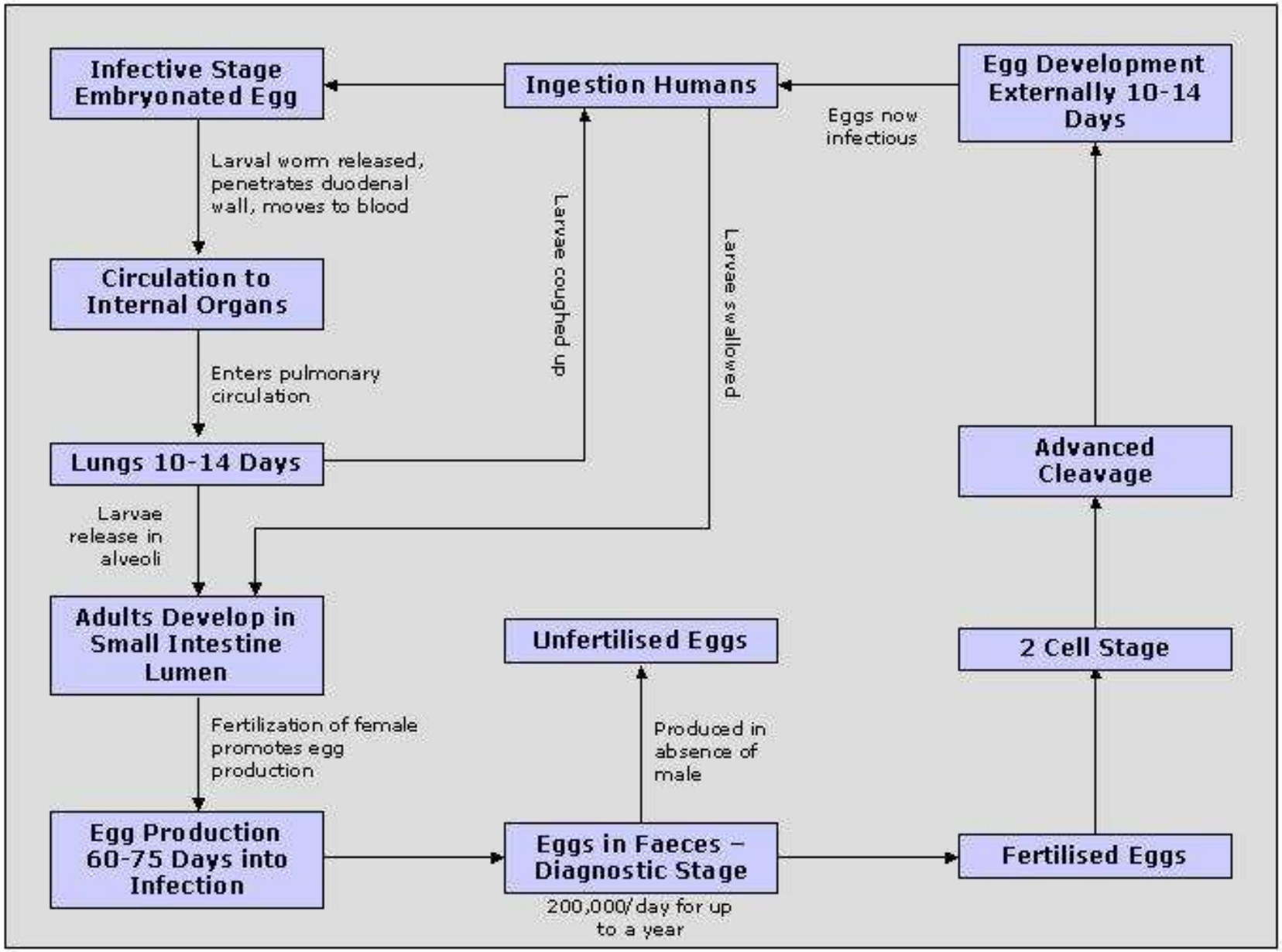
- **Most common, most widespread human infection**
- **1.2 million people infected worldwide**
- **Estimated 12 million cases of acute illness & 10.000 deaths annually**
- **Similar global distribution than *Trichuris***
- **Large worm**
  - **Female ♀ : 20-25 x 3-6 cm**
  - **Male ♂ : 15-31 x 2-4 cm**
- **Eggs (60 x 45 µm), surrounded by mamillated shell**
- **Live in small intestine**

A. lumbricoides- Adult female

Ascaris - Cycle



Ascaris - cycle



- **From eggs ingested to larvae in intestine:**

  10-14 days
- **From infection to eggs in stools : 60-70 days**

- **Transmission via accidental ingestion of eggs in contaminated soil (usually children)**
- **Geophagia**

A. lumbricoides - egg

Ascaris - Aggregate masses of worms

Ascaris – adults

Ascaris – Differential Diagnosis

- **Pulmonary symptoms, lung infiltration & hypereosino -philia are common to several helminths and other infections (toxocariasis, hookworm, *Strongyloides*, schistosomes, TPE, aspergillosis**)

- **Larval ascariasis** is short-term (2-3 w) with rapidly falling eosinophilia
- ***Toxocara* spp** also cause the visceral larva migrans(VML) syndrome but last many months with high eosinophilia
- **Hookworms** invasive stage lasts 2-3 m (specif. serol.)
- **Schistosomes** invasive stage (Katayama syndrome) lasts 2-3 m with splenomegaly (specif. serol.)
- **TPE** is mainly in adult, longer duration, filarial tests + and responds to diethylcarbamazine
- **Aspergillosis** and drug reactions are more chronic

Type 2: MODIFIED DIRECT TRANSMISSION

Toxocariasis (*Toxocara canis & T cati)*

- **Dogs & cats ascarids**
- **Man abnormal host and development stops at larval stage which causes toxocariasis, visceral larva migrans (VLM), ocular and covert toxocariasis**
- **Worldwide (2% to 86% prevalence in humans)**
- **Same morphology as Ascaris**
- **Large worm**
  - **Female ♀ : 6.5 – 10 cm long**
  - **Male ♂ : 4 – 6 cm long**
- **Eggs (85 x 75 µm), pitted superficially. In dogs, cats faeces and soil**
- **Transmission: Mostly children playing in contaminated soil or eating earth**

Toxocara canis - adults

Toxocara canis - egg

Toxocara - Cycle

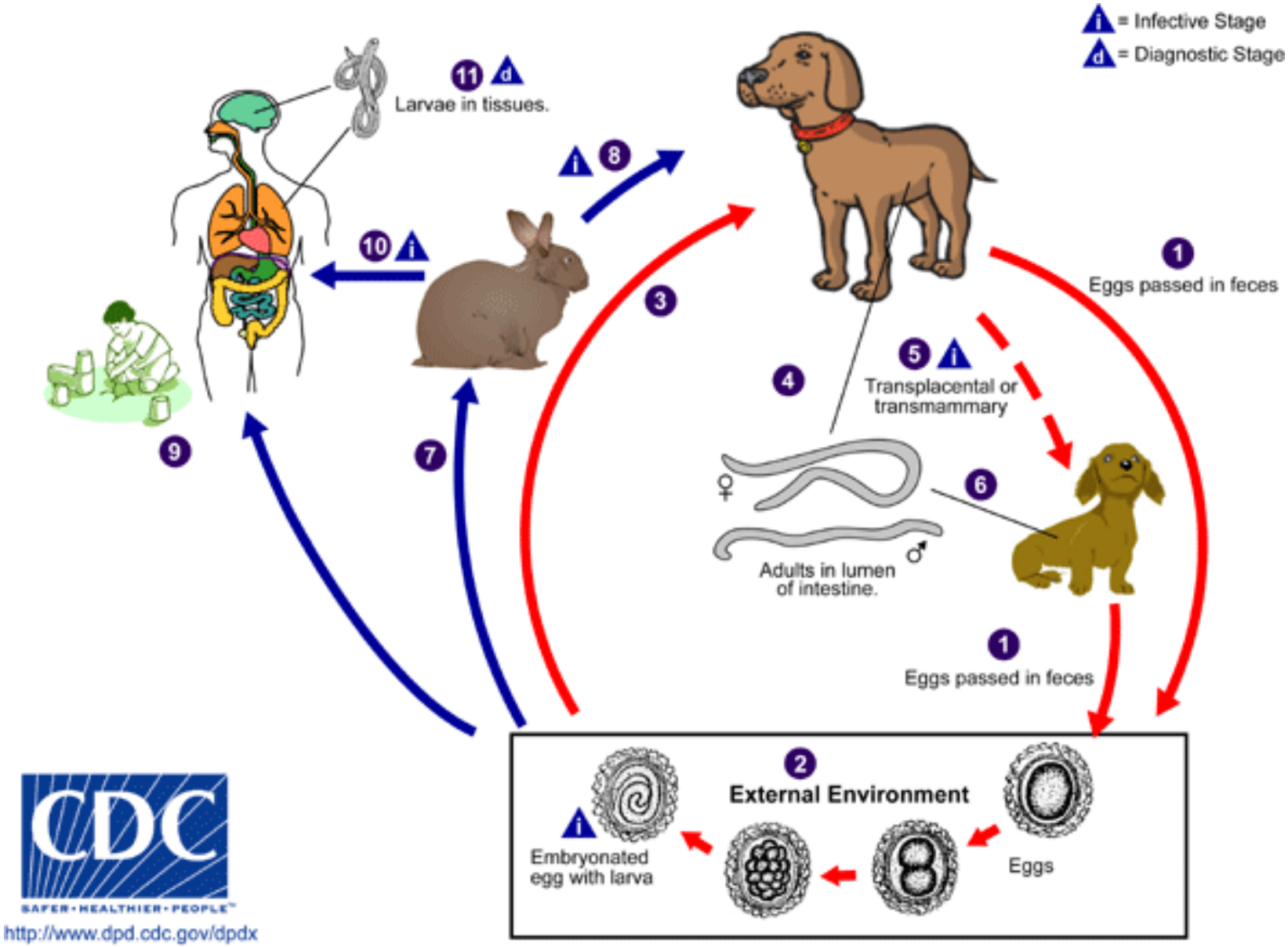
In dog/cat :

- **Ingestion of eggs from soil and cycle similar to *Ascaris* but transplacental infection is possible**

In humans:

- **Eggs ingested hatch in stomach $\longrightarrow$ L_2 which penetrates mucosa $\longrightarrow$ circulation via mesenteric vessels $\longrightarrow$ intestinal viscera & liver (held up by capillaries). They may pass into general circulation via lungs to brain, eyes, etc. causing granulomatous reaction (pathology)**
- **Larvae do not mould but can live up to 11 years**

Toxocara canis - Cycle



Toxocara - Pathology

- **Depends on intensity of infection**
  - **VLM (heavy infected children)**
  - **Toxocariasis (lighter infections, later in life)**

- **VLM:**

- **L_2 (450 length x 20 µm ϕ) are arrested mostly in liver and cause granuloma**
- **Also in lungs, kidneys, heart, striated muscles, brain, eye**

Toxocara - Pathology

- **VLM:**

❑ **Granuloma:**

- ❑ **Centre of packed eosinophils & histiocytes**
- ❑ **Surrounded by large histiocytes (palisade)**
- ❑ **Maybe giant cells**
- ❑ **L_2 remains sometimes**

- **Ocular toxocariasis:**

❑ **Granulomatous reaction in eye ⟶ sub retinal mass with patch of choroiditis Resembles retinoblastoma.**

Toxocara - Immunity

- **Larvae provoke humoral response**
- **Rise in IgG, IgM, IgE**
- **Rise in peripheral eosinophils**
- **Cellular response: cell-mediated granuloma response around the larvae**

Toxocara – Clinical features

- In most cases, larvae are destroyed without trouble
- In some cases, larvae survive for years and cause lesions
- Only trouble is with heavy infection and VLM. This can be self-limiting or fatal (rare)
- In eye: loss of vision, loss of sight

Toxocara – Symptoms & signs

VLM

- **Mostly in younger children**
- **Enlarged liver, fever, asthma**
- **Hypereosinophilia & hyperγglobulimaemia**
- **Sometimes pulmonary signs, cardiac dysfunction, nephrosis**
- **Severe: neurological lesions (fits, paresis, transverse myelitis)**

Toxocara – Symptoms & signs

- **Most VLM cases recover after 2 years but some fatal**
- **Retinal lesions (at/near macula) lead to vitritis, cystoid macular oedema & traction retinal detachment**
- **Strabismus due to damage is often the symptom**
- **L_2 may rarely be seen in anterior chamber of eye**

COVERT TOXOCARIASIS: cough, sleep disturbance, headache, abdominal pain

Toxocara – Diagnosis

- **VLM : - Stable eosinophilia**
  - Leukocytosis
  - Decrease of albumin:globulin ratio
  - Increase of IgG, IgH, anti A or B iso-haemagglutinin titres
  - Hypoechoic area in liver by US

Toxocara – Diagnosis (Ctd)

- **Serology:**
  - **ELISA using excretory – secretory (ES) antigens from L_2 in vitro but some cross reactions . Improved if based on specific IgE & IgG subclasses**
  - **Cannot differentiate between past and present infection**
- **Ocular toxocariasis:**

  Ophtalmologic examination. Serum antibodies detection, fluorescein angiography, computed tomography to differentiate from ocular larva migrans

Toxocara – Differential diagnosis

- **VLM: See ascariasis.**
- **Ocular toxocariasis must be distinguished from retinal tumour (retinoblastoma) and other causes of choroiditis (toxoplasmosis)**
- **Exclude toxocariasis in all cases of retinoblastoma in children (ELISA)**

Toxocara – Management

- **Albendazole & Mebendazole (2xd for 5d)**
- **Thiabendazole**
- **Diethylcarbamazine not recommended**
- **In VLM, high eosinophilia may persist but relapses do not occur**
- **In severe ocular toxocariasis, corticosteroids may be needed**

Toxocara – Epidemiology

- **Common in adult dogs and cats**
- **In tropical areas, often associated with *Ascaris* and *Trichuris***
- **Seroprevalence increases throughout early childhood and stabilizes around 2.5 years**
- **Uncommon after 5 years of age, except ocular form**

Toxocara – Control

- **Control in dogs/cats by treatment**
- **Health education**
- **Prevent soil contamination by animals faeces**
- **Hand washing**

Transmission
3 types according to life cycle

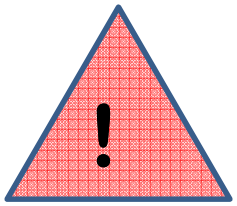
- Type 1: DIRECT TRANSMISSION
- Type 2: MODIFIED DIRECT
- **Type 3: PENETRATION OF THE SKIN**

Transmission- Ctd

- **Type 3: PENETRATION OF THE SKIN**
  - **Eggs in stools ⟶ soil ⟶ hatching ⟶ larvae ⟶ penetrate the skin ⟶ circulation ⟶ lungs ⟶ esophagus ⟶ small intestine ⟶ adults**

E.g. *Ancylostoma* spp (hookworm)

Strongyloïdes stercoralis



Autoinfection can also occur at the anal margin & can exist in soil without developing in humans!

Type 2: PENETRATION OF THE SKIN

Ancylostomiasis (*hookworm disease)*

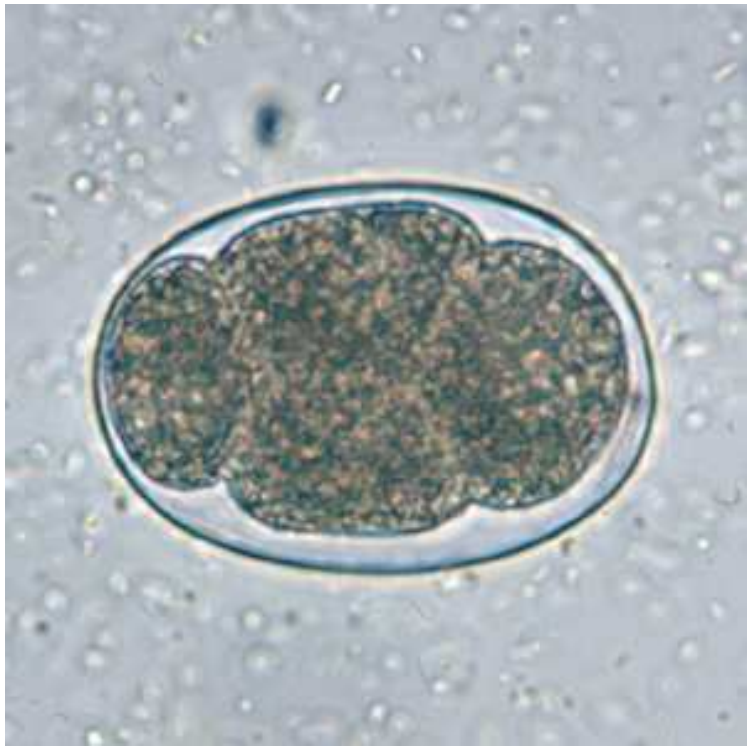
- **Caused by 2 hookworms : *Ancylostoma duodenale* and *Necator americanus***
- **740 million people infected in tropics/subtropics**
- **Causes more morbidity than other geohelminths**
- **Often in huge number attached causing hookworm aneamia, iron deficiency anaemia**
- ***N.americanus* is the predominant hookworm of Sub-Saharan Africa, Southern Asia, Caribbean America**
- ***A. duodenale* is predominant in EU, North Africa, China, Japan, Latin America, Northern India**

Hookworm - Aetiology

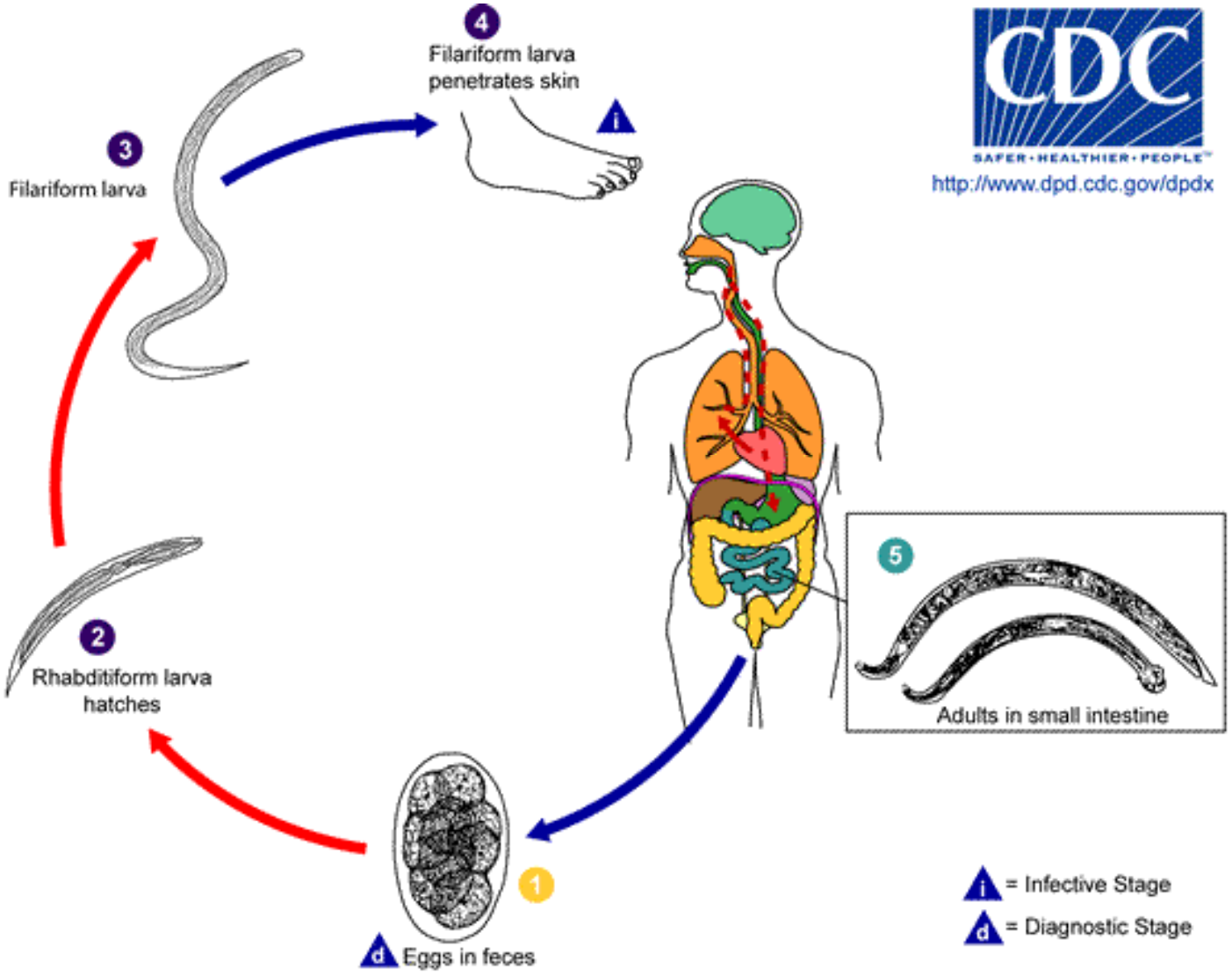
A. duodenale	*N. americanus*
♂ and ♀ have buccal capsule with teeth	Same but smaller with cutting plates
♂ 1 x 0.5 cm	♂ smaller, more slender
♀ 1.2 x 0.6 cm	♀ smaller
25-35.000 eggs/d	6-20.000 eggs/d
Egg: 50x35 µm, elliptical, transparent shell	Egg: 70x40 µm
Average stay in host : 1y	Life duration: 3-5 y
♂ with copulatory bursa	-

Anterior end of an adult of *Ancylostoma caninum*

Hookworm egg in unstained wet mount



Hookworms - Cycle



A. duodenale & *N. americanus*

Differences in life cycle

A. duodenale	*N. americanus*
Live 1 to 3 years	**Live 3 to 10 years**
Can also infect by ingestion	**Infects only through skin**
No larval development in lungs	**Larvae grow and develop in lungs**
Can remain as L in host for months before developing to adult	

Ancylostoma – anterior end with teeth

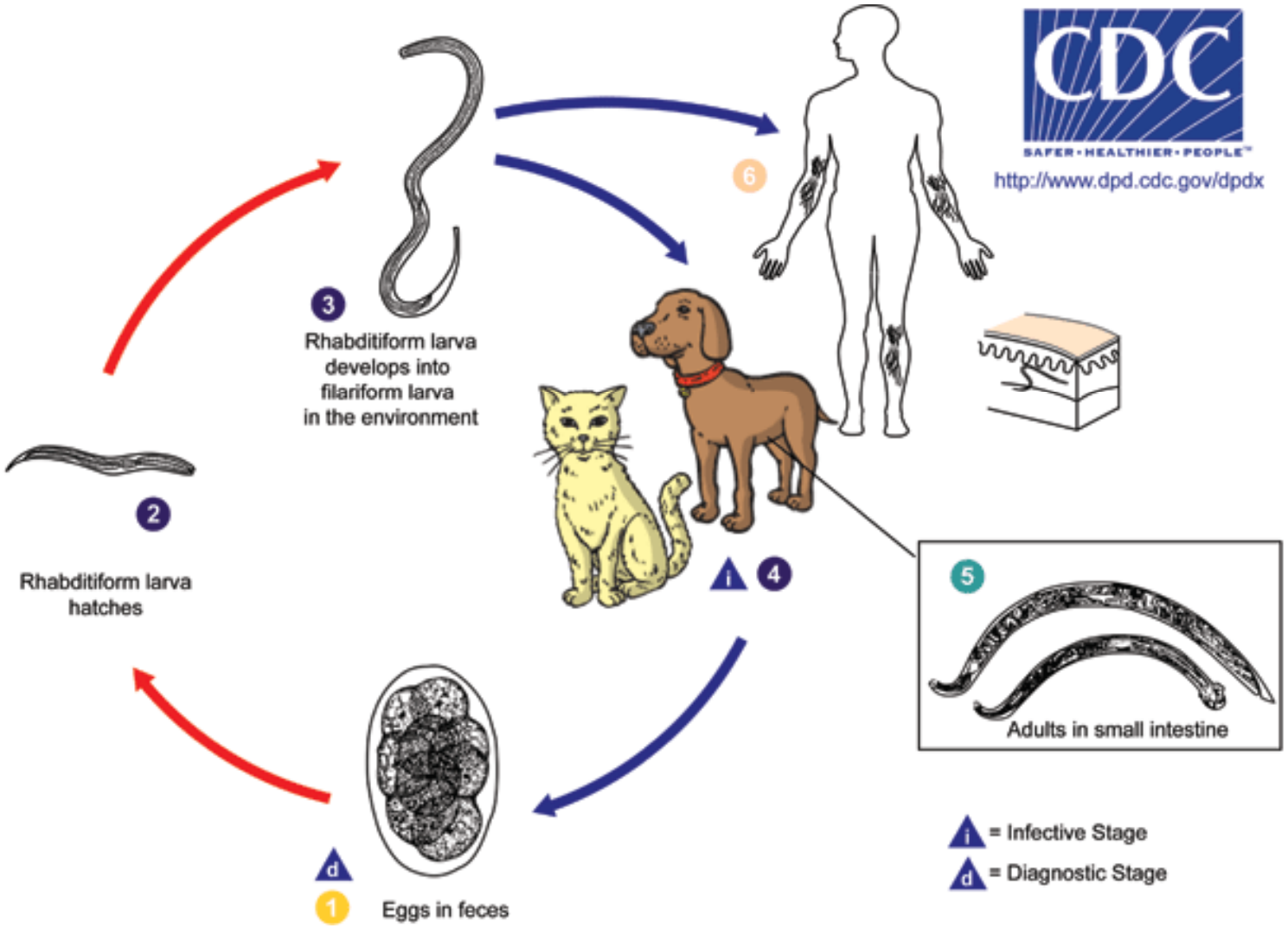
Hookworms - Cutaneous larva migrans

Hookworms - Diagnostic

- **Microscopic identification of eggs in the stools**
- **Concentrate using the formalin–ethyl acetate sedimentation technique**
- **Kato-Katz can be used for quantitative assessments of infection**
  www.tropeduweb.ch/Parasitology_Methods_PDF/8_Stool_Kato-Katz.pdf
- **Examination of the eggs cannot distinguish between *N. americanus* and *A. duodenale.* Filariform larvae of the 2 species can be differentiated in a fecal smear on a moist filter paper strip**

Life Cycle (cutaneous larval migrans)

A. braziliense and *A. caninum*

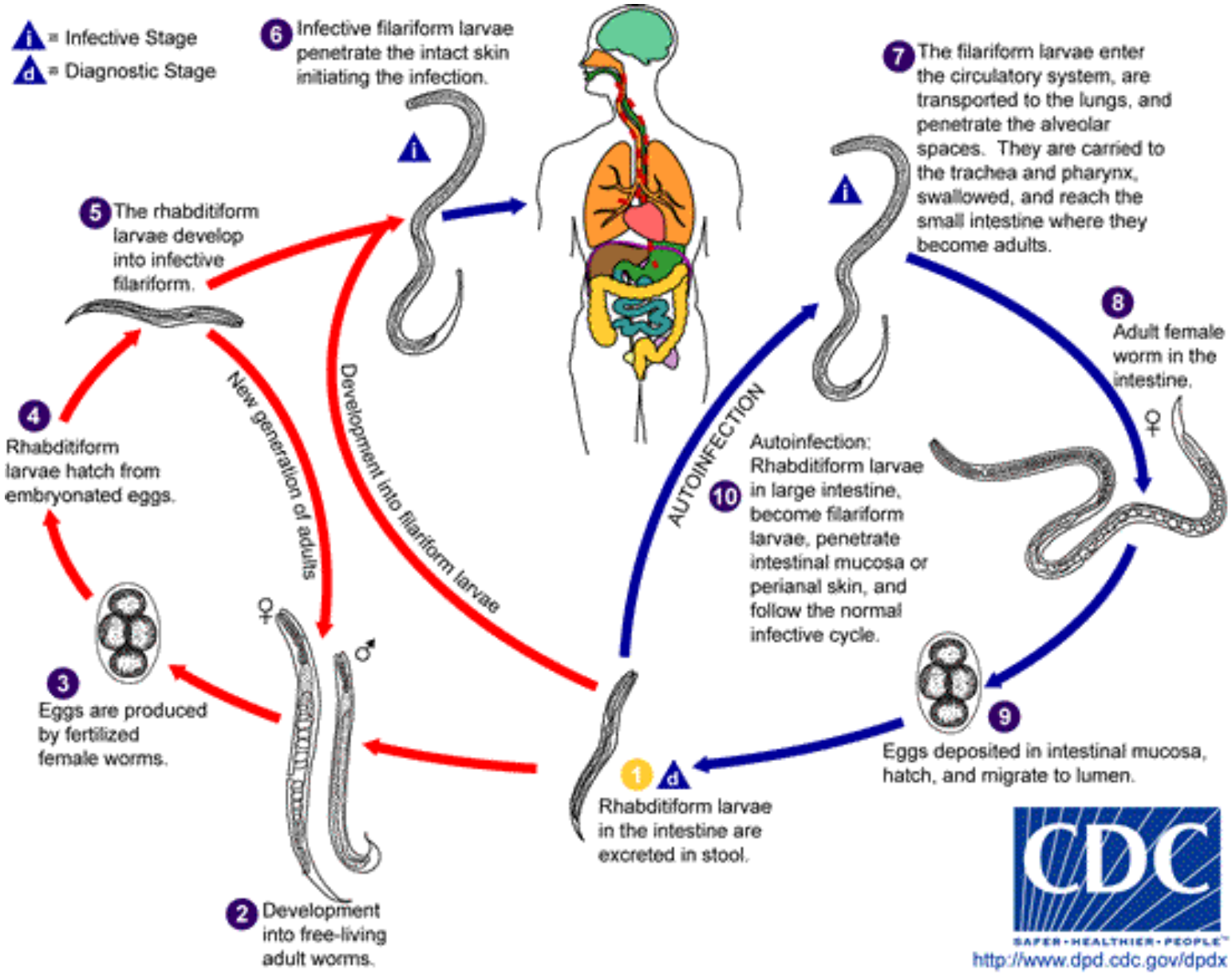


Type 2: PENETRATION OF THE SKIN

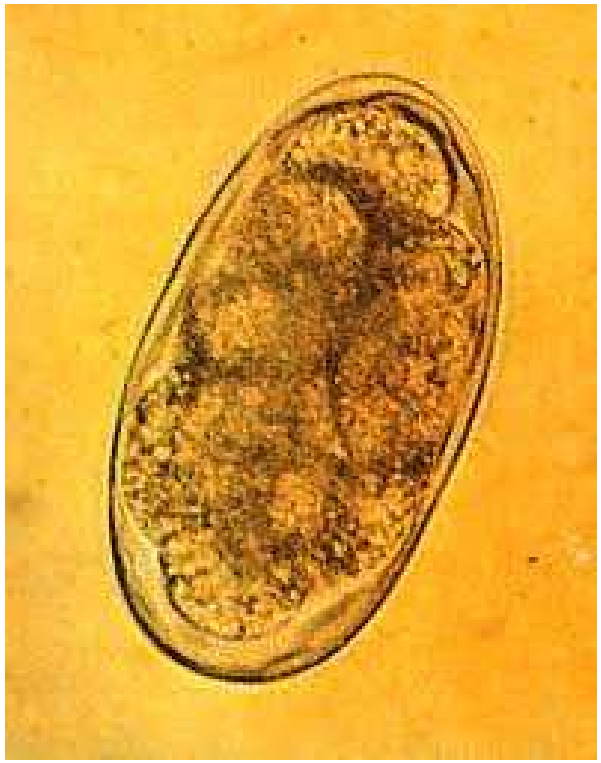
Strongyloidiasis (*Strongyloides stercoralis)*

- Tropical and subtropical areas, but cases also occur in temperate areas.
- More frequently found in rural areas, institutional settings, and lower socioeconomic groups
- Males grow to only about 0.9 mm in length
- Females can be anywhere from 2.0 to 2.5 mm
- Both genders also possess a tiny buccal capsule and cylindrical esophagus without a posterior bulb
- It has become a serious problem in individuals receiving immunosuppressive treatment, or immuno-compromised
- Three developmental forms: adult, rhabditiform larva & filariform (infective) larva

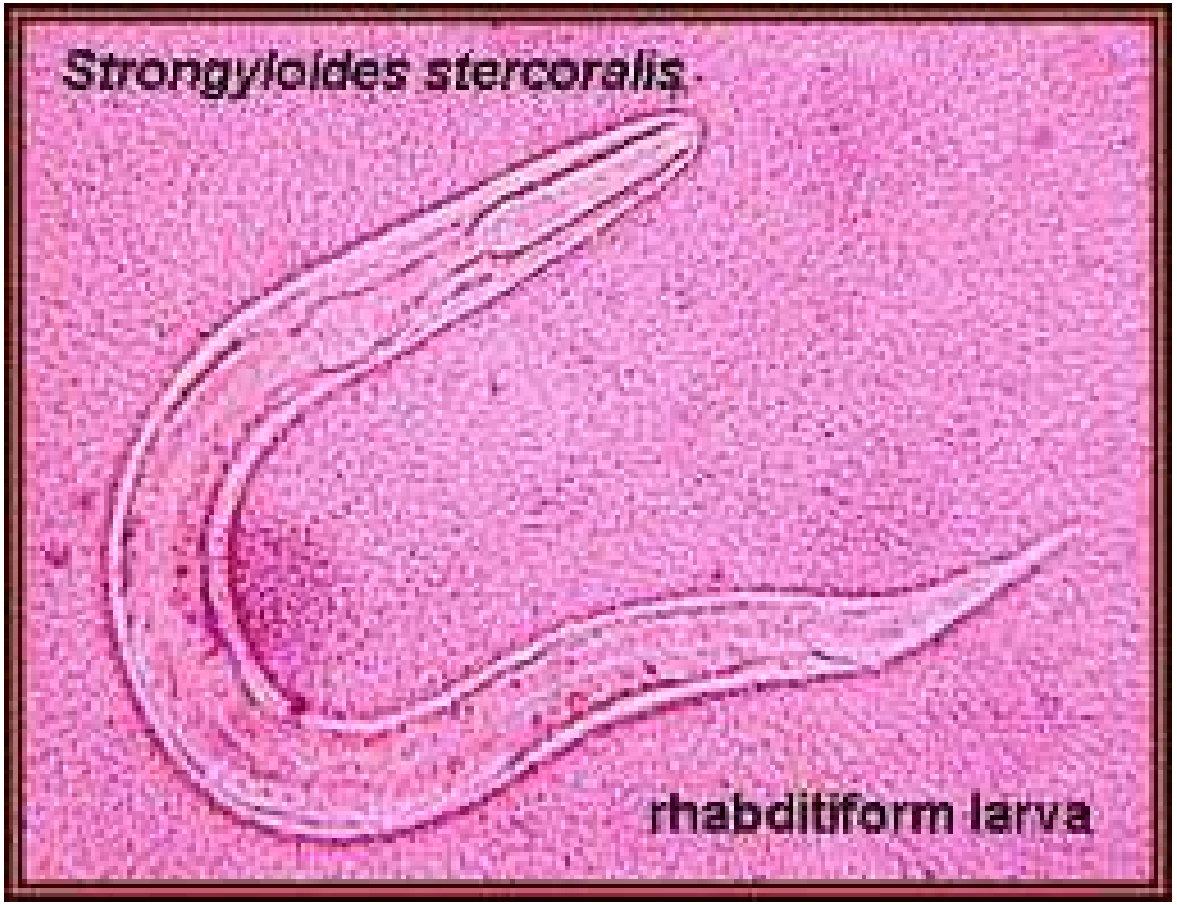
S. stercoralis – Life cycle



S. stercoralis – Egg



S. stercoralis – rhabditiform larva



Longitudinal-section of a larva of *S. stercoralis* from an intestinal biopsy specimen

S. stercoralis – Diagnosis

- Diagnosis rests on the microscopic identification of larvae (rhabditiform and occasionally filariform) in the stool or duodenal fluid.
- For stools:
    - directly
    - after concentration (formalin-ethyl acetate)
    - after recovery of the larvae by the Baermann funnel technique
    - after culture by the Harada-Mori filter paper technique
    - after culture in agar plates
- Examination of serial samples may be necessary, and not always sufficient, because stool examination is relatively insensitive
- The duodenal fluid can be examined using techniques such as the Enterotest string or duodenal aspiration
- Larvae may be detected in sputum from patients with disseminated strongyloidiasis

L_1 larva (rhabditiform) 250-300 µm

L_3 larva (filariform) 500-600 µm

S. stercoralis – Management

- **The drug of choice for the treatment of uncomplicated strongyloidiasis is Ivermectin**
- **Albendazole, Mebendazole are alternatives**
- **All patients who are at risk of disseminated strongyloidiasis should be treated**
- **There is often a decrease in the efficacy of treatment in persons co-infected with HTLV-1**