MLS-409-LEC | CLINICAL PARASITOLOGY - LECTURE

MIDTERMS - LESSON 1: Introduction to Medical Parasitology
LECTURER/S: Mr. Francis Ian L. Salaver, RMT, MD | Mr. Edilon Liwag, RMT
SOURCE/S: **Introduction to Medical Parasitology**| Belizario, V.Y., De Leon., W. U. "Medical Parasitology in the Philippines" 3rd ed., p. 80 | Markell and Voge's Medical Parasitology 9th Ed. | Onsite Discussion

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INTRODUCTION

PARASITOLOGY

It is a branch of medical microbiology that deals with the study of eukaryotic unicellular and multicellular organisms and their medical significance in human communities.

Branch of science that is concerned with the study of the biology of parasites and parasitic diseases, including the distribution, biochemistry, physiology, evolution and clinical aspects of parasites, including the host response to these agents.

Medical parasitology or Human parasitology is restricted to studying those parasites that are living in or on the body of human, their geographic distribution, the diseases caused by them, clinical picture and the response generated by human against them.

KINGDOM ANIMALIA	→ **Metazoans** = multicellular (invertebrates) ● Example: Helminths (parasitic worms)
KINGDOM PROTISTA	→ **Protozoans** = unicellular (microorganisms) ● Example: Entamoeba histolytica (amoeba)

TROPICAL MEDICINE

- **Tropical Medicine** is a branch of medicine which deals with tropical diseases and other special medical problems of tropical regions.
- A **tropical disease** is an illness, which is indigenous to or endemic in a tropical area. Many tropical diseases are parasitic diseases.

BIOLOGICAL RELATIONSHIP

SYMBIONT → *Any organism that spends a portion or all of its life cycle intimately associated with another organism of a different species*
- The relationship is called **SYMBIOSIS** (symbiotic relationships).

→ **ECOSYSTEM =** co-exist, survival of species - nourishment.
- Predator and prey, scavengers

SYMBIOSIS → a relationship in which 2 species live in close association with one another

3 TYPES OF SYMBIOSIS

COMMENSALISM	an association in which the commensal takes the benefit without causing injury to the host. Organism #1 → benefits from Organism #2 Organism #2 → remains unharmed because Organism #1 is not causing injury to it. *Best exemplified by the established by the **remora fish** and the **shark***  Remora fish can also eat scraps of food that the shark is eating. Another example = Carabao + Birds
MUTUALISM	an association in which both partners are metabolically dependent upon each other and one cannot live without the help of the other None of the partners suffers any harm from the association.  Best exemplified by clownfish and sea anemone. → Sea anemone contains toxins and they prey on fish and crustaceans – so whenever fish would come close to them, fish would get the toxins and become paralyzed, and sea anemone can feed on the fish. ● The only sea animal that

	can tolerate the toxin of the sea anemone is the clownfish. ● This is why the clownfish can use the sea anemone as its habitat. Therefore, the sea anemone can provide protection to the clownfish from its protectors. ● On the other hand, the sea anemone would also benefit from the clownfish – because it can get nutrients from the **waste materials** / feces from the said fish. Another example: termites + flagellates 1. Termites cannot digest cellulose because they cannot synthesize and secrete cellulase 2. Myriad flagellates living in their gut produce cellulase and digest wood eaten by the host 3. The molecules derived from flagellates metabolism is used by the host.
PARASITISM	an association where one of the partners is harmed and the other lives at the expense of the other. → E.g. Worms like Ascaris lumbricoides reside in the gastrointestinal tract of man, and feed on important items of intestinal food causing various illnesses.  **HOST →** an organism that harbors and provides sustenance for another organism. **PARASITE →** an organism that obtains its nourishment at the expense of another organism which it affects adversely, but does not immediately kill them.

A wasp is seen injecting its egg into the body of a caterpillar, the eggs eventually hatch and release larva, and for the larva to survive, will begin eating the caterpillar from the inside

- This causes the caterpillar to eventually *die*

Once the larva are ready to evolve to the adult stage of the wasp, they will emerge **out of the body** of the caterpillar, thereby killing the caterpillar.

The Life Cycle of the Green-Banded Broodsac *(Leucochloridium paradoxum)*

- This is a parasite of birds

1. In the bird, the parasite moves to the digestive system and becomes an adult. The adult parasite sexually or asexually reproduces and lays eggs
2. Oval shaped eggs are excreted by a bird in its feces.
3. The fecal matter is eaten by a snail. In doing so the snail is now infected with the parasite.
4. A mobile larva hatches from the egg, and travels to the digestive gland of the snail.
5. The larva grows into a long tube called a broodsac. This broodsac contains hudreds to thousands of the next larval stage of the parasite.
6. The parasite leaves the digestive gland and pushes into the eye stalks of the snail. The parasite takes over the rudimentary brain of the snail
7. No longer afraid of sunlight because of the parasite the snail leaves the safety of the surrounding foliage and travels up into highly exposed areas. The parasite pulsates to mimic a caterpillar.
8. The pulsating parasite is eaten by a bird, and becomes infected with the parasite
9. The snail is often not killed. its eaten eye regenerates. It remains infected for the rest of its life, and continues to infect other birds.

GENERAL TYPES OF PARASITES

ACCORDING TO HABITAT

ECTOPARASITES	Parasites that live **outside** the host's body **INFESTATION – presence of ectoparasites in the body** Example: lice, ticks, mites, etc.
ENDOPARASITES	Parasites that live **inside** the host's body **INFECTION – presence of endoparasites in the body** Example: entamoeba histolytica

ACCORDING TO ITS RELATIONSHIP WITH THE HOST	
OBLIGATE	parasite that take up permanent residence in a host and are completely dependent upon them. **Ex. Ascaris lumbricoides**
FACULTATIVE	parasites that are not normally parasitic (free) but can become when they accidentally enter the host. organism that exhibits both parasitic and non-parasitic stages of living and hence does not absolutely depend on the parasitic way of life, but is capable of adapting to it if placed on a host. **Ex. Strongyloides stercoralis**
ERRATIC OR ABERRANT PARASITE	one that wanders in to an organ in which it is not usually found. Example: Ascaris lumbricoides in humans Enatmoeba histolytica reaching liver from intestine
INTERMITTENT OR TEMPORARY	parasite is free living during part of its existence and seeks it's host to obtain nourishment. those that only visit the host from time to time for feeding **Ex. Mosquitoes (malaria and plasmodium)**
SPURIOUS/ COPROZOIC PARASITE	organisms that have been swallowed and passed through the alimentary tract without causing any effect
HYPERPARASITE	parasite that parasitizes other parasites
INCIDENTAL	is one that establishes itself in a host in which it does not ordinarily live. **Ex. Anisakis**
PERMANENT	parasite that remains on or in the body of the host from early life until maturity, sometimes for it's entire life.
PSEUDO PARASITE	artifacts mistaken as parasites, such as pollen, hairs.

GENERAL TYPES OF HOST	
DEFINITIVE HOST	is one in which the parasite reaches sexual maturity reproduction. (harbours the adult stage of the parasite) **Example: Humans**
INTERMEDIATE HOST	is one that requires for parasite development, but does not reach sexual maturity. (asexual/larval stage) in some cases, larval development is completed in two different intermediate hosts, referred to as first and second intermediate hosts. In some cases, larval development is completed takes place in soil and water and there is no need for an intermediate host **Ex. Snails, fish**
PARATENIC/ TRANSPORT HOST	is one in which the parasite does not undergo any development, but remains alive and infective to another host. a host that serves as a temporary refuge and vehicle for reaching an obligatory host, usually the definitive host, i.e. it is not necessary for the completion of the parasites life cycle. **Ex. Prawns, aquatic plants** Example: Paragonimus westermani Diphyllobothrium latum
RESERVOIR HOST	any organism that harbours an infection that can be transmitted to humans. a host that makes the parasite available for the transmission to another host and is usually not affected by the infection. **Ex. Rodents, insects** **Leishmania donovani**
NATURAL HOST	a host that is naturally infected with certain species of parasite.
ACCIDENTAL HOST	a host that is under normal circumstances not infected with the parasite. Accidental parasite: Echinococcus granulosus

LIFE CYCLE

- Many parasites complete their developmental cycle in a single host species (monoxenous life cycles)
- Other parasites require multiple host species (heteroxenous life-cycles)

- Definitive hosts
- Intermediate hosts

DIRECT LIFE CYCLE	parasite does not require intermediate host to complete its life cycle
INDIRECT LIFE CYCLE	parasite requires the presence of an intermediate host to complete its life cycle

EPIDEMIOLOGY

- Is the scientific study that presents the pattern of disease in a given community and the study of those factors influencing its presence or absence.
  - Ex. Characteristics of various pathogens, nutritional status, inadequate sanitation procedure, lack of immunization.

PARAMETERS OF EPIDEMIOLOGY

INCIDENCE AND MORBIDITY RATE	Is the number of new cases of disease per block of population in a specific time period (Sickness/Illness rate
PREVALENCE	is the number of cases of disease in existence at any given time in that population
MORTALITY RATE	is the ratio number of people who died of a particular disease during a specified period per a specified population. (death rate)

CLASSIFICATION OF DISEASE

ACCORDING TO THE MANNER IN WHICH THEY ARE TRANSMITTED IN A GIVEN POPULATION OR COMMUNITY	
SPORADIC	a disease that occurs occasionally in a community or population of a particular geographic area.
ENDEMIC	a disease that is always present to greater or lesser degree within the population or community of a particular geographic area.
EPIDEMIC	a disease that attacks a larger number of persons in a community within a relatively short period of time.
PANDEMIC	A disease that becomes an epidemic in great number of countries at the same time.
ZOONOTIC	Infectious diseases that humans acquire from animal sources. (zoonosis)

GLOBAL PREVALENCE OF SOIL-TRANSMITTED HELMINTH INFECTION

- Globally, an estimated 438.9 million people were infected with hookworm
- In 2010, 819.0 million with A. lumbricoides
- 464.6 million with T. trichiura.
- Most people lived with disability attributable to STH, 65% were attributable to hookworm, 22% to A. lumbricoides and the remaining 13% to T. trichiura.
- The vast majority of STH infections (67%) (68%) occurred in Asia.
- WHO-2013 states that there were estimated 207 million new cases of parasitosis
- Resulting in 627,000 deaths, 90% of deaths occurred in Sub-Saharan Africa. Over 90% of deaths are children under the age of five
- Protozoan infection such as Giardia, Cryptosporidium, Entamoeba caused an estimated 60 million cases of childhood diarrhea
- Schistosomiasis is estimated to infect 200 million people

DISABILITY OF SOIL-TRANSMITTED HELMINTH INFECTION (STH)

- Abdominopelvic problems and symptoms occurs in 100% of individuals
- Wasting especially among children- stunted growth and malnourished, affecting their cognitive development and severely impair the ability to achieved their full socioeconomic potential
- Anemia Hook worm
- Death attributable to heavy worm burden
- Systemic invading vital organs of the body

GENERAL TYPES OF DISEASE

COMMUNICABLE	is one whose causative agent is directly or indirectly transmitted from host to host.
CONTAGIOUS (under Communicable)	is a disease that is easily spread directly from person to person.
NON-COMMUNICABLE	is one whose causative agent either normally inhabits the body. Only occasionally producing a disease or resides outside it, producing a disease only when introduce into the body. (wound or injury)

EFFECTS OF PARASITES ON HOSTS

MECHANICAL INJURY	may be inflicted by a parasite by means of pressure as it grows larger Example: Hydatid cyst causes blockage of ducts such as blood vessels producing infarction.
Under direct effects of the parasite on the host	
DELETERIOUS EFFECT OF TOXIC SUBSTANCES	Example include the tissue damaging enzymes of E. histolytica

DEPRIVATION OF NUTRIENTS, FLUIDS AND METABOLITES	parasite may produce disease by competing with the host for nutrients. Diphyllobothrium latum competes with the host for Vitamin B12 
Indirect effects of the parasite on the host	
EXCESSIVE PROLIFERATION OF CERTAIN TISSUES DUE TO INVASION BY SOME PARASITES CANAL CAUSES TISSUE DAMAGE IN MAN	e.g. fibrosis of liver after deposition of the ova of Schistosoma

MODE OF TRANSMISSION

DIRECT CONTACT	refers to the spread of infection from person to person or from lower animals to humans. (Orifice) ● Sexual contact - Trichomonas, STI/STD ○ Pubic louse ○ Trichomonas vaginalis ○ Entamoeba histolytica ● Blood transfusion - Malaria, babesia spp., hepatitis, HIV, Syphilis ● Placental transmission - toxoplasmosis, Syphilis, Hepatitis, HIV ● Droplet infection- talking, laughing, sneezing, coughing. TB, Measles, and Diptheria
INDIRECT CONTACT/ PERORAL	refers to the spread of causative agent(infection/infestation) by conveyors. (mouth). ● Food ● Air/dust ○ Air-borne transmission ○ Enterobius vermicularis “kigwa” ● Contaminated fingerS ● Fomites
SKIN PENETRATION	Example Schistosoma species and Hookworms
ANIMAL TRANSMISSION (ZOONOSIS)	· Direct contact with animals · Contamination of food with discharges · Rodent vectors · Consumption of animal products that had the disease.
INSECT CARRIERS	· Transmit infection through **MECHANICAL/BIOLOGICAL** means **BIOLOGICAL VECTOR** ● An organism which not only transports a pathogen but also plays a role in the life cycle of the pathogen (virus inside of mosquito, bacteria inside of tick) **MECHANICAL VECTOR** ● An organism which only transports a pathogen (fly)
VERTICAL TRANSMISSION / TRANSPLACENTAL	Toxoplasma gondii

PORTAL OF ENTRY

MOUTH	Ingestion
SKIN	active penetration - Hookworm (filariform) - Trematodes (cercaria)
GUT	Trichomonas vaginalis
TRANSPLACENTAL	Toxoplasma gondii
TRANSMAMMARY	Strongyloides stercoralis
INTRANASAL	Naegleria fowleri

PORTALS OF EXIT

STOOL/FECAL MATERIAL	
URINE	Trichomonas vaginalis, Schistosoma heamatobium
BLOOD	Malaria
SPUTUM	Paragomus westermani
BIOPSY	● Muscle - Trichinella spiralis, Taenia solium, Taenia saginata ● Rectal - amoeba

FOUR PERIODS IN THE COURSE OF AN INFECTION

INCUBATION PERIOD	period of time **between the entry and the onset of symptoms** **CLINICAL INCUBATION PERIOD** ● Time between infection and evidence of symptoms **BIOLOGIC INCUBATION PERIOD** ● Time between infection and acquisition of the parasite as demonstration of the infection
PRODROMAL PERIOD	period of weakness
PERIOD OF ILLNESS	the time during which the patient experiences the typical symptoms associated with the particular disease
CONVALESCENT PERIOD	period of recovery

HOW DO PARASITES EVADE IMMUNE RESPONSES?

1. **SURFACE TURNOVER**

- Schistosoma mansoni
- Shed their teguments in abundance can neutralize antibody response at a distance away from the parasite.

SURFACE TURNOVER: RESISTANCE TO IMMUNE RESPONSE

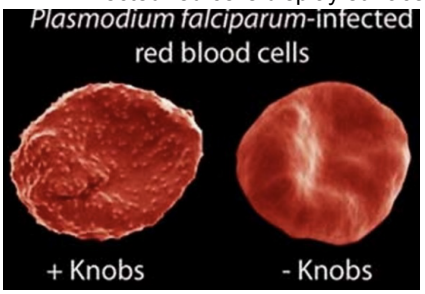
RESISTANCE TO IMMUNE RESPONSE	➜ Cuticle covering of worms can prevent phagocytosis and protect them from cytokines and complement proteins ➜ Some shed the contents of their outer covering to neutralize antibody at a site away from the parasite
IMMUNE SUPPRESSION	➜ Entamoeba histolytica produces suppressor factor which inhibits movement of phagocytes
ANTIGENIC VARIATION	➜ Some parasites can change the antigenic compositions of their surfaces
HOST MIMICRY	➜ Schistosoma will coat it self with the host antigen

2. **ANTIGEN DISGUISE**

- Adult Schistosoma cover themselves with host proteins to be considered as self and will not be attacked by the immune factors.

RESISTANCE

- **INTRACELLULAR SEQUESTRATION**
  - Toxoplasma, Trypanosoma and Leishmania parasites multiply within the cytoplasm of macrophages thus they can evade phagocytosis
  - Plasmodium falciparum can make the infected red cells display surface knobs



FACTORS THAT PROMOTE PARASITIC INFECTIONS

1. Source of infection
2. Mode of transmission
3. Susceptible host - age, nutritional status, heredity, stress, socioeconomic status

MECHANICS OF PARASITIC TRANSMISSION

- Stages in the organisms development that can lead to infection.

SOIL TRANSMITTED	undergoes development in the soil to reach the infective stage Example: Ascaris, Trichuris
INTERMEDIATE HOST/ INCIDENT HOST TRANSMITTED	undergoes development in IH to react to the infective stage Example: Snail, crabs, prawns, aquatic plants
ARTHROPOD TRANSMITTED	undergo development in the insect to reach their infective stage. Example: Malaria and Filaria → mosquito Leishmania → sandfly, Trypanosomes → Am-reduviid bug and African tse-tse fly
FOOD TRANSMITTED	undergo further development in animals to reach their infective stage. Example: Tape worms → pork and beef C. sinensis → fish
AUTO INFECTION	infecting oneself Occurs when an infected individual becomes his direct source of infection Example: Pinworm (Enterobius vermicularis)
SUPERINFECTION OR HYPERINFECTION	Happens when the already infected individual is further infected with the same species leading to massive infection with the parasite Female adult worms can be

	OVIPAROUS or LARVIPAROUS
PARTHENOGENESIS	Form of reproduction in which an egg can develop into an embryo without being fertilized by a sperm. Parthenogenesis is derived from the Greek words for "virgin birth.
CONTACT TRANSMITTED	no further stage of development required Example: pinworm, Trichomonas vaginalis

TREATMENT

DEWORMING	Use of antihelminthic drugs in an individual or a public health program
CURE RATE	Refers to the number of previously positive subjects found to be egg negative on clinical samples using standard procedure
EGG REDUCTION RATE	Refers to the percentage fall in the egg counts on clinical samples after deworming
SELECTIVE TREATMENT	Individual-level deworming with selection for treatment based on diagnosis of infection and severity
TARGETED TREATMENT	Group level deworming where the group to be treated (w/out prior diagnosis) may be defined by age, sex or other social characteristics irrespective of infection status.
UNIVERSAL TREATMENT	Population-level deworming in which the community is treated irrespective of age, sex, infection status or other social characteristics
PREVENTIVE CHEMOTHERAPY	Regular, systematic, large scale intervention involving the administration of one or more drugs to selected population with the aim or reducing morbidity and transmission

TYPES OF STAGES

INFECTIVE STAGE	The stage in the life cycle at which the parasite is able to initiate an infection to its host
DIAGNOSTIC STAGE	is the life cycle stage that exits the definitive host.

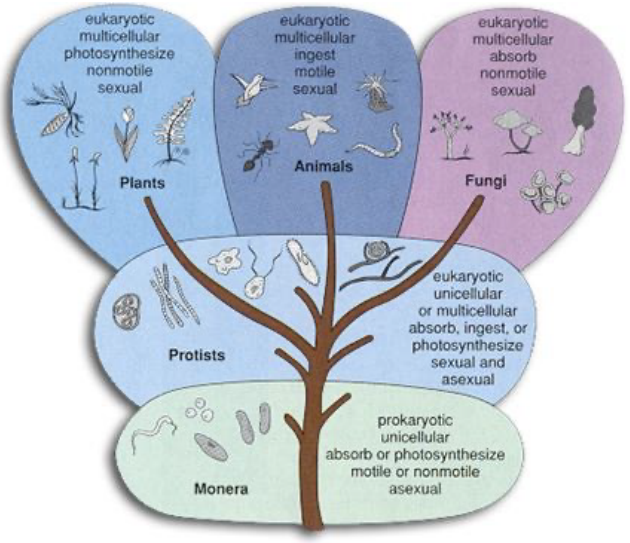
TAXONOMY

- The science of classification of living organisms

CLASSIFICATION	arrangement of organism into taxonomic groups (taxa) on the basis of similarities and relationship. 1. Domain 2. Kingdom 3. Phylum 4. Class 5. Order/Suborder 6. Family 7. Genus 8. Species ****BASED SA INTERNET, PERO ACCORDING SA PPT NI SIR - NAUNA ANG KINGDOM, FOLLOWED BY DOMAIN/PHYLUM**
NOMENCLATURE	assignment of names to various "TAXA" according to international rules. ● Binomial system- **Carrolus Linneaus** in the 18th Century ○ Sweden ● Genus – first letter capital ● Specie – First name- first letter capital,second name-small letters

ROBERT HARDING WHITTAKER

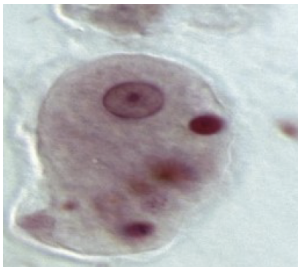
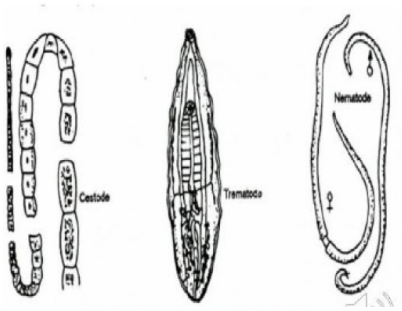
a renowned plant ecologist and the first to proposed the 5 kingdom taxonomic classification of the world's biota in 1969.



PLANTS	- eukaryotic - multicellular - photosynthesize - nonmotile - sexual
ANIMALS	- eukaryotic - multicellular - ingest

	- motile - sexual
FUNGI	- eukaryolic - muiticellular - absorb - nonmotile - sexual
PROTISTS	- eukaryotic - unicellular or multicellular - absorb, ingest, or photosynthesize sexual and asexual
MONERA	- prokaryotic - unicellular - absorb or photosynthesize - motile or nonmotile - asexual

CLASSIFICATION OF PARASITES

PROTOZOANS	 ● Unicellular eukarvotes ● Has nucleus, cytoplasm, membrane and organelles ● Some have locomotory apparatus
HELMINTHIC PARASITES	● Roundworms (Nemahelminths) ○ NEMATODA ● Flatworms (Platyhelminths) ○ Flat and Segmented - CESTODA (tapeworms) ● Flat and Unsegmented - TREMATODA (flukes) 
ARTHROPODS	also serve as vectors and transmit parasites through their bites. Examples are agents of malaria, filariasis, leishmaniasis,

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MIDTERMS - LESSON 2: Phylum Nematoda
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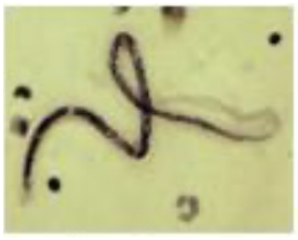
TOPIC OUTLINE

PHYLUM NEMATODA / NEMATHELMINTHES

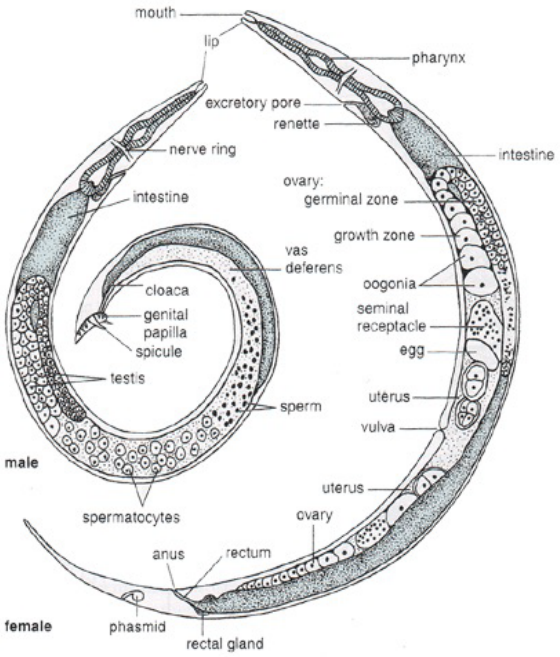
	***RHABDITIS***

	***DRACUNCULUS***
	***ASCARIS***
	***ENTEROBIUM***
	***TRICHURIS***
	***LOA LOA***

ROUNDWORMS

- The most abundant animal on earth
- ***DIOECIOUS*** **–** male and female worm (larger) a few mm-meter in length.
- Free living in soil, marine and freshwater habitat.
- 500,000 species
- Nonsegmented, cylindrical, tapered at both ends
- **BODY COVERING:** cuticle with longitudinal muscles

- **BODY CAVITY**: *(pseudocoel)* complete digestive tract with both oral and anal openings.
- Mouth is provided with spines, hooks, cutting plates, stylets, or other structures for attachment or penetration of tissue
- Reproductive organs are tubular and lie coiled in the body cavity.
- **Fertilization is internal.**
- Males have chitinized spicules for copulation
- **SENSORY ORGANS:** anterior end (amphids), posterior end-caudal (phasmids) nerve endings that act as chemoreceptors.
- **PAPILLAE:** sensory hairs at the head and tail
- ***Separate classes of nematodes***
    - Aphasmidea
    - Phasmidea
- **LIFE CYCLE:** Egg stage, Larval stage, Adult stage
- **ADULT FEMALE:** Oviparous, Vivaparous, Parthenogenetic



GENERAL CHARACTERISTICS		HABITAT	
APHASMIDS	Trichinella spiralis Trichuris trichiura Capillaria philippinensis	***SMALL INTESTINE***	Ascaris lumbricoides Strongyloides stercoralis Hookworms Capillaria philippinensis Trichinella spiralis
PHASMIDS	Ascaris lumbricoides Strongyloides stercoralis Hookworms Enterobius vermicularis Filarial worms	***LARGE INTESTINE***	Trichuris trichiura Enterobius vermicularis
		TISSUE NEMATODES	Trichinella spiralis Wuchereria bancrofti Brugia malayi Onchocerca volvulus Loa loa Dirofilaria immitis Gnathostoma spinigerum
		LARVA MIGRANS IN MAN	Dracunculus medinensis

		Angiostrongylus cantonensis Ancylostoma caninum Ancylostoma braziliense

TRICHINELLA SPIRALIS

TRICHINELLA SPIRALIS (p. 305)	OTHER ADDITIONAL INFORMATION
● Parasite of the carnivorous mammals. Smallest nematode parasite of humans ● **"Trichina worm"** – minute and threadlike ● Common in rats and swine fed with uncooked garbage and slaughterhouse scraps ● Occurs worldwide. Especially in areas where raw garbage containing pork scraps is fed to hogs ● Highest incidence rate is now reported in China -- 10,000 cases annually. ○ Spain, France, Italy, Yugoslavia, Middle East, Africa. ● **Discovered in 1835 by James Paget.** Name by Prof. Richard Owen ○ Seen by first-year medical student James Paget - however, it was named by Richard Owen ○ **Five distinct species of Trichinella** are now recognized – **Trichinella spiralis is the most important** - **Parasite of carnivorous mammals** - Especially common in rats and swine-fed uncooked garbage and slaughterhouse scraps - May *occur* in humans who consume **uncooked pork and most common in groups who make a practice of consuming raw pork products (salami, wurst)** **TRICHINELLA** → cosmopolitan in distribution → occurs much more frequently in areas where raw garbage containing pork scraps is fed to hogs **STAGES OF INFECTION** 1. Initiated by the consumption of raw/undercooked pork containing encysted larvae 2. Larvae excyst after cysts are digested and penetrate into the intestinal mucosa → develop into adult worms within 30-40 hours. 3. Mating of males and females takes place as soon as worms are mature, larvae may be produced 3 days after fertilization. 4. Within 5 days, larvae grow to maturity and **larval deposition** begins – this continues for as long as female worms remain in the intestine 5. Duration depends on the number of worms present in the intestine AND the immune status of host 6. Intestinal phase persists in congenitally **athymic** (nude) mice much longer than those heterozygous for the ahytmic gene a. T cell-dependent antigen is **NECESSARY** for protection against intestinal phase for infection b. Human intestinal infections last as long as **54 days;** avg. duration is shorter 7. Once deposited in the mucosa, larvae **enter lymphatic vessels** and gain access to the general circulation	● As readily be considered with the intestinal helminths. ● **INTESTINAL INFECTION** → adult worms are found in the mucosa ○ Transitory ○ Asymptomatic ● Phase of migration and encystment of larvae in muscles is **PROLONGED** and **FREQUENTLY ACCOMPANIED** by serious symptoms. **DIFFERENT SPECIES OF TRICHINELLA ASIDE FROM SPIRALIS** **1.) Trichinella nativa** – occurs in arctic and subarctic zones ● High pathogenicity ● High resistance to freezing **2.) Trichinella nelsoni** – occurs in tropical Africa ● Intermediate in pathogenicity **3.) Trichinella britovi** – occurs in temperate Paleoarctic region ● Very low pathogenicity **4.) Trichinella pseudospiralis** – cosmopolitan ● Doest not encyst ● Infectious to birds ● Pathogenicity in humans not well characterized **5.) Trichinella murrelli** – parasite of wild carnivores in North America ● Moderate to severe pathogenicity for humans ● No resistance to freezing **6.) Trichinella zimbabwensis** – parasite of crocodiles in Zimbabwe ● Experimentally infective in mammals including primates ● First species of trichinella to be described from a cold-blooded animal **7.) Trichinella papuae** – parasite of wild and domestic pigs in Papua New Guinea ● Experimentally infective in reptiles

PATHOGENESIS OF TRICHINELLA SPIRALIS

STAGES

1. Intestinal phase of a Trichinella infection ends when adult worms are rejected because of development of **local tissue immunity**
2. Development of immunity CAN be suppressed
    a. Intestinal phase is prolonged by **administration of corticosteroids**

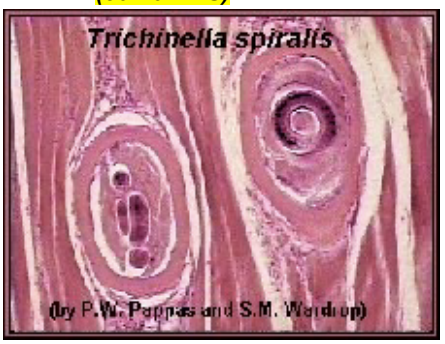
- Primary pathogenic effect of Trichinella comes from **destruction of striated muscle fibers** in which it encysts
- **Vasculitis** may accompany migration of the larvae
    - Accounts for splinter hemorrhages and periorbital edema
    - Certain of the neurologic manifestations of trichinellosis

→ Granulomas that contain Trichinella larvae have been found in **brains of patients** who have died with this infection.

- Clinically apparent trichinal myocarditis is rare.
- Death from trichinellosis may be ascribed to:
    1. myocarditis
    2. encephalitis
    3. pneumonitis

➜ Possible connection between trichinellosis and polyarteritis nodosa is noted and may involve deposition of circulating immune complexes in vessel walls

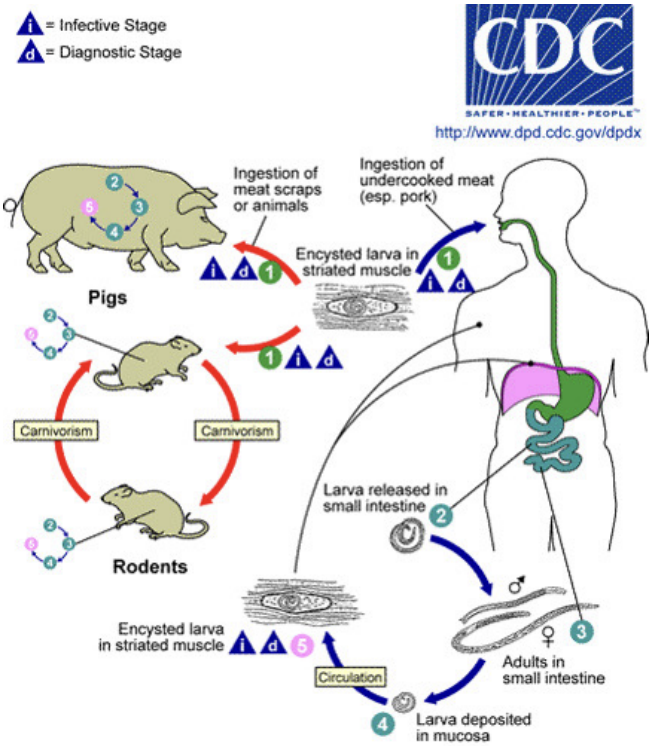
MORPHOLOGY OF TRICHINELLA SPIRALIS

Male adult worm	*Female adult worm*	*Larva-infective stage*
→ **Less than 2mm long** • (1.4-1.6 mm x 40-60 um) → Cloaca is found at caudal end which is **evertible** during **coitus** → Guarded by 2 conspicuous conical papillae which *clasp the female during copulation* → Spicule is absent → Posterior end is curved ventral 	→ Less than 5mm • 3.4 mm x 60-80 um → Vulva opens at the **anterior fifth of the body** → Single uterus which contain the larva → Single ovary found near the posterior end (caudal). 	• 80-120 um long x 5-6 um in diameter (100x6um) • Fully developed larva measures 1mm long • Provided with a spear-like burrowing tip at the anterior end • Readily transported throughout the body. ○ Lymphatic vessel and gain access to the general circulation • They leave the capillaries in striated muscle to penetrate through the sheaths of the muscle fibers. • Larva excyst after the cyst are digested and penetrates the im- develop to adult ***(30-40 HRS)***  They are coiled in a spiral and gradually become surrounded by a sheath derived from muscle fiber **EXCEPT FOR T. PSEUDOSPIRALIS** 

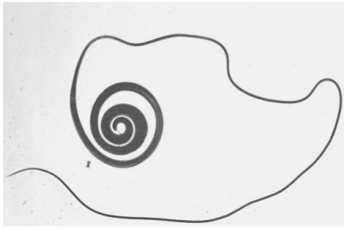
PATHOLOGY OF TRICHINELLA SPIRALIS

DIsease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*
1. Trichinosis 2. Trichiniasis 3. Trichinellosis **Ingestion of carcasses with encysted larva (infectious stage)**	→ Mild to moderately severe symptoms within **2-7 days after ingestion** ● Gastroenteritis ● Diarrhea with or without abdominal pain for several weeks ● Weakness → Fever and eosinophilia ● Eosinophilia increases rapidly during the early stages and gradually decreases after a period of months → Leukocytosis is common, but not always present → Hyperimmunoglobulinemia E is characteristic → Myositis appears early ● Clinical sign / classical sign ● Circumorbital edema (eyelids) ● RARELY absent in patients who develop clinical symptoms of infection ● Related to the vasculitis responsible for the production but may come later → Photophobia, diplopia, or other visual disturbances may occur → Muscle pain reaches height in 12th-20h day ● Muscles are sensitive to pressure	→ Skeletal muscle biopsy (gastrocnemius) ● Reveals encysting larvae at any time after the first week or infection → Serologic test ● Bentonite flocculation → Knott or membrane filtration ● Concentrates of venous blood during larval migration may demonstrate these organisms ○ Ch 15/16 → Radiographs cannot demonstrate calcified cysts in muscle ● These are seen by xeroradiography → Splinter hemorrhages beneath the nails are seen in a large percentage of patients → CNS involvement ● Acute psychosis ● Meningoencephalitis ● Cerebrovascular accident/brain tumor ➜ These begin to abate in 5th or 6h week ➜ Death may occur 4-6 weeks after infection	● **Mebendazole** → Drug of choice for treatment of trichinellosis → Dosage for adults and children >2yrs is 200~400 mg 3x a day → Followed by 400~500 mg 3x daily for 10 days ● Thiabendazole ● Albendazole → alternative drug → 400mg twice a day for 8-14 days ● Prednisone ➜ Cook meat properly ➜ Maintain proper sanitation ➜ Public education

LIFE CYCLE OF TRICHINELLA SPIRALIS



TRICHURIS TRICHIURA

TRICHURIS TRICHIURA p. 263	**OTHER ADDITIONAL INFORMATION**
• "**whipworm**" - whip-like in appearance - Descriptive- anterior 3/5 is very thin and hair like, houses the esophagus. While the posterior 2/5 is thick and stout resembling the handle of a whip which houses the intestine and reproductive organs. - The thin, almost colorless anterior three fifths of the body consists of the much reduced esophagus - The expanded posterior part of the worm is pinkish gray and contains the intestine and reproductive organs.  Worldwide distribution but common in tropical countries and in regions where sanitation is poor • Prevalence rate is high in **Asia- 50-80%**. Due to defecation onto the soil takes place or when human feces are used as fertilizers. • **Trichuris = Hair Tail**	Infection is acquired by **ingestion of the fully embryonated eggs.** • Requires a period of **10 days** or more outside the body to reach the infective stage. 1. **HATCH IN THE SMALL INTESTINE:** The shell is digested in the *small intestine* a. They develop into larvae 2. **LARVAL DEVELOPMENT AND MIGRATION:** Then passes to the *cecal area (large intestine)* a. They mature into adult worms 3. In heavy infections, worms are found in the *rectum* **DIAGNOSIS OF T. TRICHIURA OVA**  Diagnosis is by demonstration of the characteristic **barrel- or football-shaped eggs** in the feces

PATHOGENESIS OF TRICHURIS TRICHIURA

CLINICAL SIGNIFICANCE

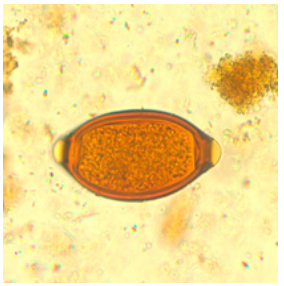
The **anterior ends of the worms**, interlaced in the colonic mucosa (large intestine), apparently **produce little damage** to the host unless present in large numbers

1. **Appendicitis** – brought about by blockage of the lumen of that organ by worms, has been frequently reported
2. **Edema of the rectum** – produced by numbers of worms embedded in that area, is responsible for rectal prolapse
3. **Blood loss** – Attributed per worm, with radioisotope techniques it has been calculated to be approximately 0.005 ml per worm per day
4. **Chronic Dysentery** – Infections of 200 worms or more in children

In many ways **trichuriasis mimics inflammatory bowel disease**, but unlike that condition it is readily curable

MORPHOLOGY OF TRICHINELLA TRICHIURA

Male adult worm	***Female adult worm***	***Ova-infective stage***
➔ **3-3.5 cm in length** ➔ Spicule protrudes through a refractile penial sheath which has a bulbous termination covered with small recurved spines. ➔ Distinguished from the female by its coiled caudal extremities about 360 or more ➔ Adult worm life span = 1 year	➔ **3.5-5.5 cm in length** ➔ **vulva** opens at the anterior end which is the fleshy portion of the body ➔ Has a **single uterus** and **ovary** ➔ Bluntly rounded at the posterior end ➔ Produces 3,000-20,000 eggs daily	➔ **50-54 um x 22-23um in diameter** ➔ Barrel – football-shaped in feces ➔ **Golden brown in color** ➔ **Intralaminar (refractile) prominences** usually referred to as polar plugs at either end. ➔ 3 shells: 1. Chorionic layer 2. Albuminous layer 3. Bile-stained layer ➔ When passed out it is usually immature *(unsegmented)*. **Requires 3 wks in soil to mature.**

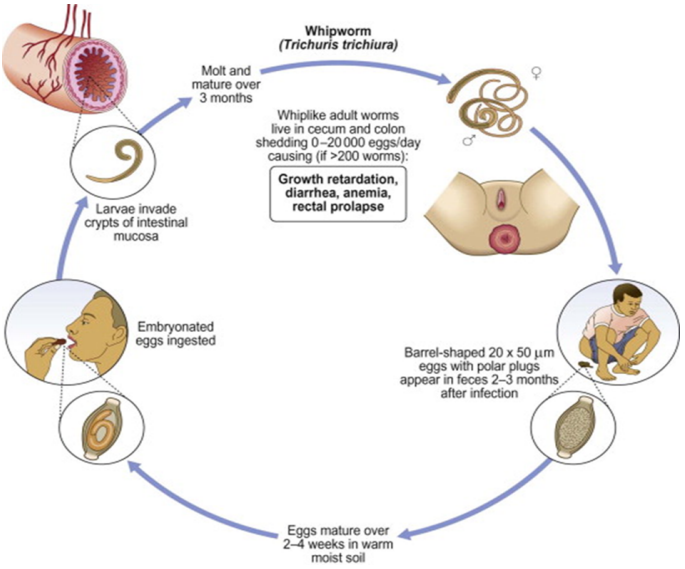
PATHOLOGY OF TRICHURIS TRICHIURA

Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*

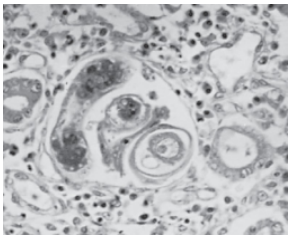
Trichuriasis
 	
Mode of Transmission: **Ingestion of Ova**
	• **Asymptomatic** - light infection
	  ○ Abdominal pain, distention, appendicitis
	• Bloody or mucoid diarrhea
	• **Tenesmus** *(distressing urge to defecate)*, weight loss and weakness – heavy infection
	• **Rectal prolapse** (edematous rectum)
	• Moderate eosinophilia
	• Nutritional deficiency – stunted growth
		• DFS *(Direct Fecal Smear)*
		• Kato-Katz
		• FECT *(Formalin Ether Concentration Technique)*
			• Mebendazole
			• **Albendazole**
			  ○ **Drug of choice**
			• Piperazine citrate
			• Pyrantel pamoate
			• Loperamide hydrochloride (Imodium)
			
			➔ Proper hygiene-hand washing
			➔ Proper sanitation-disposal of feces
			➔ Avoid using fecal matter as fertilizers
			➔ Education-public

 **Trichuris Colonoscopy**		 **Rectal Prolapse**	

LIFE CYCLE OF TRICHURIS TRICHIURA



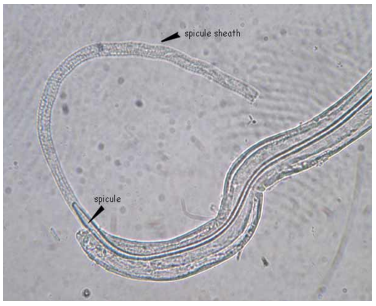
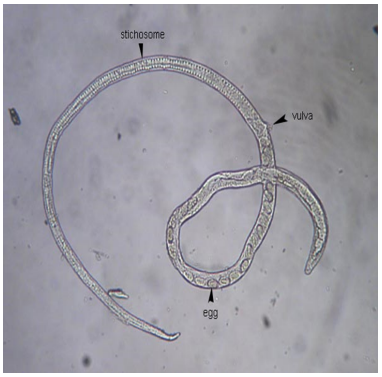
CAPILLARIA PHILIPPINENSIS

CAPILLARIA PHILIPPINENSIS p. 262	**OTHER ADDITIONAL INFORMATION**
HISTORY ● was 1st recognized in the Philippines in 1963 at PGH ○ **Bacarra, Ilocos Norte** ○ 1967, an epidemic of Capillariasis in Pudoc, West Tagudin, Ilocos Sur *(approximately 1,300 persons became ill and 90 persons died of the infection).* ● Adult worms are usually **slender anteriorly** and **stout posteriorly** but tapering is gradual and less pronounced. Occurs elsewhere in the Philippines, and in Thailand, Taiwan, Japan, Korea, Egypt, Iran, and Colombia - various freshwater fish are eaten raw **Rare Capilliaria human infections:** ● **C. hepatica** - hepatic capillariasis ● **C.aerophila** - pulmonary capillariasis	● Adult worms are slender, approximately 4 to 5 mm long. ● They live in the intestinal mucosa, primarily in the **jejunum**  The f**inding of larval stages**, and of **oviparous** as well as **larviparous females** in the bowel, suggests that the parasite multiplies in the intestine and that overwhelming infections are the result of **autoinfection.** ● Eggs develop outside the host ● And are ingested by fresh-water and brackish-water fish 

PATHOGENESIS OF CAPILLARIA PHILIPPINENSIS

CLINICAL SIGNIFICANCE
1. **Pronounced eosinophilia** – surprisingly, does NOT seem to be a consistent feature of this disease. 2. **Hypoproteinemia** – low blood calcium, potassium, and cholesterol levels; and other features of a protein-wasting enteropathy are encountered and are reflected in the pathologic picture 3. **Deepening of Crypts of Lieberkühn** – The villi are blunted, flattened, or completely obliterated with an inflammatory submucosal infiltrate 4. **Giardiasis** and **Coccidiosis** – Malabsorption stages

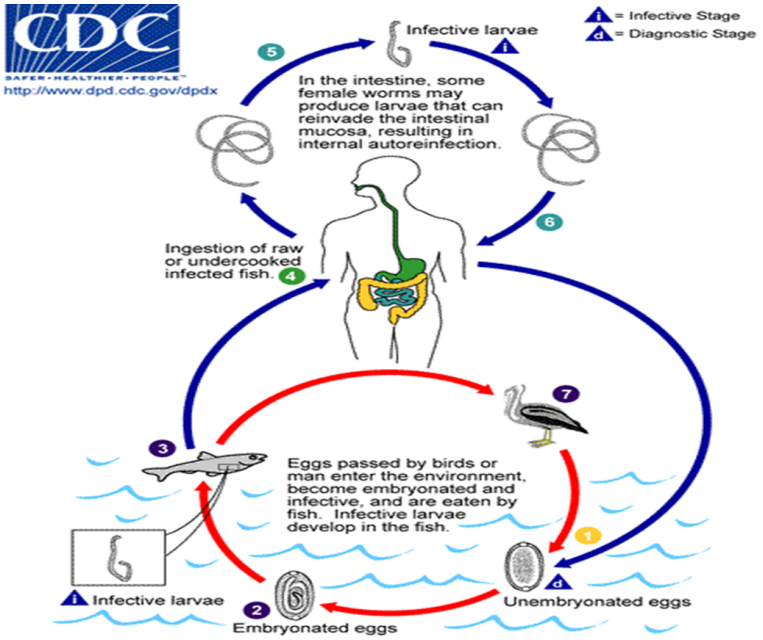
MORPHOLOGY OF CAPILLARIA PHILIPPINENSIS

Male adult worm	*Female adult worm*	*Ova-Larvae infective stage*
➔ **1.5-3.9 mm in length** ➔ Characterized by caudal alae and long, non spiny sheaths. ➔ **Spicule** - copulatory organ	➔ **2.3-5.3 mm in length** ➔ Body is divided into 2 equal parts. ➔ **anterior esophagus+esophageal glands.** ➔ Posterior – intestine, reproductive organs with slightly prominent vulva 2 types of female: - **Larviparous** *(population build up)* - **Oviparous** *(infection)*	➔ **42x20um** ➔ Single or 2 stage development ➔ Similar to trichuris egg but smaller and more oval in shape The shell is thick with striations hence **peanut shape**. With **bipolar mucus plugs** but are not protuberant.
		  

PATHOLOGY OF CAPILLARIA PHILIPPINENSIS

Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*
• **Intestinal capillariasis** Mode of Transmission: **Ingestion of larvae from contaminated fish**	• Abdominal pain • **borborygmi**- gurgling of the stomach • Loss of weight • vomiting • Edema • **Hypoproteinemia** - malabsorption due to destruction of intestinal wall	• DFS- eggs passed out in feces • Identification of larva and worm in stool	• **Mebendazole** ○ **Drug of choice** • Albendazole • Fluid and electrolyte + high protein diet • Proper cooking of fresh water fish(IH) • Proper sanitation-disposal of feces

LIFE CYCLE OF CAPILLARIA PHILIPPINENSIS



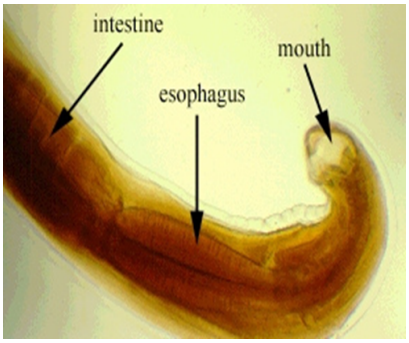
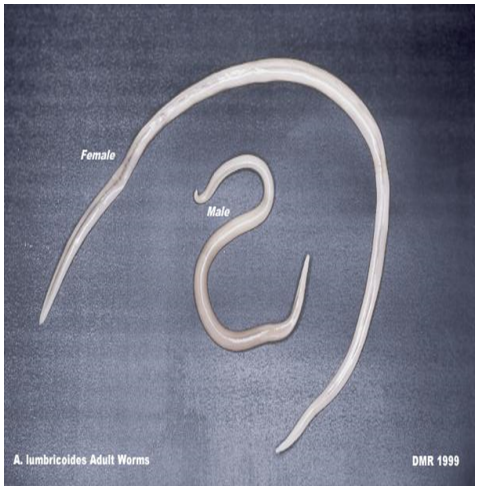
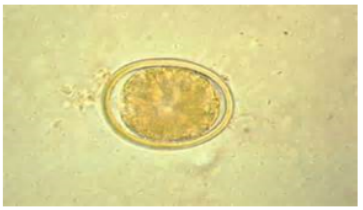
ASCARIS LUMBRICOIDES

ASCARIS LUMBRICOIDES p. 240	**OTHER ADDITIONAL INFORMATION**
• Found in the **temperate and tropical areas** of the globe, under conditions of poor sanitation and where feces are used for fertilizers. *(cosmopolitan parasite)* • Affects **more of the world's population** than any other parasitic disease. (1.3 billion; China-500 million) **"Giant Intestinal roundworm"**- *(a tribute to its resemblance to the earthworm)* - **"Lumbricus"** ○ Pink worm ○ A.suum- pigs • The female worms may be as thick as a lead pencil • The males are definitely more slender and has an incurved tail Both sexes are creamy white, sometimes with a **pinkish cast**, and the cuticle has **fine circular striations** • Ascaris eggs are **unsegmented** when passed • under favorable conditions they require a period of about 2 or 3 weeks outside the host to develop to the infective stage.	• Swallowed embryonated eggs hatch in the **duodenum** 1. The larvae penetrates the wall of the duodenum 2. Then enters blood or lymphatic vessels to be carried to the; a. Liver b. heart c. Pulmonary Circulation 3. Then they are filtered out by the capillaries of the lungs and break from them into the **alveoli** 4. **In the alveoli** is where they grow and molt 5. After about 20 days they migrate through the respiratory passages to reach the esophagus 6. And eventually reach the small intestine • Two or 3 months after ingestion of the eggs, the mature worms commence egg laying in the **intestine** ○ The larvae are not able to grown to adulthood in the intestine as long as any considerable number of worms remain from a previous infection Ascaris infections are sometimes diagnosed on radiography or in cholangiograms

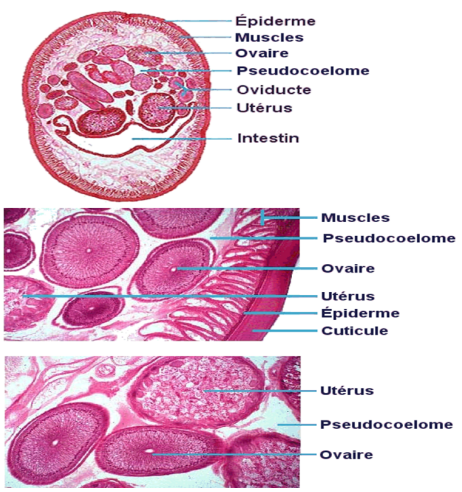
PATHOGENESIS OF ASCARIS LUMBRICOIDES

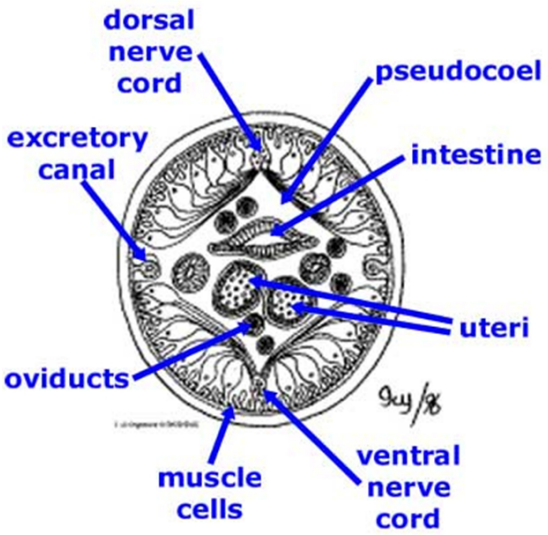
STAGES
• **Impressive eosinophilia is NOT characteristic of** ascariasis – but usually peaks during period of *tissue migration* ○ Declines after worms **re-enter the gut** 1. Increased levels of Immunoglobulin G (IgG), and especially of IgE are seen in infected persons → Damage caused by ascaris seems related to their size 2. The larvae, 20 um in diameter, trapped in 10-um alveolar capillaries, break out with **consequent** (minor) hemorrhage. 3. Large and muscular adult worms do not attach to the intestinal wall a. They maintain their position by constant movement b. They may occasionally force their way into extraintestinal sites or, if present, in large numbers from tangled masses that occlude the bowel. • **Young children are at greatest risk of infection** and ectopic migration of the parasites.

MORPHOLOGY OF ASCARIS LUMBRICOIDES

Male adult worm	*Female adult worm*	*Ova-Infective stage*
➔ **10-31 cm x 2-4 mm** ➔ Smooth striated cuticles ➔ Terminal mouth with **3 lips w/a sensory papillae** ➔ They have ventrally curved posterior end with **2 spicules**  	➔ **22-35/49 cm x 3-6mm** ➔ Smooth striated cuticle ➔ Mouth with **3 lips w/a sensory papillae** ➔ They have **paired reproductive organs** in the posterior 2/3 ➔ Posterior end is conical and straight ➔ Can lay **200,000 eggs/day**	**Fertilized** - 45-70 um x 35-50um ➔ Golden brown ➔ Outer albuminoid coating-**coarsely mamillated** which may be absent (decorticated) ➔ hatch the **larva-14 days** **Thick, transparent hyaline shell w/ a thick outer layer** and a delicate vitelline lipoidal inner membrane **Unfertilized** - 88-94 x 39-44 um ➔ Golden brown ➔ Thin shelled w/ mammillated irregular coat filled with refractile granules ➔ Found only in the absence of males
 		 

CROSS SECTION OF ASCARIS

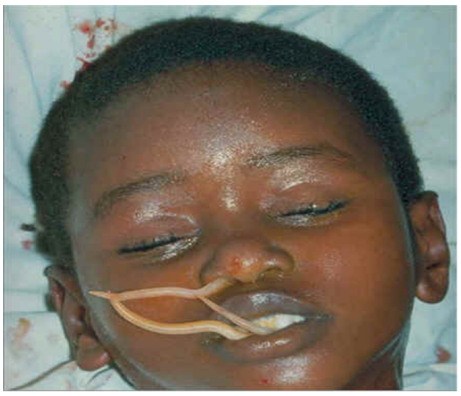




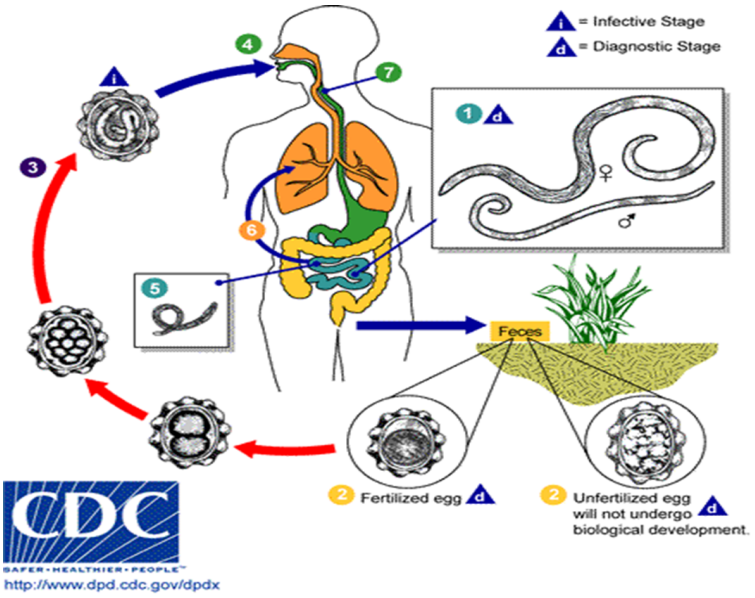
PATHOLOGY OF ASCARIS LUMBRICOIDES

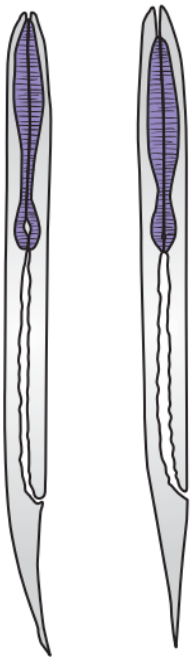
Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*

• Ascariasis
• Ascaris pneumonitis *(loeffler's pneumonia/endocarditis/ syndrome)*
  ○ eosinophilic pneumonia-parasitic infxn
 	
Mode of Transmission:
Ingestion of fertilized ova-Infective stage (ST)
	• **Bowel obstruction** or obstruction of other organs (appendix) in heavy infection
	• Asthma, pneumonitis- due to migration of the juvenile worm to the lungs
	• Cardiac damage - eosinophilic granular proteins
	• Fever, malaise
	• Abdominal distension and tenderness, vomiting
		• DFS-eggs
		• Kato-katz
		• Recovery of worms in stool, sputum
		• Radiographic images may also reveal their presence
			• Mebendazole
			• **Albendazole**
			  a nitroimidaloze that binds irreversibly to tubulin, blocks microtubule assembly and inhibiting glucose uptake by the worm is the **drug of choice**.
			• Pyrantel pamoate
			• Piperazine citrate
			
			➔ Proper hygiene-hand washing
			➔ Proper sanitation-disposal of feces
			➔ Avoid using fecal matter as fertilizers
			➔ Education-public



LIFE CYCLE OF ASCARIS LUMBRICOIDES



STRONGYLOIDES STERCORALIS	OTHER ADDITIONAL INFORMATION
• Occurs in the tropical and subtropical areas of **abundant rainfall**. Loose sandy loam soil, ambient temperature. • **"Threadworm"**  *Free-living female S. stercoralis* Free-living generation = male & female Parasitic generation = parthenogenetic females • More frequently, perhaps, there is but a **single free-living generation**, producing rhabditiform larvae, which transform into infective filariform larvae • The filariform larvae are incapable of further development in the soil and **must penetrate the skin of their host** to continue the life cycle. • Rhabditiform larvae that pass out of the host in the stools **MAY ALSO** transform into filariform larvae directly, without developing into free living adults • Migratory routes involving organs other than lungs are **NOT ONLY** are possible but it may predominate  *S. stercoralis larvae* Male worms are eliminated from the body early in the infxn. • **Definitive Host** - man, cats, dogs, other mammals • Prevalence rate is lower compared to hookworm infxn. • Infxn may persist due to **autoinfection**	When they (larvae) molt within the intestinal tract, they **usually retain their rhabditiform characteristics** but may transform into infective filariform larvae If filariform larvae are formed, they **MAY PENETRATE** immediately into the wall of the gut and enter the bloodstream **DIAGNOSIS OF S. STERCORALIS LARVAE** • Larvae resemble those of hookworms but can be distinguished by their very short buccal cavity  *Strongyloides* Hookworm Hookworm larvae have a long buccal cavity, the space between the oral opening and the esophagus, whereas Strongyloides has a short buccal cavity – this is the most common way to distinguish them both **With severe diarrhea**, the embryonated eggs **MAY BE PRESENT** in the stools and can be differentiated from hookworm eggs by the fact that they always contain well-developed larvae

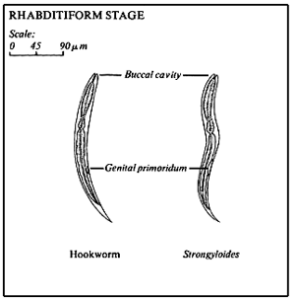
PATHOGENESIS OF STRONGYLOIDES STERCORALIS

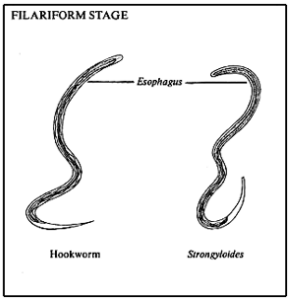
CLINICAL SIGNIFICANCE
1. **Local Lesions** – occurs due to penetration of the larvae into the skin 2. **Creeping eruption** – chronic cutaneous larval migration, migration of larval (immature) worms beneath the skin, resulting in itchy, winding, and raised tracks or lines on the skin's surface. 3. **Patchy pneumonitis** – larvae may be found in sputum 4. **Worm infestation** – adult female worms may be found in all parts of the intestinal tract; especially the **jejunum** 5. **Disseminated strongyloidiasis** – can produce extensive ulceration and sloughing of the mucosa, and fibrosis and inflammatory infiltration of the submucosal layers and granulomas 6. **Eosinophilia of 10% to 40%** – is common; in occasional patients it is higher. **a.** Lack of eosinophilic response is occasionally seen, generally in overwhelming infections. b. Total serum IgE is usually elevated.

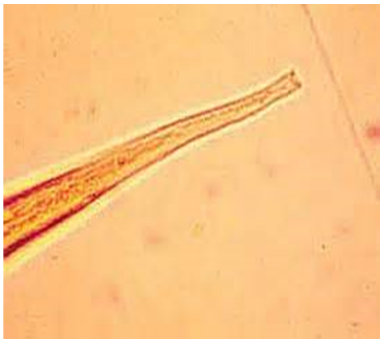
MORPHOLOGY OF STRONGYLOIDES STERCORALIS

Male adult worm	*Female adult worm*	*Ova-Larvae infective stage*
➔ **0.7mm x .04 mm** ➔ free-living ➔ Smaller than the females ➔ Ventrally curved tail with 2 copulatory spicules a **gubernaculum** ➔ No caudal alae ➔ Does not persist in the host (Namatay sa sarap)	**free-living**; smaller than the parasitic ● Muscular double-bulbed esophagus and the intestine is straight cylindrical ● **1mm x .06 mm** **Parasitic** - colorless, semi-transparent, w/ finely striated cuticle ● It has slender tapering anterior end and a short conical pointed tail ● Short buccal cavity w/ long slender esophagus that extends 1/4 of the body, intestine is continuous to the anus ● Vulva is located 1/3 the length of the body from the posterior end ● the uteri contain a single file 8 to 12 thin shelled , transparent, segmented ova ● Parthenogenetic ● **2.2mm x .04 mm**	➔ **RHABDITIFORM** – 225um x 16um ● Elongated w/ pyriform posterior bulb ● Slightly smaller than HW and less attenuated posteriorly ● **Shorter buccal capsule** and **larger genital primordium** ➔ **FILARIFORM** - Infective stage and the non-feeding stage ● 550 um ● Similar to HW but smaller with a distinct cleft(notched) at the tip of the tail ➔ **OVA** - rarely seen in the stool ● Clear, thin shell similar to HW, 8-cell stage development. Larva inside ● 50-58um x 30-34 um
	 	 **Rhabditiform Larvae**  **Filariform Larvae**

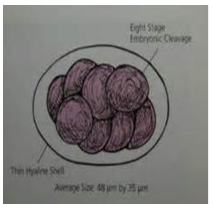
DIFFERENT INFECTIVE STAGES OF STRONGYLOIDES STERCORALIS







Filariform Larvae



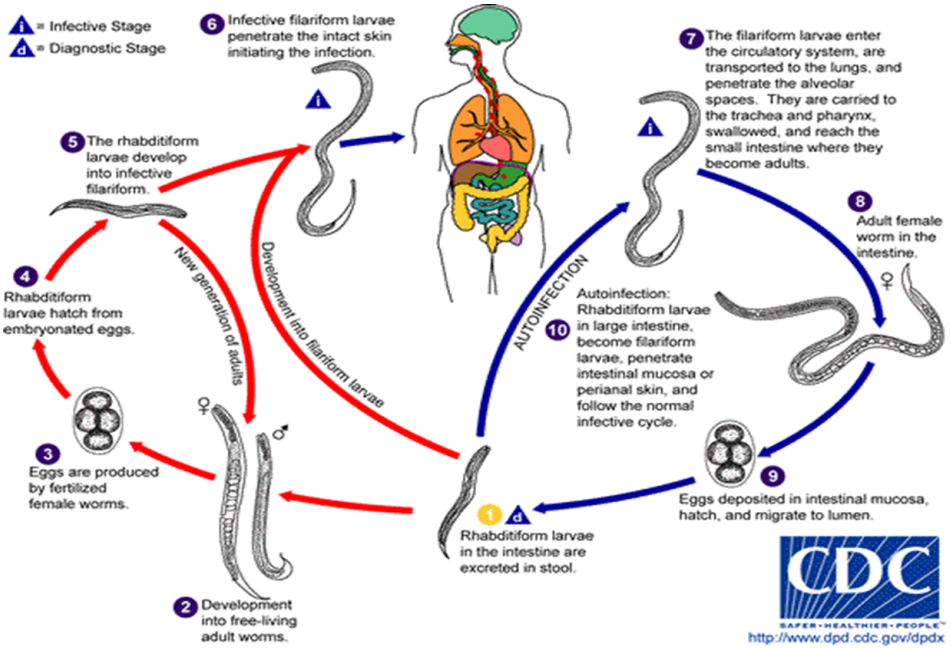
Ova Stage

A. worm & Larva migrans

PATHOLOGY OF STRONGYLOIDES STERCORALIS

Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*
• Strongyloidiasis • DH - humans, monkeys Mode of Transmission: **Filariform larva- soil/skin penetration**	• Lesions – intense itching at the site of penetration • Pneumonitis- migration of worm to the lungs • Moderate to severe diarrhea • Malabsorption syndrome w/ steatorrhea • Malnutrition • Ulcers- LGIT and UGIT-bleeding • Hyperinfection-AIDS or immunocompromised pts. • Transmitted- mammary glands, organ transplant	• Identification of larva in stool • Sputum exam • DFS • FECT • Baermann technique. CH 14 pg. 407 • Agar plate method	• **Albendazole** • **Ivermectin** Both are drug of choice ➜ Proper sanitation-disposal of feces ➜ Avoid walking barefoot on soil

LIFE CYCLE OF STRONGYLOIDES STERCORALIS



ENTEROBIUS VERMICULARIS

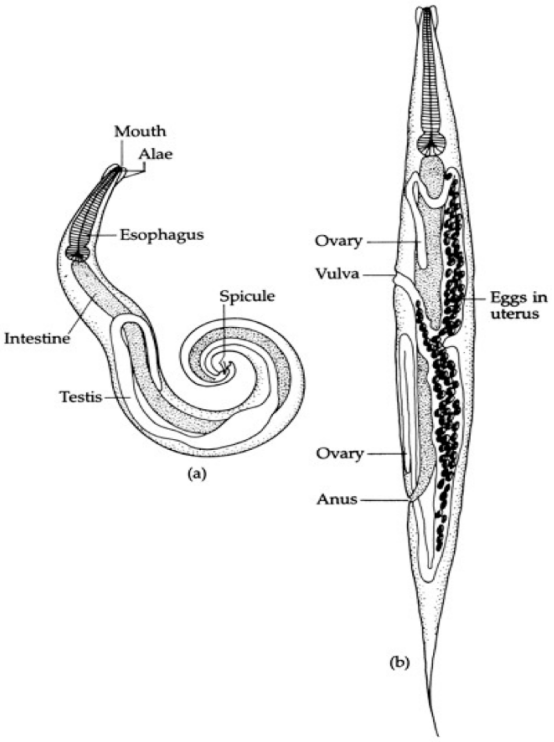
ENTEROBIUS VERMICULARIS p. 246	OTHER ADDITIONAL INFORMATION
• "**Pinworm, Seatworm, Oxyuris**" • **MOST COMMON HELMINTH** parasite of the temperate regions but is also common to tropics and subtropics region • 10% of the pediatric population may be infected in tropic regions • Found in families of **high socioeconomic status** where sanitation levels are relatively rigorous • Spread is facilitated by **crowded indoor living** in temperate climates – *orphanages, hospitals, sanitariums.* Pinworms caused no disfiguring or debilitating effects but, their presence is an embarrassment and irritation The worms may migrate several inches out of the anus, depositing eggs as they crawl or liberating masses of them as the worms **dry and explode**. • The eggs are fully embryonated and are infective within a few hours of the time they are deposited ○ If climatic conditions are suitable, they survive for some *weeks* outside the body ○ The eggs live longest under conditions of fairly high humidity and moderate temperature	• Development of adult worms requires **6 weeks** – however shorter periods have been reported. • Reinfection of the patient by contamination of the hands is common and makes control of the parasite very difficult • **RETROINFECTION** → involves hatching of the embryonated eggs *after* their deposition in the perianal area and subsequent migration back into the rectum and large intestine. **DIAGNOSIS OF PINWORM OVA:** • Made on recovery of the characteristic eggs, although it may be suspected in children who have pruritus ani. ○ The ova has a **translucent shell of moderate thickness** and are conspicuously flattened on one side ○ *This flattening and consequent reduction in diameter, plus the thicker shell, differentiates them from hookworm eggs.* • Occasionally, the adult female worms are seen crawling in the perianal region or in the feces. females ordinarily do not oviposit until they leave the intestinal tract

PATHOGENESIS OF ENTEROBIUS VERMICULARIS

STAGES
- **Enterobius** may be considered a **commensal** in all persons save those whose hypersensitivity to the secretions and excretions of the worms leads to rectal pruritus 1. Attachment of adult worms to the intestinal wall may produce some inflammation. 2. Inflammation of appendix might be expected to be the common occurrence a. Any relationship between the invasion and appendicitis remains unproven. 3. Entrance into peritoneal cavity via female reproductive system may result in the formation of granulomas around eggs or worms; rarely of clinical significance but are responsible for **chronic pelvic peritonitis** 4. Pinworms, or their eggs, have occasionally been reported from other ectopic sites (Liver and lung).

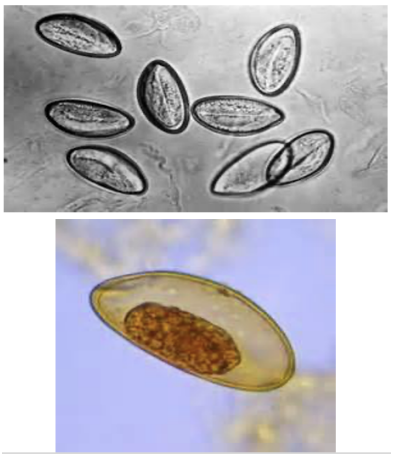
MORPHOLOGY OF ENTEROBIUS VERMICULARIS

Male adult worm	*Female adult worm*	*Ova – infective stage*
• **2-5 mm x 0. 1-0.2 mm in diameter** ○ Smaller than the female worm • Oral end is provided w/ 3 lips and a pair of lateral cephalic alae. • Posterior end is curved ventral w/ caudal alae supported by 6 pairs of caudal papillae • They are rarely seen because they **DIE AFTER COPULATION** *(Namatay sa Sarap)*	• **8-13 mm x 0.3-0.5 mm in diameter** • Light yellowish white in color • Long, thin sharply pointed tail • The uteri of the gravid female are distended with eggs • Can lay eggs as much as 4,600- 16,000 eggs daily • Females **dies soon after oviposition** *(Namatay sa Hirap)* • More females than males in the host • When fully gravid, migrate down the intestinal tract to pass out the **anus** and deposit their eggs.	• **50-60 um x 55-36 um** • Fully embryonated • Infective in 4-6 hrs. total time from ingestion to maturity is 6 weeks • **Asymmetrical w/ one side flattened and the other convex.** • Translucent shell consists of an outer triple albuminous layer (mechanical protection), Inner lipoidal membrane (chemical protection) • Live longest under conditions of fairly high humidity and moderate temperature • **Light, can be airborne, carried by dust**



Male (left) and female (right) Enterobius Vermicularis

Adult worm (E. vermicularis)



Ova (E. vermicularis)

PATHOLOGY OF ENTEROBIUS VERMICULARIS

Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/Care*
• Enterobiasis • Oxyuriasis Mode of Transmission: **Ova-infected stage →** ingestion/retroinfection	• **Pruritus ani** - migration of female worm to the anus during the resting state (itching) → Migration of female worms from the anus in some persons produces **pruritus** which may be	• **Cellophane tape swab**- recovery of ova in the peri-anal region • Usually performed	• **Albendazole** ○ **Drug of Choice** • Mebendazole, • Pyrantel Pamoate 1. Quarantine/ Tx for

	severe at some times. → In small children, worms may invade the vagina after leaving the rectum, producing **local irritation.** ● Vaginal migration- causing irritation → Local itching may interfere with sleep of infected px. ● Appendix, liver, lungs ● Retrofection/ Autoinfection	before pts. Bowel movement or taking a bath → Made on recovery of the eggs, although it may be suspected in children who have **puritus ani** → Adult female worms are seen crawling in the **perianal region** or in the feces. ● Eggs may be found in the stools.	entire members of the family 2. Disinfect contaminated fomites (clothings, beddings, towels, etc.) 3. Proper hygiene – washing of hands 4. Education – family

FAMILY ANCYLOSTOMIDAE

FAMILY ANCYLOSTOMIDAE p. 248 *Foundations of Parasitology (8th ed.) Chapter 25*	**ADDITIONAL INFORMATION**

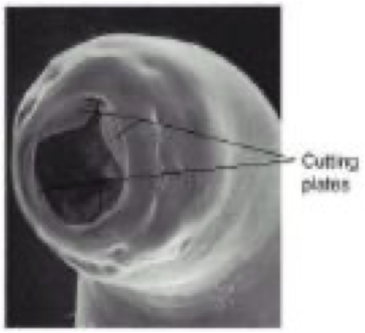
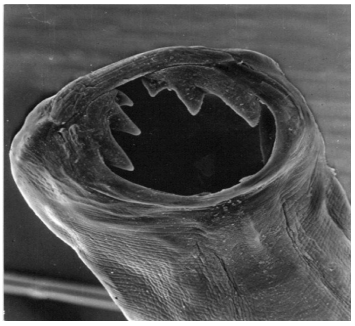
- "Hookworms"
- Most species are stout, and the anterior end is curved dorsally, giving the worm a hook like appearance
- Live in their host's intestine, attaching to the mucosa and feeding on blood and tissue fluids sucked from it.
- Common in tropics and sub tropics with abundant rainfall, ambient temperature, loose sandy loam soil.
- Buccal capsule is large and hardened by fibrous tissue and usually armed with cutting plates, teeth, lancets or a dorsal cone. Lips are reduced or absent

BURSA COPULATRIX

- The esophagus is stout with a swollen posterior end giving it a club shape. It is mainly muscular, corresponding to its action as a powerful pump
- Males have spicules, needle-like, and are provided with conspicuous copulatory bursa, consisting of 2 broad lateral lobes and a smaller dorsal lobe. All are supported by a fleshy ray. ***IMPORTANT TAXONOMIC CHARACTERISTICS***
- Females have a simple, conical tail
- The vulva is post-equatorial, and 2 ovaries are present
- About 5% of the daily output of eggs are found in the uteri at any one time.

BUCCAL & TAIL

NECATOR AMERICANUS	● New world hookworm; introduced from Africa(south of Sahara) with the slave trade. ● The only hookworm found in North and South America, India, China, SE Asia, Indonesia, SW Pacific, parts of Australia → Adults resemble those of Ancylostoma **but are slightly smaller** ● Head is *sharply bent* in relation to the rest of the body forming a **definite hook shape** at the anterior end, from which worms derive their common name.

	
ANCYLOSTOMA DUODENALE 	• Old world hookworm; the only hoowkorm of Europe and the areas bordering the Mediterranean, West coast of South America, parts of India and China (N. americanus), Brazil, SE Asia, Indonesia, South and Southwest Pacific → Adults are grayish white or pinkish; head has a slight bend in relation to the rest of the body • **MOUTH IS WELL DEVELOPED** • Ancylostoma infections may be percutanous, oral, transmammary or transplacental.
ANCYLOSTOMA BRAZILIENSE 	• Found in domestic cats & dogs and wild carnivores in most tropics. Human infxn found in Brazil, Africa, India, Sri Lanka, Indonesia, Philippines
ANCYLOSTOMA CANINUM 	• The most common HW of domestic dogs in the Northern Hemisphere • Common cause of creeping eruptions • Eosinophilic enteritis in Northern Australia and the US. → Hookworm of dogs • Known not only to produce abortive infection in humans • Larvae is unable to complete their life cycle and establish intestinal infection
ANCYLOSTOMA CEYLANICUM	• A parasite of the carnivores in Sri Lanka, SE Asia, East Indies, Philippines.
CUTANEOUS LARVA MIGRANS	When humans are infected with Ancylostoma braziliense or Ancylostoma caninum, skin penetration takes place, but **larvae is unable to complete their migratory cycle.** • Ancylostoma braziliense may survive for some weeks/months migrating through subcutaneous tissues • Ancylostoma caninum encyst & remain dormant in skeletal muscle after shorter cutaneous migratory period → Both may evoke a fairly severe reaction, forming **serpiginious tunnels** through the tissues, erythematous and

	sometimes vesicular at the advancing end, fading out and becoming dry and encrusted in the older portions. ● There is often **intense pruritus** and scratching may lead to secondary bacterial invasion. ➜ **The syndrome is called creeping eruption or as cutaneous larva migrans** vs. visceral larva migrans. ➜ Albendazole is the treatmentof choice ● Oral ivermectin may also be used

PATHOGENESIS OF FAMILY ANCYLOSTOMIDAE

STAGES
- Significant negative correlation between worm burden and hemoglobin level can be demonstrated in any representative series of patients infected w/ hookworm ● When **anemia** is present, it is of the ***microcytic hypochromic*** type. The bone marrow is generally markedly **hyperplastic** and there may be erythroid and myeloid hyperplasia of the spleen. ● Ancylostoma duodenale has a **considerably greater host blood loss per day** compared to Necator americanus. 1. Histologic changes in the mucosa of the affected intestine appear to be minimal. 2. Flattening or atrophy of the intestinal villi has been noted on occasion 3. Malabsorption is apparently uncommon and malnutrition does not seem to be characteristic of pure hookworm disease in areas where good diet is available. 4. Emaciation, mental and physical retartation are frequently associated w/ this disease a. More ascribed to a combination of other nutritional and disease factors that are more common in many endemic areas

MORPHOLOGY OF FAMILY ANCYLOSTOMIDAE

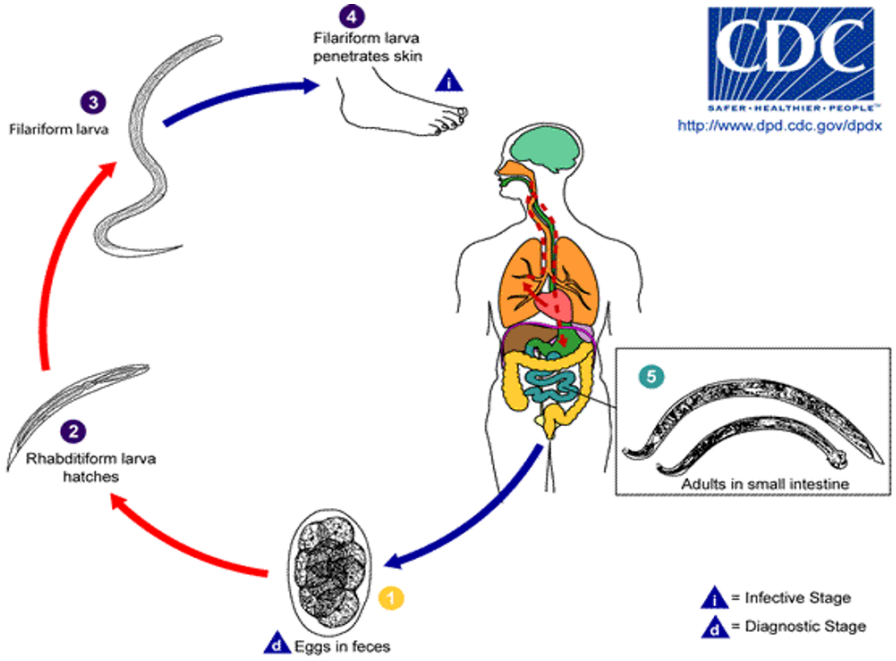
FAMILY ANCYLOSTOMIDAE	***Male adult worm***	***Female adult worm***	***Ova – infective stage***	***Larva – infective stage***
Necator Americanus	→ 5-9mm x 0.3 mm → Small, cylindrical, fusiform, grayish white shorter than the female → Buccal capsule – ventral pair of semilunar cutting plates on either side of the median line	→ 9-11mm × 0.35mm → Blunt posterior end → Buccal capsule- same w/ male → 5,000-10,000 eggs daily → Life span - ***18 years***	→ 64-76 um × 36- 40 um → Regularly oval → Colorless and usually seen in 2-8 cell stages	***Filariform*** – infective stage ⇒ 700um → non feeding stage → buccal spears are conspicuous and parallel throughout their lengths → Tail is pointed covered w/ sheath → Must penetrate through the skin
Ancyolostoma duodenale	→ 1 cm x 0.5 mm ● larger than americanus → Buccal capsule – 2 pairs of curved ventral teeth on either side of the median line	→ 12-15 mm x 0.7 mm → Blunt posterior end → Buccal capsule- same w/ males → 10,000-30,000 eggs daily → Life span - 1-5 years		→ Incospicuous buccal spears → Tail is pointed curved w/ sheath
Ancylostoma braziliense	→ Buccal capsule- pair of big teeth → Parasite of dogs and cats and wild carnivores			**Rhabditiform** – feeding stage ● resembles that of Stronevloides, but larger ● More attenuated posteriorly (pointed) → Longer buccal capsule

				→ Genital primordium is incospicuous
Ancylostoma caninum	→ Buccal capsule - 3 pairs of ventral teeth, the innermost is the smallest → Parasite of dogs only			
Ancylostoma ceylanicum	→ Buccal capsule- 2 pairs of ventra teeth but the outer pair is bigger than the inner → Parasite of dogs and wild carnivores			

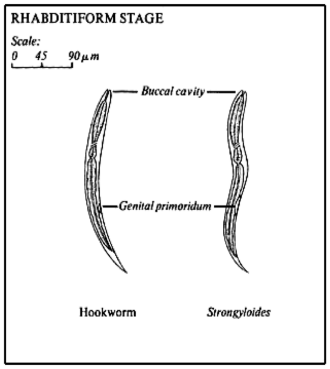
PATHOLOGY OF FAMILY ANCYLOSTOMIDAE

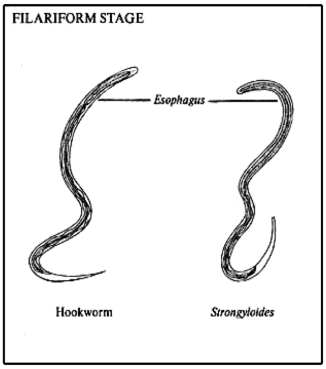
Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/Care***
• Hookworm disease • Ancylostomiasis • Necatoriasis • Uncinariasis • Cutaneous Larva Migrans (creeping eruption)   **Filariform -** skin penetration/soil (Infective Stage)	• Allergic reaction – ground itch(dermatitis, pruritus, 2nd infectionn) • Cutaneous Larva Migrans (CLM) - serpiginous (A. braziliense, caninum) • Eosinophilic Enteritis – A.caninum • Pneumonitis – not as prominent as Ascaris • Gastrointestinal discomfort- pain, diaarhea, flatulence • Microcytic hypochromic type anaemia – A. duodenale sucks 0.26 ml of blood/ day/worm. ○ N. americanus sucks 0.03ml/ day/worm	• Direct Fecal Smear- ova (2-8 cell stage) • Larva cannot be seen in stool until after 24 hrs. At room temp. must be differentiated with Strongyloides stercoralis	• Albendazole • Mebendazole • Pyrantel pamoate • Ferrous sulfate 1. Education- public 2. Proper hygiene-proper disposal of feces 3. Avoid walking barefooted on soil.
 			

LIFE CYCLE OF FAMILY ANCYLOSTOMIDAE



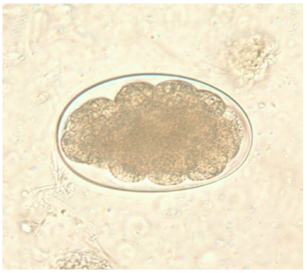
LARVA OF FAMILY ANCYLOSTOMIDAE

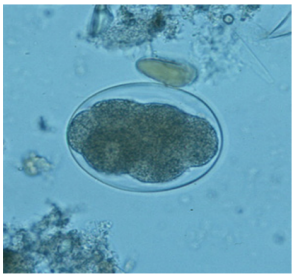




FILARIFORM LARVA

RHABDITIFORM LARVA





OVA

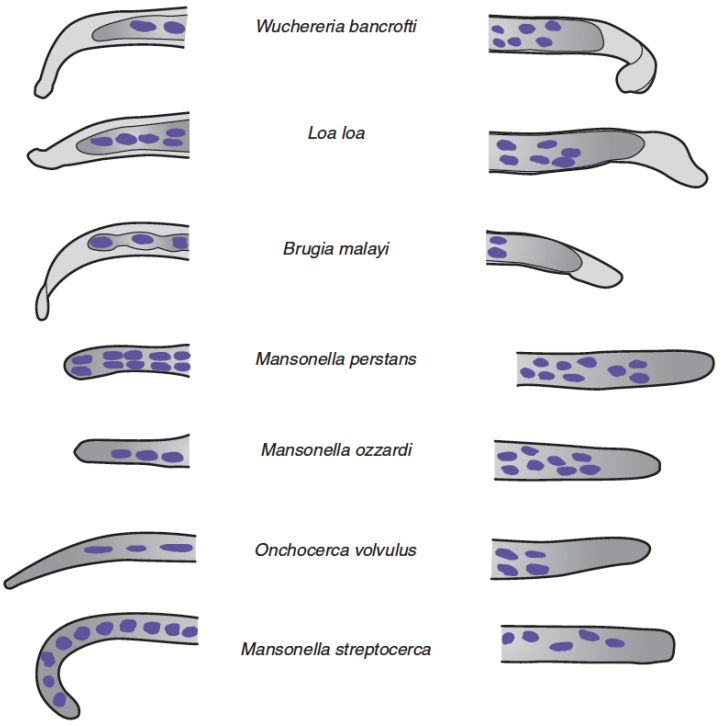
OVA

BLOOD AND TISSUE-DWELLING NEMATODES

BLOOD AND TISSUE-DWELLING NEMATODES

- The "Microfilariae group"
- Requires an arthropod as an intermediate host
- Diagnosis is made by examining thick and thin Giemsa stained blood smears. (except O. volvulus-skin scrapings from nodular lesions)
- They exhibit periodicity.
- Long, threadlike nematodes
- Various species inhabit the human lymphatic system, while others the subcutaneous and deep connective tissues
- The adults of all species of filariae are parasites of vertebrate host
- The adult female worm produces eggs that during their development become elongated and wormlike in appearance. "Microfilariae" (Ovoviviparous)
- Microfilariae migrate within the vascular system and through the tissues
- Microfilariae can live a long time in the body of the vertebrate host but will not undergo further development
- Developing further only when ingested by their IH and vector, an Insect. Transforming into infective arva
- Lymphatic filariasis

1. Wuchereria Bancrofti
2. Brugia Malayi
3. Loa Loa
4. Onchocerca Volvulus



WUCHERERIA BANCROFTI

WUCHERERIA BANCROFTI (ch. 9)	**ADDITIONAL INFORMATION**

• Cosmopolitan in distribution – tropics and sub tropics
• Originated in SE Asia in early civilization (BC), parasitizing Indonesian leaf monkeys
• Recent prevalence rate is 120million cases globally
• Diseases:
  ○ Bancroftian filariasis
  ○ Wucheriasis
  ○ Elephantiasis
	• Wuchereria kalimantani parasitizes the Indonesian leaf monkey
	  ○ Closest known relative of Wuchereria bancrofti
	• Life cycle was discovered by Manson in 1878
	  ○ Transmission of this parasite by mosquitoes was the **first demonstration** of an arthropod as a vector of a parasitic organism → The mosquito Manfson found in China was **Culex fatigans**
	  - Mosquito was more than a simple agent of transmission of the

	parasite; an essential developmental cycle takes place within the body of the insect 1. Upon taking blood from an infected person, the mosquito may ingest microfilariae with its blood meal 2. Microfilariae bore through the stomach wall to enter the body cavity of the insect 3. Microfilariae migrate to the thoracic musculature for a period of gorwth 4. During the next 10 days or so, larvae grows and motl to become **infective-stage larvae** 5. Larvae increased in length from about 300 um to 1.5 to 2 mm. 6. Infective larvae enter proboscis of mosquito and when next blood meal is taken, they escape from proboscis onto the skin. 7. They enter through the puncture hole left by mosquito to infectn ew host

PATHOGENESIS OF WUCHERERIA BANCROFTI

STAGES/CLINICAL SIGNIFICANCE
● Adult worms are found in lymph vessels but particularly found in the **axillary, epitrochlear, inguinal** and **pelvic nodes** ○ Lympahtics distal to them – testis, epididymis, cord 1. Inflammatory changes in and around the lymphatic system comprise *basic reaction to infection* 2. Attacks of lymphangitis and lymphadenitis may begin before worms mature a. Attacks are marked by retrograde lymphangitis if limb is involved; funiculitis, epididymitis, or orchitis may be seen if the worms are located in the scrotal lymphatics; fever and other constitutional symptoms without localizing signs are seen if the pelvic or abdominal lymphatics are the site of inflammation. 3. It is now recognized that bacterial/fungal superinfection of the compromised lymphatics *may* play an important role in the recurrent attacks of adenolymphangitis. 4. Repeated attacks of inflammation lead to dilation and thickening of the affected lymphatic vessels, which may become incompetent and lead to lymphedema. **5.** **Lymphedema** may be intermittent early in the course of the disease, *but* lymphatic vessels tend to become fibrosed after repeated attacks of lymphangitis. a. With chronic lymphedema, there is **hyperplasia** of the connective tissue, and infiltration of plasma cells, macrophage, and eosinophils. **6.** **Woody induration** of the tissues take place with thickening and verrucous changes of the skin, producing **elephantiasis.** **a.** **Elephantiasis** may develop in any limb; scrotal elephantiasis is common b. Women → elephantiasis of the breasts and vulva is seen c. Elephantisis → disfiguring and frequently disabling condition d. Circulation is badly impaired e. Elephantoid limb or organ is in constant danger of **secondary bacterial or fungal infection** 7. Scrotal lymphatics, when involved, may lead to **hydrocele** way early and more common vs. *scrotal elephantiasis*. 8. Lymph varices may form in affected vessels

MORPHOLOGY OF WUCHERERIA BANCROFTI

Male adult worm	***Female adult worm***	***Microfilariae – Diagnostic stage***
● 20-40 um x 0.1 mm in diameter ● copulatory spicules are distinctly unequal and dissimilar ● Gubernaculums is crescent-shaped ● Caudal end is curved ventrally ● Found tightly coiled in nodular dilations in lymph vessels and sinuses of lymph glands	● 80-100 um × 0.24-0.3 mm in diameter ● Vulva is cervical in position(near the level of the middle of the esophagus) ● Posterior end is narrow and abruptly pointed ● Found tightly coiled in nodular dilations in lymph vessels and sinuses of lymph glands	● Minute snake-like organism constantly moving among the RBC ● 270-290 um enclosed in a hyaline sheath which is much longer than its body ● Contains nuclei along the axis of its body ● Conspicuously arranged in 2-3 rows and do not extend to the tip



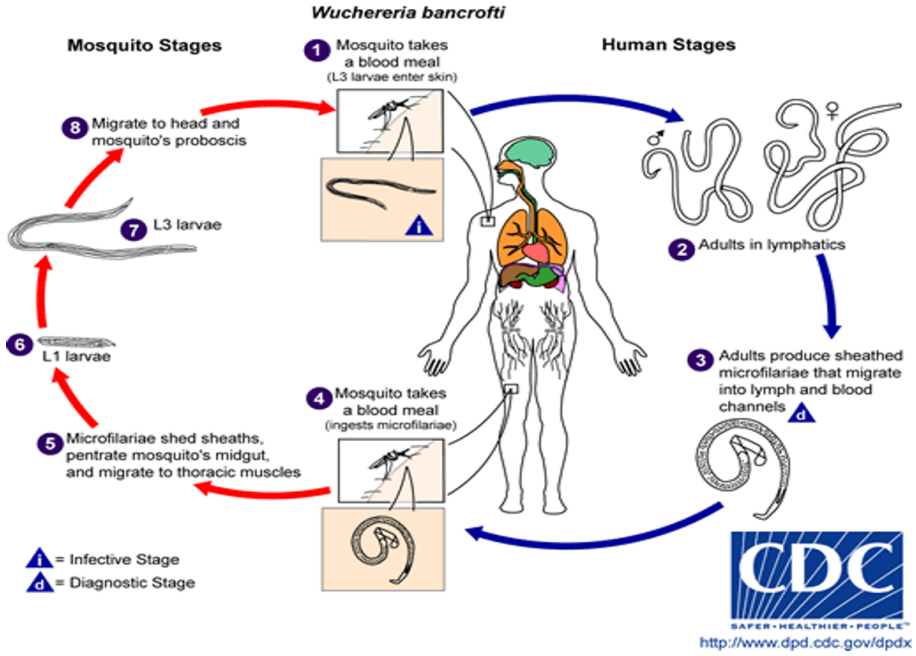
PATHOLOGY OF WUCHERERIA BANCROFTI

Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/ Care***
• Bancroftian filariasis • Wucheriasis • Elephantiasis → Mosquito bite • Anopheles & Culex- nocturnal periodic strain • Aedes- subperiodic Pacific strain	**ASYMPTOMATIC** → "endemic individuals" • tolerance of the immune system which does not recognize the worm as foreign – "microfilaremia". • some will develop Renal (hematuria/proteinuria) and Lymphatic disease - Hydrocele **INFLAMMATORY (ACUTE)** → fever (high 1-2 days and gradually subsides after 2-5 days). • Lymphangitis – inflammation of the lymphatic vessels. • Lymphadenitis- inflammation of the lymph nodes. ○ Affects the limbs, breast, scrotum(orchitis) epididymitis (spermatic cord) or elsewhere. • Chyluria – lymph in the urine **OBSTRUCTIVE PHASE** • blockage of the lymph vessels- dilating it and the accumulation of chyle (chyluria) .-Milky urine **ELEPHANTIASIS** • The enlargement of one or more limbs, scrotum, breast, or vulva with dermal hypertrophy and varicose changes. Adult worms are found in the lymph vessel throughout the body.	1. Blood smear (Thick & Thin preparation) stained w/ Giemsa stain → identify microfilariae- sheathed w/ no nuclei at the tip of the tail. 2. Serologic test- ELISA employing monoclonal abs to detect circulating Ag. 3. ICT- whole blood Ag card test **NOCTURNAL PERIODICITY** • 9pm- 2am is their greatest concentration in the blood Subperiodic • exhibit microfilaremia at all times but most between noon and 8 pm.	• DEC- diethylcarbamazine • Ivermectin - Eradicate breeding areas of mosquitoes - Pest control - Mosquito nets - Tx of infected individuals

	• Axillary, epitrochlear, inguinal, pelvic nodes, testis, epididymis, spermatic cord. Worms die and tend to calcify		

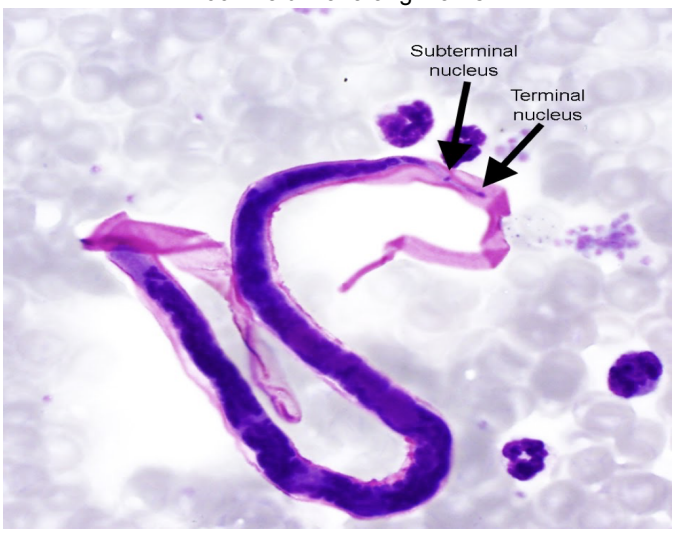
ELEPHANTIASIS

LIFE CYCLE OF WUCHERERIA BANCROFTI

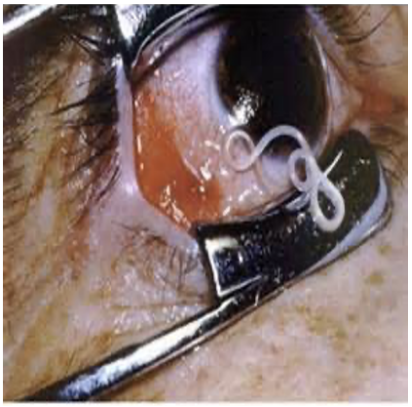


BRUGIA MALAYI

BRUGIA MALAYI p. 285	
• Malayan form of lymphatic filariasis- "Malayan filariasis" • Common in the Far east. 30% occur in South China, 20% in India. While the rest occur in Thailand, Vietnam, Indonesia, Malaysia, Philippines, and South Korea • exhibit nocturnal periodicity in the bloodstream • Pathology is similar to Wuchereria bancrofti but confined to lower extremities • Mosquito vector belongs to genus Mansonia and some Anopheles • **Diagnosis**- Blood smear (Thin & Thick) • **T/P/C**- same with bancrofti • Microfilariae- 177-230 um in length. Sheathed, body nuclei extends almost to the tip of the tail. 2 terminal nuclei are distinctly separate from the others in the tail.	• Life cycle is similar to that of Wuchereria bancroft, except that in most areas, the principal mosquito vectors belong to the genus **Mansonia.** ○ Anopheles may be an important vector, or the only one (in some areas) • **IMPORTANT RESERVOIRS:** Macaques (Macaca spp.) and leaf monkeys (Presbytis spp.) of *certain strains of Brugia malayi* ○ Can be transmitted to cats and civet cats. → In Indonesia - Zoophilic: transmitted by Mansonia mosquitoes - May be aperiodic, nocturnally subperiodic, or nocturnally periodic in humans - Nocturnally subperiodic in jirds - Anthropophilic strain: transmitted by Anopheles mosquitoes - **Always** exhibits nocturnal periodicity

○ 200-275 um ana ang Markell  	● Adult brugia malayi seem to be *smaller* than Wuchereria bancrofti **CLINICAL FEATURES** - smaller than bancroftian infections ● Lymphadenitis occurs in inguinal area ○ Followed by retrograde lymphangitis ○ Then lymphedema of foot and ankle ● When Elephantiasis occurs in Malayan filariasis, it involves the **leg below the knee or the arm below the elbow**

LOA-LOA

LOA-LOA p. 289	
● "African eye worm" ● Found in the rain forest of Sudan, Congo and West Africa. ● The scientific name is a native term for the worm. Discovered during the slave trade ● Migrates into the conjunctival tissue across the eyeball. ● Exhibit diurnal periodicity (Microfilariae is shed in the bloodstream day or night) 10am-2pm ● Microfilarae- 275 um in length. Sheathed, nuclei extends to the tip of the tail  	● Adult Loa migrates actively **throughout the subcutaneous tissues of the body** ● Derives its popular name for the fact that it is the **most conspicuous and irritating when crossing the conjuctiva**

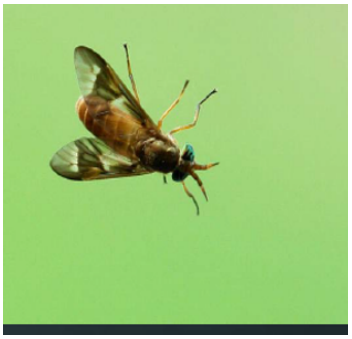
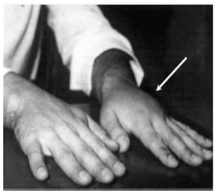
PATHOGENESIS OF LOA LOA

CLINICAL SIGNIFICANCE
- Little evidence that Loa loa produces any lasting damage to host during its life - Rapid migration through subcutaneous tissue (1cm/ minute) - **Completely painless** in areas other than the face - Eosinophilia of 50%-70% is noted especially when Calabar swellings are present   - Increase of IgE - Hypersensitivity to worms and microfilariae - Lymphadenitis

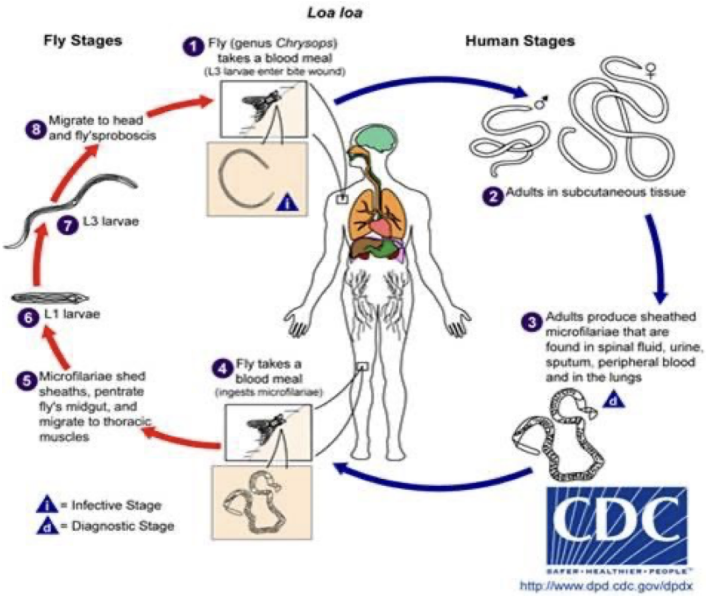
MORPHOLOGY OF LOA LOA

Male adult worm	***Female adult worm***	***Microfilariae – Diagnostic stage***
→ 2 to 3.5 cm long → **Adult worms** migrate through the subcutaneous and deeper connective tissues	→ 5 to 7 cm → Neither male nor female is more than 0.5 mm wide	● 250 to 300 um long ● Sheathed, differ from Wuchereria bancrofti and Brugia malayi ● Have **body nuclei** that are continuous to the tip of the tail ● Microfilariae make their way into the blood stream where they **circulate** ○ There is **diurnal periodicity** ● May be ingested by any of several species of mango fly **Chrysops** (Mango fly) - Large with mouth parts that can produce a *painful bite* - Microfilariae undergo a **developmental cycle** in the thoracic musculature of the fly similar to Wuchereria bancrofti in the mosquito - After 10-12 days, reaches the **infective stage** - When the fly bites, the infective larvage migrate out onto the surface of the skin and then enter through the bite wound.

PATHOLOGY OF LOA-LOA

Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/ Care***
● Loiasis ● Bites of Mango fly-genus Chrysops 	● Calabar swelling – allergic reaction to the metabolic products of the worm or dead worms when they appear in one area. (Wrist & ankle)   ● Localize pain and pruritus ● Appearance of the worm in the conjunctivae ● Migration of adult worm through the tissues	● Blood smear-(thin and thick) collect between 10am-2pm. ○ Giemsa stain or H&E stain ● Worm can also be found in spinal fluid, urine, sputum, and lungs. - Made on the basis of a history of Calabar swellings (Fugitive swellings) or appearance of worm in conjunctiva, since microfilariae do not appear in the blood until years after the worms or the results of their activities become apparent.	● DEC - diethylcarbamazine ● Ivermectin ● Surgical removal of the worm in the eye ● Pest control ● Control breeding areas of the insect vectors

LIFE CYCLE OF LOA-LOA



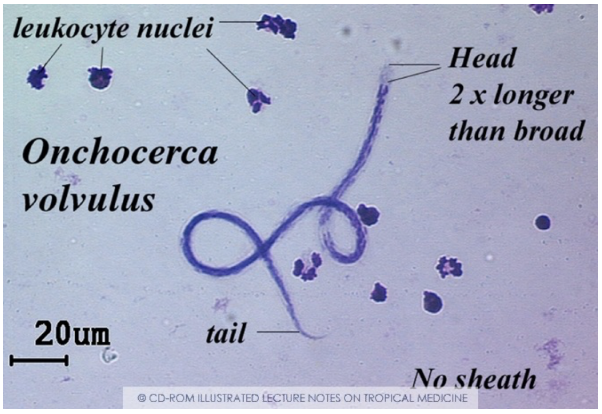
ONCHOCERCA VOLVULUS

ONCHOCERCA VOLVULUS p. 294	

- Also known as the "Blinding Worm"

DIAGNOSIS
→ Nodules - encapsulation of adult worm in a fibrous tissue tumor-like mass

- Widely distrbuted throughout **Central Africa** – also present in Saudi Arabia, Yemen, and in the Western Hemisphere
- Introduced to the Americas by the **slave trade**
- Intermediate host and vector: species of **Simulium** (blackfly or buffalo gnat)
    - Widespread in distribution
    - Only certain species are suitable vectors due to feeding habits and/or other reasons



- severe dermatitis
- microfilaria in ocular structures may result in blindness (trachoma)
- leading cause of blindness in Africa

1. Upon biting an infected person, the **simuliid** ingests *microfilariae* which have a <u>developmental cycle</u> in the insect similar to other filarial larvae
2. Transforms into infective forms that may enter a new host when the simuliid takes **another blood meal**
3. After introduction to the new host, developing worms wander through **subcutaneous tissues** but settle down, usually in groups of **two or more**
4. Most worms become encapsulated.
5. Nodules (produced by encapsulation) usually form **within a year after infection**
    a. Most frequently subcutaneous but occur in connective tissues deeper in the body.
6. Nodules range from a few milliliters to several centimeters in diameter and may be numerous.
    a. In Venezuela and Africa, majority of nodules located on the patient's **trunk or limbs**
    b. Few are found on the head
    c. In Mexico and Guatemala they are seen on the patient's scalp

PATHOGENESIS OF ONCHOCERCA VOLVULUS

CLINICAL SIGNIFICANCE
1. Encapsulation in onchocercoma is the universal fate of Onchocerca volvulus. 2. Some worms, especially during ithe early stages of disease in young children and in lightly infected persons **produce no apparent tissue reaction** – these worms are found free in the tissues. a. Dermal lesions - allergic reaction / can be duplicated by DEC, resulting to death in many microfilariae in the skin b. These reactions can be suppressed with corticosteroids 3. Loss of elasticity of the skin in the pelvic region - occurs not only in the hanging groin, but in a high prevalence of inguinal and femoral hernias. 4. Deposition of immune complexes in tissues as a result of the constant antigenic stimulation provided by the microfilariae - this is **basic to the inflammation** a. Obstructive lymphadenitis follows

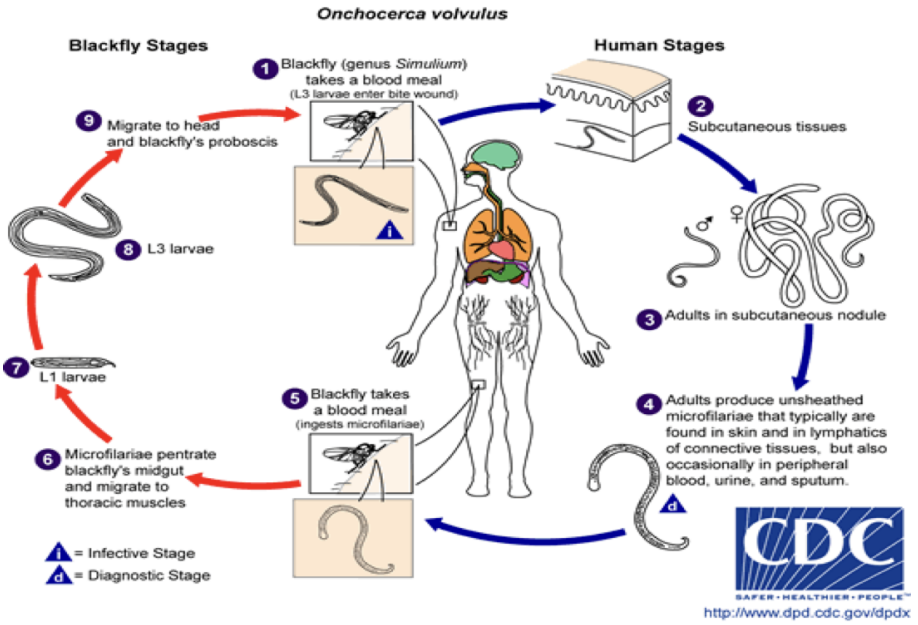
MORPHOLOGY OF ONCHOCERCA VOLVULUS

Male adult worm	***Female adult worm***	***Microfilariae - Diagnostic stage***
Wirelike, whitish adult worms lie coiled within fibrous tissue capsules ● Shorter than females, not more than 5 cm	● As long as 50 cm, but less than 0.5 mm in diameter.	- Only pathogenic tissue nematode which is not sheathed - no nuclei at the tip of the tail - found in nodules under skin, not in peripheral blood ● Make their way out of the nodules and migrate actively through the dermis and in the connective tissues, not *only* in the vicinity of the nodules - but at some distance from them.

PATHOLOGY OF ONCHOCERCA VOLVULUS

Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/ Care***
● Onchocerciasis ● Onchocercosis ● River blindness	● Nodules ae *not* painful ● Importance of infection lies in the effects produced by the microfilariae of the adult worms ● Accute inflammatory reaction in face, eyes, ears, neck, shoulders, body ○ Mexico and Guatemala ● Skin is hot, edematous, painful ● Associated pruritus ● Inflammation subsides slowly and may recur many times, resulting in **permanent thickening** of the skin ● Skin has violaceous color ○ This reaction is caused by the death of microfilariae in the skin and liberation of antigenic materials from them ● Onchodermatitis ● Atrophy of skin	- demonstrate from **skin snips**/tissue scrapings unsheathed microfilariae with no nuclei in tail - If skin snips reveal **no microfilariae, MAZZOTTI TEST** is done → 50mg DEC (diethylcarbamazine), that provokes intense pruritus within a few hours. ● Itching can be controlled by short-term administration of corticosteroids and subsides within 2-3 days without treatment.	● similar to other filariasis

LIFE CYCLE OF ONCHOCERCA VOLVULUS



Microfilae	Disease	Arthropod Vector	Diagnostic stage found in
Wuchereria bancrofti	Elephantiasis	Mosquito (Culex/Anopheles)	Blood
Brugia Malayi	Elephantiasis	Mosquito (Mansona)	Blood
Loa loa	Calabar swelling Blindness	Fly (Chrysops)	Blood
Onchocerca Volvulus	River blindness	Fly (Simulian)	Tissue from nodule

LIFE CYCLE OF TISSUE NEMATODES

1. Ingestion of the microfilariae from the blood or tissues by a blood-sucking insect.
2. The metamorphosis of the microfilaria in the arthropod vector st into rhabditiform larva and then into an infectious filariform larva.
3. The transfer of the infective larva to the skin of a new host by the proboscis of the biting insect.
4. The development of the larva after entry to the bite wound into a mature worm at its selective site.

NB: TPE or tropical pulmonary eosinophilia is associated with the microfilariae of these nematodes

DRACUNCULUS MEDINENSIS

DRACUNCULUS MEDINENSIS p. 302	ADDITIONAL INFORMATION
COMMON NAME ● "Guinea worm" ● "Fiery serpent" ● "Medina Worm" ● Important parasite in ME (SA, Iran, Yemen), central India, Pakistan, Africa. ● North America-parasites of dogs and other carnivores ● parasite is frequently found in the subcutaneous tissues and muscles of humans, dogs, and sometimes cattle and horses. ● The disease causes cutaneous nodules and subsequent ulcers.	● NOT A TRUE FILARIA ○ Worms elongate, females ● **INGESTION OF WATER contaminated by the presence of infected** Copepods, or water fleas, become infected by the **consumption of dracunculid larvae liberated** into the same water sources by infected persons (or other mammalian hosts)

PATHOGENESIS OF DRACUNCULUS MEDINENSIS

CLINICAL SIGNIFICANCE
- One or many worms may be seen at one time, in any part of the body   - Usually on the legs or feet - Majority of infections consist of a single worm, but in endemic areas repeated reinfection is the rule. - Presence of maturing worms give rise to *mild allergic symptoms such as* ***urticaria*** - When gravid female seeks close to the skin - localized erythema and tenderness in the area where the ulcer will form - 40% patients experience severe disability, 6 weeks average, 1% suffer permanent damage from **ankylosis of a joint**

MORPHOLOGY OF DRACUNCULUS MEDINENSIS

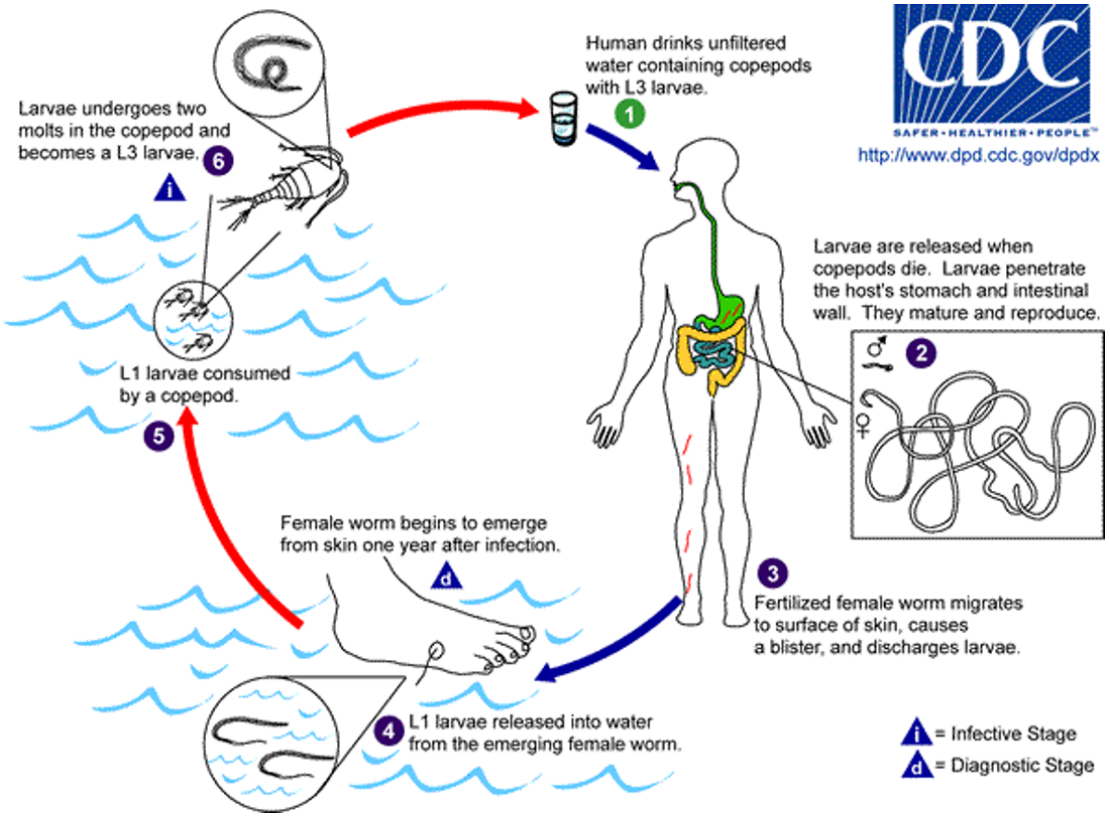
Male adult worm	***Female adult worm***	***Larvae***
→ Males are small (1.2-2.9 cm long)	→ Females measures 60cm in length → measure up to a meter or slightly more in length but averaging less → Diameter of >2mm. - Migrate to the subcutaneous tissue when they are pregnant/gravid → Body of fully pregnant female worm is filled by uterus *distended* with larvae **Papule** is produced in the skin where the head of the female lies just under the dermis - this becomes **vesicular** ● Finally ulcerates, exposing the worm.	● The larvae, which measure between 500 and 700 micrometers, can live for 6 days in clean water and 2 to 3 weeks in muddy water. ● Have a well-developed digestive tract and are **never found** in the blood or tissues of the host ○ Discharged directly into water ● If ingested larvae mature in **2 weeks** ● If infected copepods are swallowed, contained larvae are liberated to **penetrate through the digestive tract** ○ Entering the deep connective tissue where they mature ○ Maturation takes about 1 year
 *INTERMEDIATE HOST - CYCLOPS*		 **LARVAE** 

PATHOLOGY OF DRACUNCULUS MEDINENSIS

Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/ Care***
● Dracunculiasis (Guinea Worm	● 50% lesions become infected	- Usually presents no problems in	● Surgical removal of

Disease) **Acquired through ingestion of water contaminted by presence of infected copepods** (intermediate host) 	• Urticaria • Pruritus ○ Nausea, vomiting, diarrhea or asthma attacks - These disappear with appearance of ulcer, drainage of fluid that has formed around the female worm - Initial discharge of larvae	endemic areas	Worm • Metronidazole/ Thiabendazole - does not kill but facilitates removal • Mebendazole- kills them • Filtering water sources • Avoid contact with contaminated water

LIFE CYCLE OF DRACUNCULUS MEDINENSIS



- Infective Stage - 3rd stage larva
- Definitive host - humans, dogs, horses
- Intermediate host - Cyclops

ANGIOSTRONGYLUS CANTONENSIS

ANGIOSTRONGYLUS CANTONENSIS p. 312	ADDITIONAL INFORMATION
• "Rat lungworm"- zoonotic disease Was first discovered in pulmonary arteries and the heart of domestic rats in Canton, China in 1935 (Chen) • The worm normally lives in the lungs of rats but can cause eosinophilic meningoencephalitis in man • Infection is increasing in rats and bandicoots in the tropics and subtropics • First reported in the U.S. in 1987	• Slender worm up to 25 mm long • Larval stages develop in slugs and land snails ○ When eaten by rats, larvae migrate to the meninges and develop in the brain for about a month. • Young adults migrate to the pulmonary artery where they attain maturity. • Incidence of infection in rats and snails is high • **DOES NOT COMPLETE DEVELOPMENTAL CYCLE IN HUMANS**

	• When third stage larvae are ingested, they penetrate into **blood vessels** in the intestinal tract and are carried to the meninges but are unable to migrate to the lungs (Same with rats) • In rare cases, they develop to the young adult stage in the meninges – but then die ○ Inflammatory reaction provoked by dead worms that cause signs and symptoms of human infection.

PATHOGENESIS OF ANGIOSTRONGYLUS CANTONENSIS

STAGES
- Little information on its effect on CNS since patients recover - Sections of immature angiostrongylus cantonensis found in cerebrum and cerebellum as well as spinal cord   - Associated with infiltrates of eosinophils, monocytes, and foreign-body giant cells

MORPHOLOGY OF ANGIOSTRONGYLUS CANTONENSIS

Male adult worm	***Female adult worm***	***Ova***
• 16-19mm x 0.26 mm in diameter • pale, filiform (slender), delicate worm • They have a well develop caudal bursa (kidney shaped, single lobed) • they have long spicule	• 21-25 mm x 0.30-0.36 mm in diameter • has uterine tubules w/c is intertwined around the intestines giving it a barber-pole appearance • can lay 15,000 eggs daily	• 46-48 um × 68 um • Thin shelled • unembryonated when oviDosited (laid)
 Tail of adult male, showing copulatory bursa and long spicules (arrows).	 Adult female worm with characteristic barber-pole appearance anterior end of worm is to the top).	 *Fig. 1.* A. cantonensis *egg.*

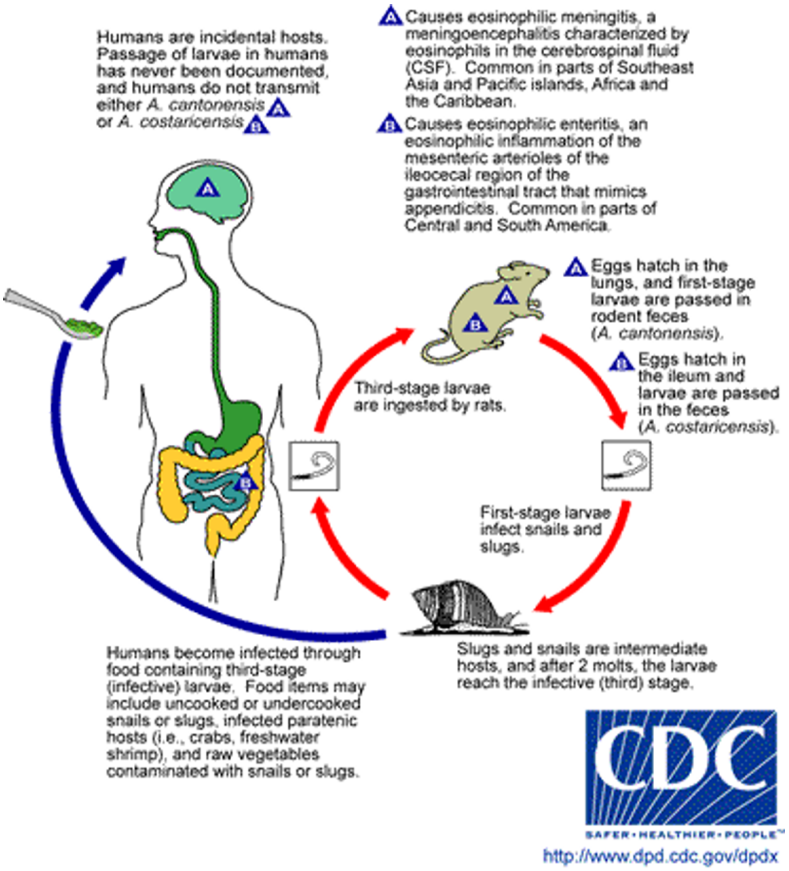
PATHOLOGY OF ANGIOSTRONGYLUS CANTONENSIS

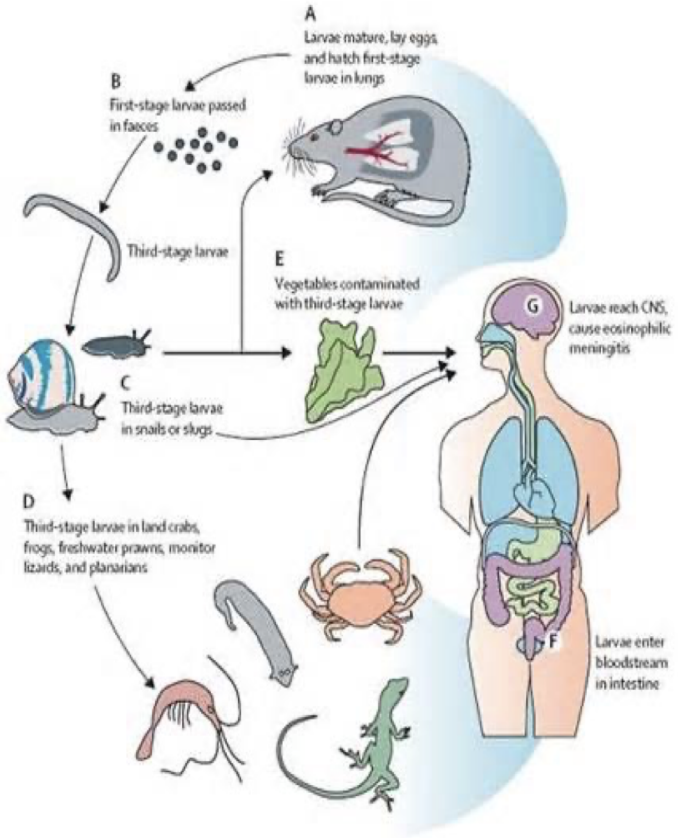
Disease	***Clinical Features (Signs and Symptoms)***	***Diagnostic (Dx) test***	***Treatment/Prevention/Care***
• Eosinophilic meningitis • Ingestion of IH/Paratenic Host infected w/ 3rd stage larva	• presence of worms in the blood vessels of the brain and meninges and free wandering worm in the brain tissue • severe headache, fever, paralysis of the lower extremities, stiff neck, coma, and death	• relatively difficult to diagnose • History of travel to endemic areas/ food habits-presumptive diagnosis • CSF exam-increased	• No tx available • proper cooking of meat and fish- (never eat raw and insufficiently cooked meat and fish) • elimination of IH • wash vegetables properly • Thiabendazole and mebendazole have some

	• destruction of the brain and spinal cord cells by trauma and immune response due to dead worms results in vague symptoms • Eosinophil counts are high in peripheral blood and CSF as well as lymphocytosis in CSF.	number of eosinophil and lymphocytes. Identification of immature worms • CT scan • ELISA (Enzyme-linked Immunosorbent Assay)	effect in animal infections • Thiabendazole is ineffective in humans

LIFE CYCLE

- Infective stage - 3rd stage larva
- Definitive host - rats
- Incidental host - Humans
- Intermediate host - snails, slugs (Achantina fulica)
- Paratenic host- Fresh water shrimps, land crabs, frogs





GNATHOSTOMA SPINIGERUM

GNATHOSTOMA SPINIGERUM p. 315	**ADDITIONAL INFORMATION**
• 1836, Richard Owen-British anatomist • In the stomach wall of a tiger that had died in the London zoo • Found in tropical wet environment • Common in Japan, Thailand, China, Malaysia, Indonesia, Philippines, Israel, Mexico, Tanzania. • Raw and pickled fish are part of the diet • Stout and pink in color • The swollen head bulb is covered with 4 circles of stout spines • Anterior half of the body is covered with transverse rows of flat toothed spines. Followed by a bare portion • The posterior tip of the body has numerous tiny cuticular spines	• **ACQUIRED THROUGH INGESTION OF RAW** insufficiently cooked or fermented fresh-water fish or amphibians, or paratenic hosts such as birds and snakes ○ Especially in the form of sashimi or ceviche (lime-marinated fish salad) • When ingested by humans, larvae does not mature but migrate throughout the body **INVOLVES 2 INTERMEDIATE HOSTS** 1. Copepod 2. Any of a number of fresh-water food fish, frogs, snakes, or birds **LARVAL WORMS** → **4mm** long with numerous spines on the head and body - encyst in **second intermediate host** ➜ **ADULT WORMS ARE FOUND IN DOGS AND CATS** • In their alimentary tract

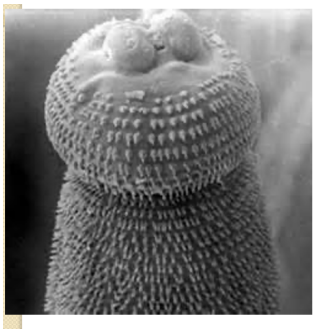
PATHOGENESIS OF GNATHOSTOMA SPINIGERUM

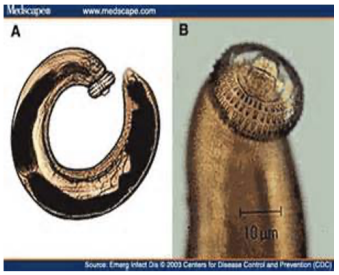
CLINICAL SIGNIFICANCE
- Few days after ingestion, migration of the larvae *through the intestinal wall* and into the abdominal cavity may produce epigastric pain, fever, vomiting, and anorexia - Persists for several weeks - Symptoms clear when the characteristic cutaneous manifestations begin • Circumscribed patches of edema (in the abdomen) last a few days and recur at different sites

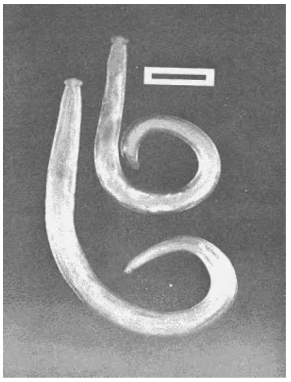
- Eosinophilia between 35% to 80% is reported in patietns with cutaneous involvement
- Eosinophilic myeloencephalitis may result from invasion of CNS by migration of worms along nerve tracts

MORPHOLOGY OF GNATHOSTOMA SPINIGERUM

Male adult worm	*Female adult worm*	*Ova*
• 11-25mm long • has pseudobursa w/ 4 pairs of peri-anal papillae and 2 unequal spicules	• 25-54 mm long • has 2 uteri and a vulva that is situated in a slightly post equatorial position	• 65-70 um x 30-40 um • Ovoidal transparent, superficially pitted • mucoid plug is found in one end • unembryonated when deposited



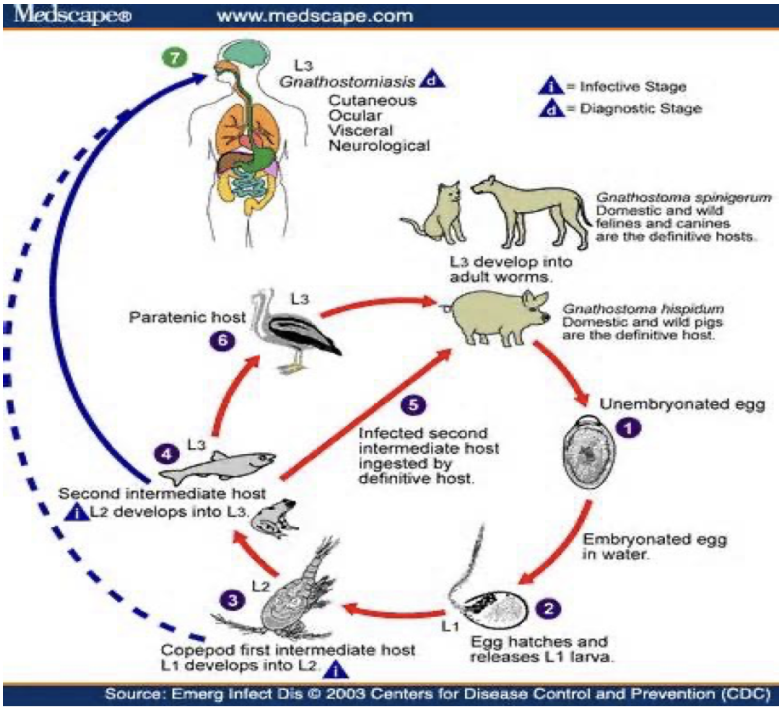




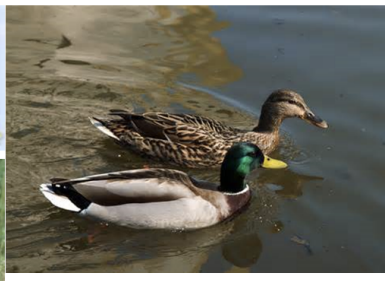
PATHOLOGY OF GNATHOSTOMA SPINIGERUM

Disease	*Clinical Features (Signs and Symptoms)*	*Diagnostic (Dx) test*	*Treatment/Prevention/ Care*
• Gnathostomiasis • Ingestion of IH/PH infected w/ 3rd stage larva	• A few days after ingestion- larva migrates in the intestinal wall and into the abdominal cavity • epigastric pain, fever, vomiting, anorexia • • CLM (Cutaneous Larva Migrans) -- creeping eruptions- swollen red trails- pruritus, rash, and stabbing pain • erratic migration- eye (blindness), brain, spinal cord (eosinophilic myeloencephalitis)- sudden severe nerve root pain, paralysis, sensory impairment	• CT scan- sub-arachnoid hemorrhages • presumptive diagnosis- patient's history and food habits • Biopsy- recovery and identification of the worm	• Albendazole • Ivermectin • Surgical removal of the worms from the subcutaneous. • Proper cooking of meat and Fish

LIFE CYCLE OF GNATHOSTOMA SPINIGERUM



- Infective stage - 1st to 3rd sttage larva
- Definitive host- Domestic and wild felines and canines, swine
- Incidental host-Humans 1st - Intermediate host- copepods
- 2nd Intermediate host- fresh water fish, frogs
- Paratenic host- crustaceans, freshwater fishes, amphibians, reptiles, birds and mammals

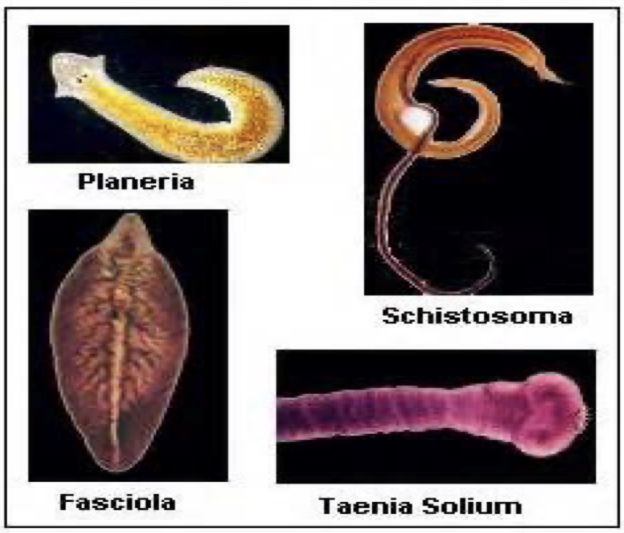



- Opichephalus argu, Anas platyrhynchos, Gallus domesticus

FLATWORMS

CLASS CESTODA – Tapeworms (9)
Diphyllobothrium latum
Dipylidium caninum
Hymenolepis spp. (Hymenolepis nana, Hymenolepis diminuta)
Taenia spp. (Taenia solium, Taenia saginata, Taenia asiatica)
Echinococcus granulosus

CLASS TREMATODA – Flukes (13)
Schistosoma spp.
Fasciola hepatica
Fasciola gigantica
Clonorchis sinensis
Opisthorchis viverrini
Opisthorchis felineus
Dicroceolium dendriticum
Fasciolopsis buski
Echinostoma ilocanum
Heterophyids - minute flukes (Heterophyes heterophyes, Megatonimus yokogawai, Haplorchis yokogawai)
Haplorchis taichui
Paragonimus westermani



GENERAL CHARACTERISTICS

- collectively called the **FLATWORMS**

SHAPE
- They are usually leaf-shaped, oval, elongated
- dorsoventrally flattened, bilaterally symmetrical

SIZE
- Size ranges from nearly **microscopic to over 60 meters in length**.

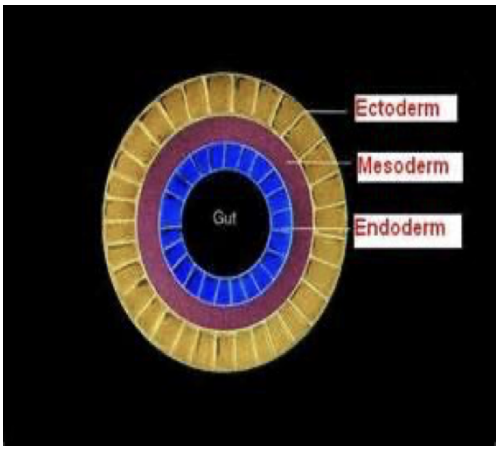
BODY
- Body is covered by 3 layers of tissue with organs and organelles
- They are covered by a membrane called tegument (microvilli that absorbs food from the host's intestine)
- Body contains no internal cavity
- They **do not have circulatory, respiratory, skeletal system, digestive system is incomplete while absent in others, lacks an anus**. (flame cell-protonephridium)
    - A *flame cell* is a specialized excretory cell found in the simplest freshwater invertebrates, including flatworms, rotifers and nemerteans; these are the simplest animals to have a dedicated excretory system. Flame cells function like a kidney, removing waste materials. **Bundles of flame cells are called protonephridia**.

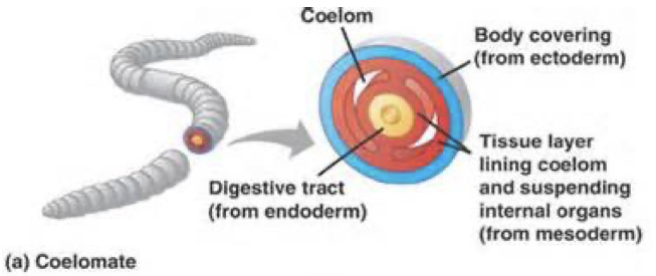
HEAD
- Head is provided with sensory organs

SEXUAL CHARACTERISTIC
- They are hermaphrodite(sexes are combined in a single organism), **except for Schistosoma species**

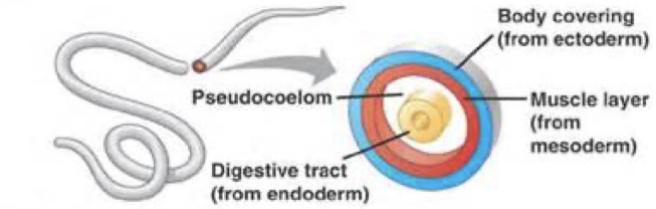
HABITAT
- Because of their elaborate nervous system, they are found in a wide variety of habitat. (lakes, streams, ponds, ocean sediments from pole to pole, moist terrestrial environments.)

CROSS SECTIONAL DIAGRAM OF FLATWORMS

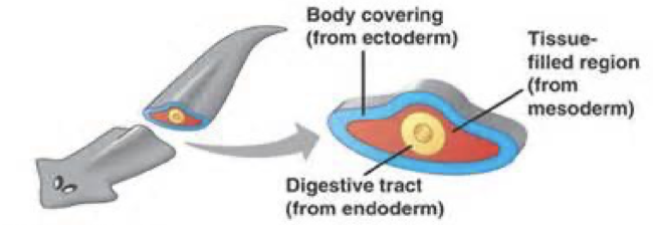




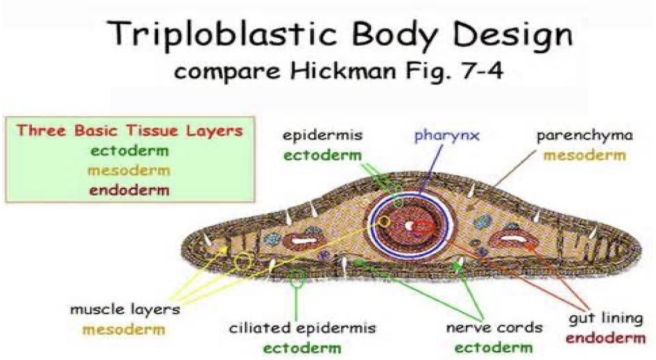
(a) Coelomate



(b) Pseudocoelomate



(c) Acoelomate



TRIPOBLASTIC BODY DESIGN

THREE BASIC TISSUE LAYERS

1. **Ectoderm**
2. **Mesoderm**
3. **Endoderm**

- **Epidermis** → **ectoderm**
- Pharynx
- Parenchyma → **mesoderm**
- Gut lining → **endoderm**
- Nerve cords → **ectoderm**
- Ciliated epidermis → **ectoderm**
- Muscle layers → **mesoderm**

CLASSES OF PHYLUM PLATYHELMINTHES [TABLE]

CLASS AND EXAMPLES	MAIN CHARACTERISTICS
Turbellaria (mostly free-living flatworms, e.g. Dugesia)	Most marine, some freshwater, a few terrestrial, predators and scavengers; body surface ciliated
Monogenea (monogeneans)	Marine and freshwater parasites; most infect external surtaces of fishes: life history simple; a ciliated larva starts an infection on a host
Trematoda (trematodes, also called *flukes)*	Parasites, almost always of vertebrates; two suckers attach to host; most life histories include intermediate hosts
Cestoidea (tapeworms)	Parasites of vertebrates; scolex attaches to hast; proglottids produce cegs and break off after fertilization: no head or digestive system; life history with one or more intermediate hosts

CLASS CESTODA - TAPEWORMS

- Adult worm inhabit the small intestines of vertebrates while the larvae parasitize different classes of vertebrates and invertebrates
- Digestive system absent, food is acquired through absorption from the host's intestine.
- Ribbon like or tape like segmented parasites varying in size from a few mm. to several meters
- Body consist of 3 distinct regions :

HEAD OR SCOLEX

organ of attachment provided with suckers (bothrium or acetabulum), rostellum (armed with hooklets or unarmed)

NECK

region of growth (immature proglottids)

STROBILA OR BODY

consists of series of segments (proglottids)

- These are complete reproductive unit

Composed of 3 regions:

1.) Immature
2.) Mature
3.) Gravid

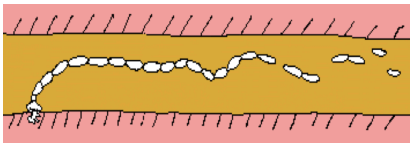
- Most require Intermediate host to complete their life cycle

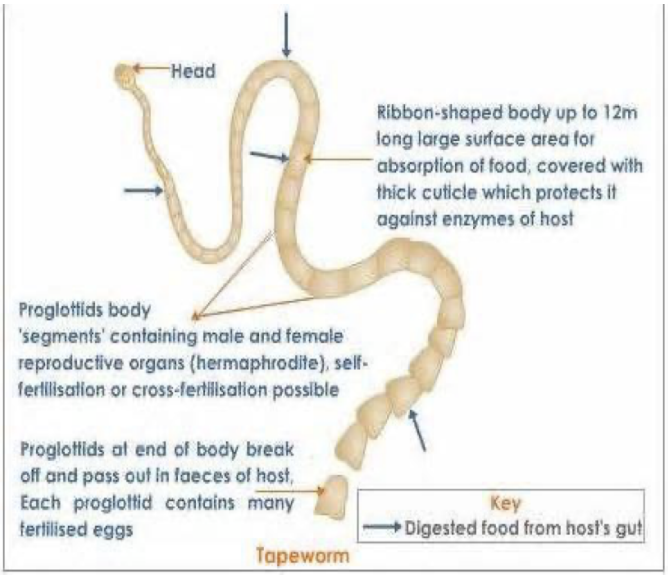
LIFE CYCLE OF CLASS CESTODA

Egg stage	Hexacanth embryo (Oncosphere)
Larval stage	Coracidium, Procercoid, Plerocercoid, Cysticercoid, Cysticercus larva
Adult stage	

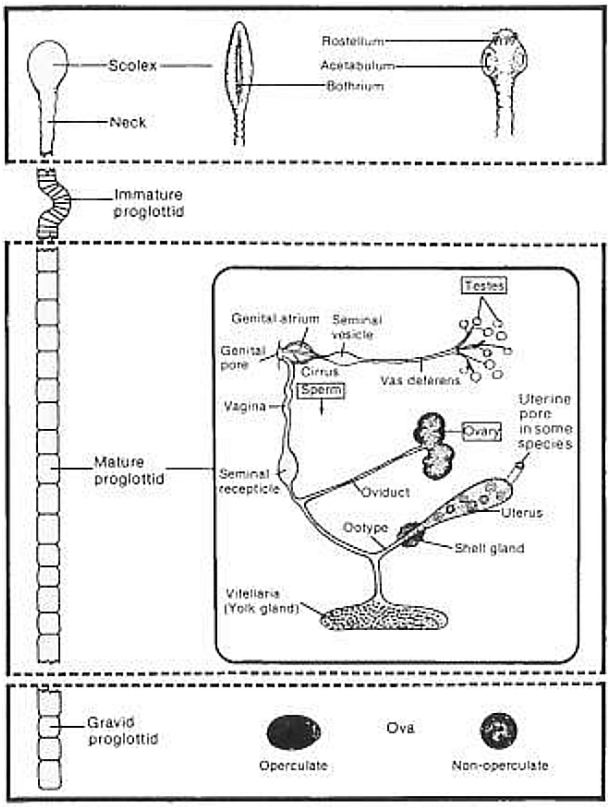
3 TYPES OF PROGLOTTID

Immature	Undeveloped sexual organ, nearest to the neck
Mature	Fully mature sexual organ
Gravid	Contains fertilized egg, undergoes

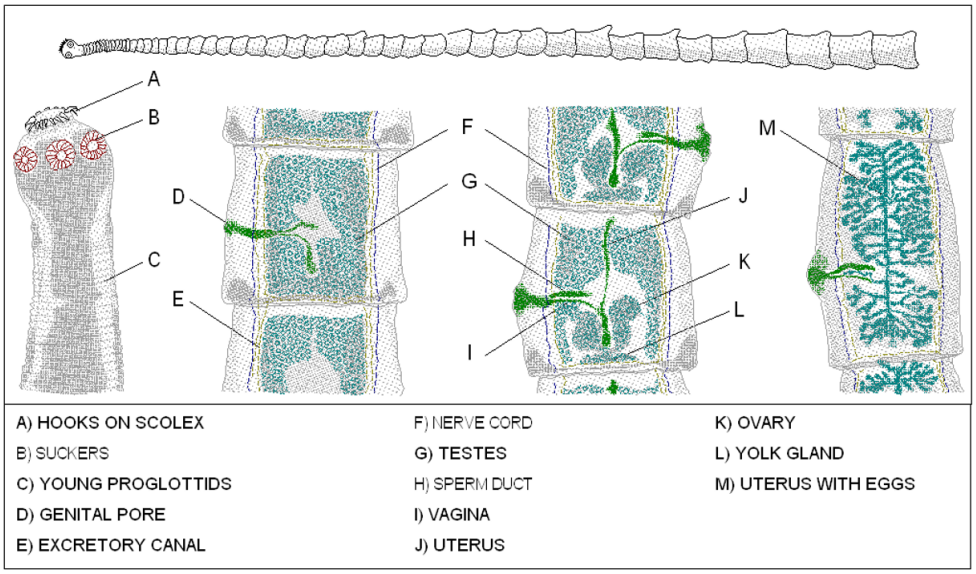
	apolysis ***APOLYSIS*** → normal process of detachment of the gravid proglottid from the adult worm 

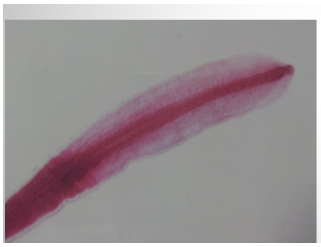
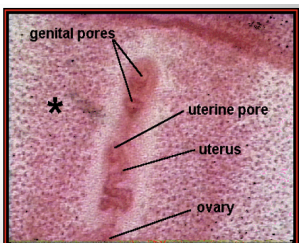
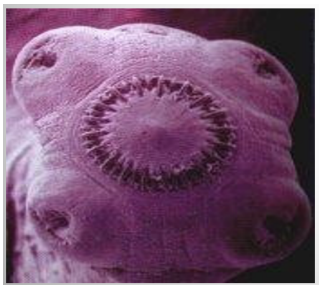
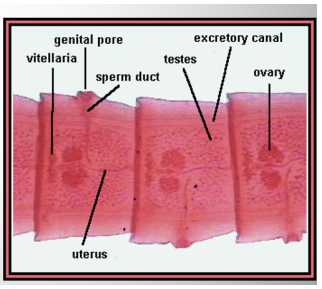
ADULT WORM OF CLASS CESTODA	EGG OF CLASS CESTODA
	

GENERAL STRUCTURE OF TAPEWORMS



GENERAL ANATOMY OF TAPEWORMS



ORDER	DESCRIPTION	OVA
ORDER PSEUDOPHYLLIDEA - scolex spatulate with bothria • Diphyllobothrium latum • Scolex • Segments - genital pore + uterine pore/ anapolytic	 	Operculated, Immature, requires aquatic vegetation to develop, coracidium 
ORDER CYCLOPHYLLIDEA – scolex globular with 4 cup like suckers • Scolex • **Segments contain no uterine pore/ apolysis**	 	Passed out readily with hexacanth embryo 

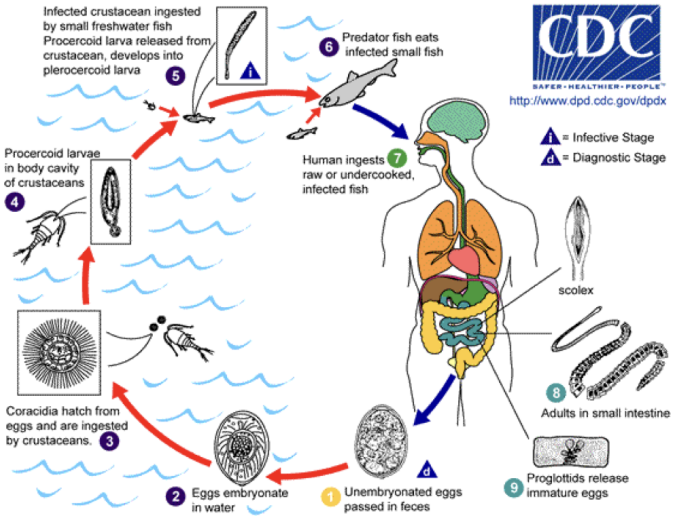
*CLASS CESTODA – **Species that require**…*	
Vertebrate Intermediate Host	• Taenia solium • Taenia saginata
Invertebrate Intermediate Host	• Hymenolepis diminuta

	• Dipylidium caninum
May or may not require Intermediate Host	• Hymenolepis nana
Species which infect man and their larval stage	• Echinococcus granulosus • Taenia solium • Diphyllobothrium latum

Diphyllobothrium latum

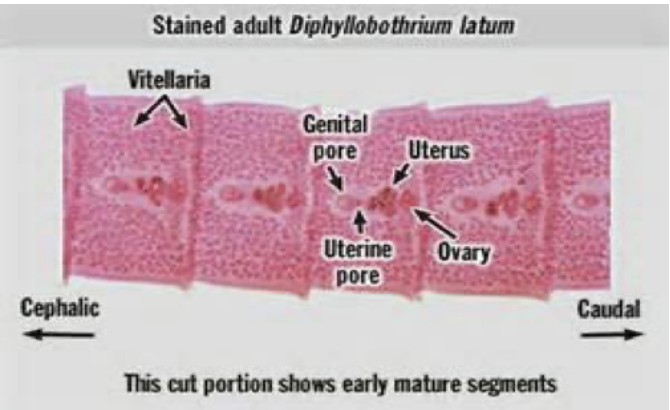
- **"Broad or Fish tapeworm"**
- Worldwide in distribution, occurring in Northern temperate areas of the world where pickled or insufficiently cooked fresh-water fish are prominent in the diet.
- High prevalence rate in humans is seen in Scandinavia, Finland, Alaska, Canada, and Japan
- Fish-eating mammals such as wild and domestic members of the dog and cat families, bears, minks, pigs, walruses, and seals.

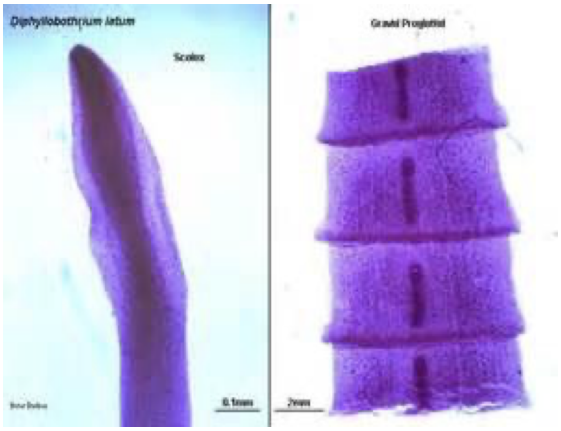
- **Infective Stage** → Plerocercoid larva
- **Definitive Host** → Man, dog, cat, and other fish-eating mammals
- **1st Intermediate Host** → copepod
- **2nd Intermediate Host** → Freshwater fish
- **Stage in man** → Adult worm
- **Habitat in host** → Small intestine



Morphology of Diphyllobothrium latum

ADULT WORM	OVA
Description → Long, tapelike, grayish yellow Size → 3-10 m ***SCOLEX* –** elongated, spoon-shaped (spatulate), with 2 longitudinal grooves (bothria) – with 2 suckers ***PROGLOTTIDS*** → 3,000 - 4,000 • Mature are **broader than** long • Uterus is centrally located and rosette shaped. Genital pore is located in the middle. • Can shed **1 million eggs** per day.	Description → has thin double-contoured shell with a lid (operculum) at one end, *which may or may not be open* Color → yellowish-brown Size → 70x50 um in diameter → Ripe eggs escape **through the uterine pore** and are discharged into the intestine. 





Pathology of Diphyllobothrium latum

DISEASE	• Diphyllobothriasis
MODE OF TRANSMISSION	• Ingestion of larvae in fresh-water fish
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Majority asymptomatic • Vague abdominal discomfort, diarrhea, nausea, weakness • proximal portion of the jejunum- tapeworm pernicious anemia due to Vit. B12 deficiency (absorbed by the worm) • **SPARGANOSIS** → drinking water containing copepods (procercoids). ○ Larva penetrates the intestine and find its way into the muscle or subcutaneous tissue to grow into a SPARGANUM larva — eosinophilia
DIAGNOSTIC TEST	• Direct Fecal Smear (DFS) → demonstration of eggs + proglottids • Serologic Test → Enzyme-linked Immunological assay (ELISA) • CT/MRI - cerebral • Biopsy
TREATMENT/ PREVENTION/ CARE	• Niclosamide • Praziquantel → Cooked fish properly, brine cured or frozen at -10° C for 24-48 ars. → cooked animal meat properly → use of potentially infected animals for medicinal purpose must be discourage → avoid drinking contaminated water

Pathology of Sparganosis

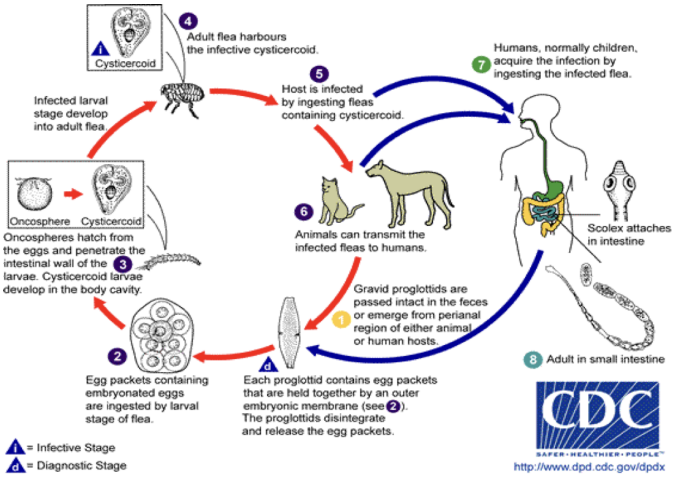
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	**SPARGANUM LARVA** → is a wrinkled, whitish, ribbon-shaped organism, a few mm wide and up to several cm. long Early migratory stage are asymptomatic but when it reached its final site and begins to grow, its presence elicit a painful inflammatory reaction in the surrounding tissue • Eyes → **ocular sparganosis**; intense pain w/ periorbital edema • CNS → **cerebral sparganosis**; seizures, paresthesias, hemiparesis

DIAGNOSTIC TEST	Surgical removal of the larva

Dipylidium caninum

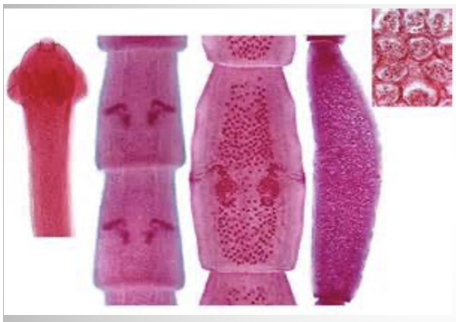
- "Dog and Cat flea tapeworm"
- Common parasite of domestic dogs and cats all over the world
- Occasionally found in humans, particularly small children
- Larva is known as cysticercoids
- The cysticercoid develops when the eggs/ova is ingested by the cat/dog flea larva and retained until it becomes an adult flea
- Infection takes place due to **accidental ingestion of fleas who kiss or are licked by their infected pets**.
- The gravid proglottid migrates to the anus and remains on the exterior to contract and expand releasing the packets of eggs in the anal area.

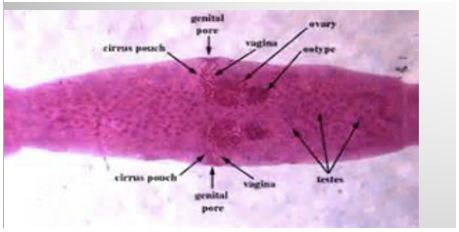
- **Definitive host** → dogs, cats, man
- **Intermediate host** → Fleas

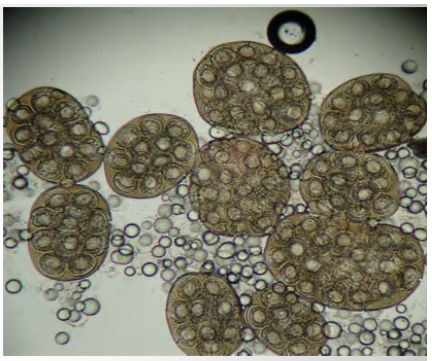


Morphology of Dipylidium caninum

ADULT WORM	OVA
Description → pale, reddish tape-like worm Length → 20-40 cm (avg. 15 cm) ***SCOLEX*** → small 250-500 um in diameter, bears 4 suckers with refractile rostellum, armed wih 3-4 rows of hooks shaped like rose thorns. Neck is short and thin ***PROGLOTTIDS*** → mature are longer than they are wide-vase-like or pumpkin seed-shaped containing 2 sets of reproductive organs. **Gravid** are saped like the mature segment (barrel-like) and are filled with polygonal uterine blocks • Egg packets containing 8-15 eggs enclosed in an embryonic capsule/membrane	Description → spherical, thin hyaline shell Hexacanth embryo Size → 23-40 um in diameter 







(Intermediate host → Flea)

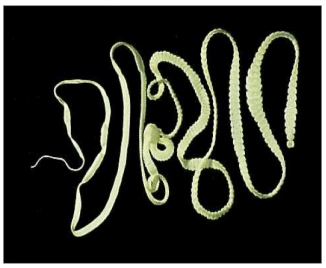
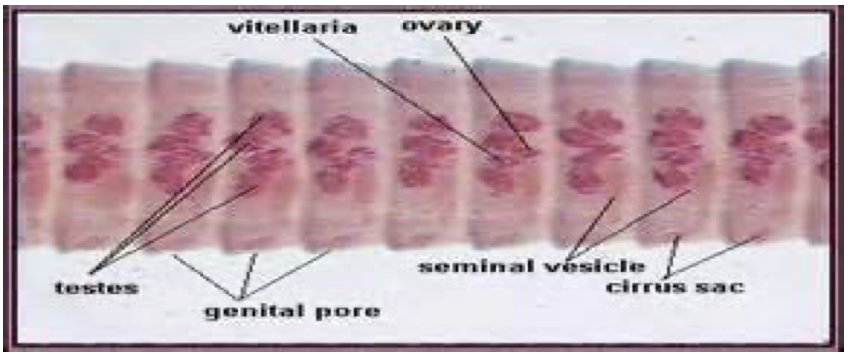
Pathology of Dipylidium caninum

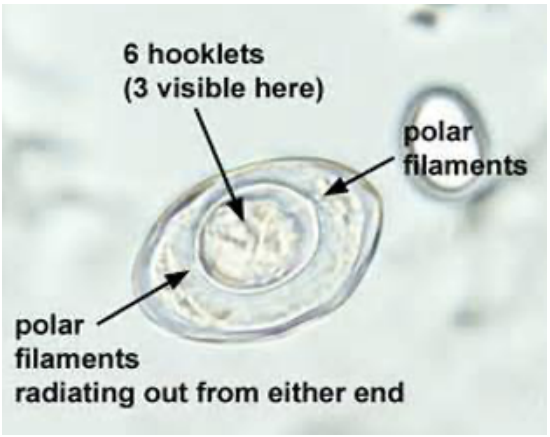
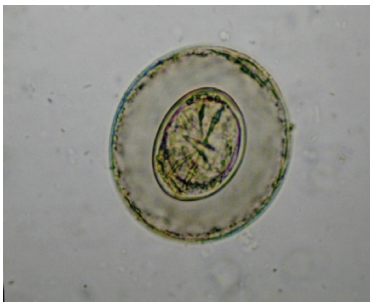
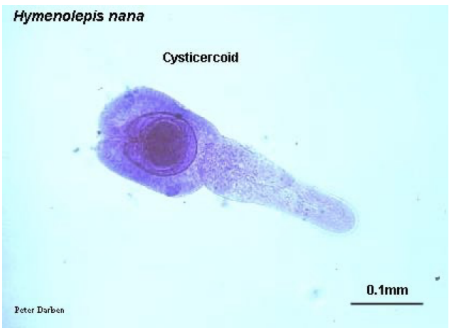
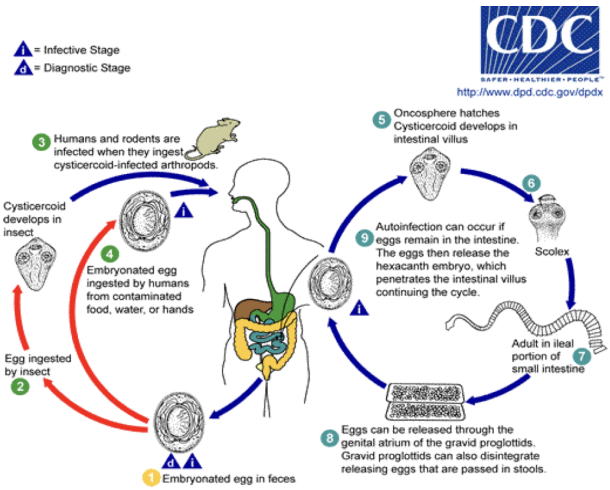
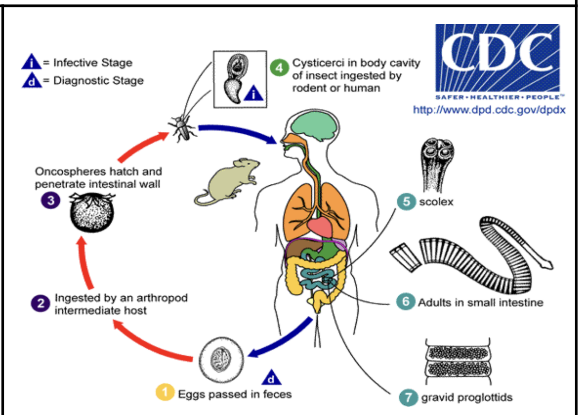
DISEASE	• Dipylidium infection
MODE OF TRANSMISSION	• Zoonoses → Ingestion of infected dog or cat flea ○ Scientific name of flea: Ctenocephalides canis / felis
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • abdominal pain, diarrhea • ***ANAL PRURITUS*** → proglottids are motile when freshly pass and might be mistaken for maggots or fly larva. • animals will scrape their anal region in grass or carpet to relieve itchiness.
DIAGNOSTIC TEST	• DIRECT FECAL SMEAR (DFS) → demonstration of egg packets or proglottids in stool
TREATMENT/ PREVENTION/ CARE	• Niclosamide • Praziquantel → Periodic deworming of dogs and cats → maintenance of dog and cat hygiene.

Hymenolepis spp. (H. nana and H. diminuta)

MORPHOLOGY OF HYMENOLEPIS SPP.

	Hymenolepis nana	**Hymenolepis diminuta**

	• **"dwarf tapeworm"**- small thread like • Common parasites of house mouse • Worldwide infection • Infection is seen most frequently in children but can occur in adults as well • May not require I.H. to complete its life cycle. **Infective stage** → Oncosphere/Cystecercoid larva **Definitive host** → Rats, mice, and man **Intermediate host** → (can be optional) Flea, grain or flour beetle, cockroaches **Stage in man** → Adult or larva **Habitat** → small intestine	• **"Rat tapeworm"**- Thread-like flatworms • Commonly parasites of rats • Worldwide infection • Requires an arthropod I.H. to complete its life cycle **Infective stage** → Cystecercoid larva **Definitive host** → Rats, mice, and man **Intermediate host** → flea, grain or flour beetle, cockroaches **Stage in man** → Adult **Habitat** → small intestine
ADULT WORM	Length: 1-4 cm, Width: 1mm 	Length: 10-60 cm, Width: 4 mm 
SCOLEX	• Globular with 4 suckers • Size → 0.3 mm in diameter • Provided with refractile rostellum armed with single row of 20-30 hooklets 	• Globular with 4 suckers • Size → 0.2-0.4mm in diameter • Provided with unarmed rostellum 
PROGLOTTIDS • Male • Uterus • Genital pore	• Up to 200 • Broader than long, 3 testes • Irregular sac-like • Lateral, on the same size	• 800-1000 • Broader than long, 3 testes • Irregular sac-like • Lateral, on the same side
		
OVUM (Oncosphere)	• broadly ovoid, covered with a thin, hyaline smooth outer shell. • Inner thick membrane with polar thickenings or	• Has a thick yellow outer and colorless (fine concentric striations) inner membrane with a granular intermediate

	knobs from which project 4-8 long, thin polar filaments. differential features from H. diminuta) • The embryophores contain 6 hooklets arranged in fan shaped pattern (3 pairs)- ***Hexacanth*** • Eggs are released as the gravid segments disintegrate, which measure 30-45 um in diameter  	layer. • Has no polar filaments • Embryophore contain 6 hooklets arranged in fan-shaped pattern 3 pairs) - Hexacanth • Eggs are larger, which measure 60-79 x 72-86 um in diameter.  
Cysticercoid larva	 	
Life Cycle		

Pathology of Hymenolepis spp.

DISEASE	• Hymenolepiasis
MODE OF TRANSMISSION	• Ingestion of **IH (diminuta)** or **oncosphere (nana)** ○ **Oriental rat flea -** Xenopsylla cheop**is** ○ **Dog flea -** Ctenocephalides canis ○ **Human flea -** Pulex irritans ○ **Flour beetle -** Tenebrio or Tribolium ○ **Sawtoothed grain beetle -** Ahasverus advena *Highlighted are the main IH of H. diminuta*
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • Enteritis • Abdominal pain, • headache, • diarrhea, • anorexia
DIAGNOSTIC TEST	• DIRECT FECAL SMEAR (DFS) → demonstration of embryonated eggs in stool (oncosphere)
TREATMENT/ PREVENTION/ CARE	• Niclosamide • Praziquantel • Nitazoxanide → Maintenance of personal hygiene → Environmental sanitation. (pest and insect control) → Proper storage of grain and cereals, especially those consumed w/ out cooking → Proper disposal of feces

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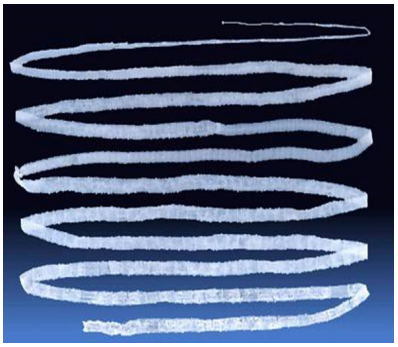
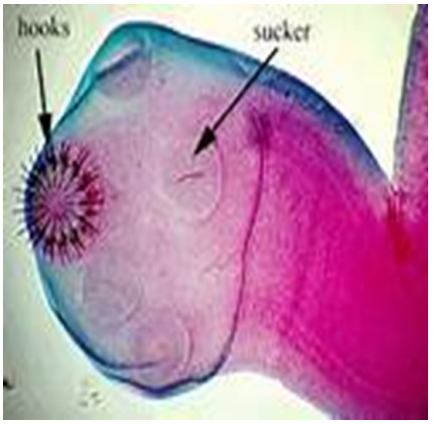
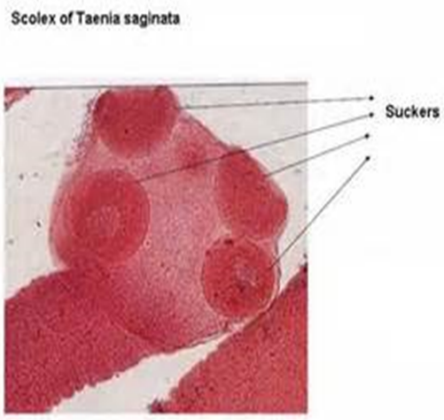
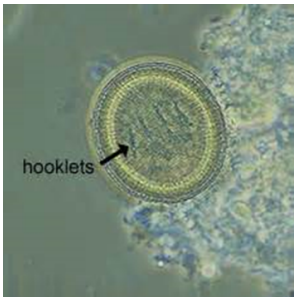
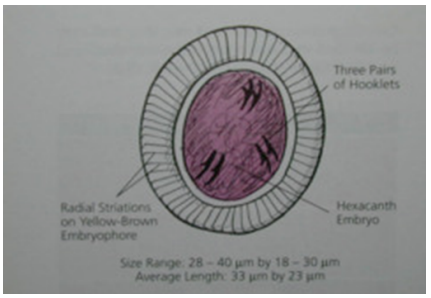
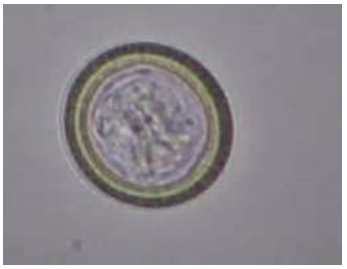
Taenia spp.. (T. solium and T. saginata)

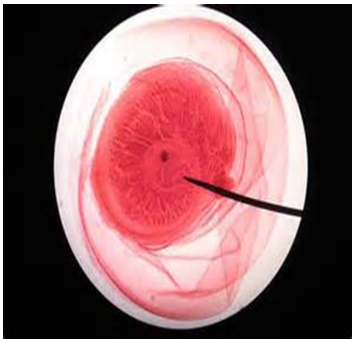
MORPHOLOGY OF HYMENOLEPIS SPP.

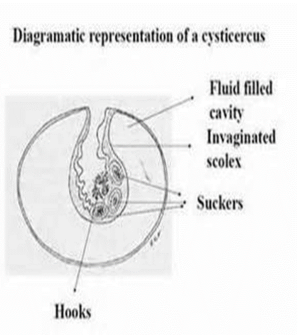
	Taenia solium	**Taenia saginata**
	• **"Pork tapeworm"** • Occurs when people eat cured or undercooked pork • Prevalent in Mexico, Latin America, Slavic countries, Africa, SE Asia, China **Infective stage** → Cysticercus cellulosae **Definitive host** → Man **Intermediate host** → Pig, Man, dogs, bears **Stage in man** → Adult or larva **Habitat** → Small intestine	• **"Beef tapeworm"**- Thread-like flatworms • Worldwide distribution • Acquired through ingestion of raw or undercooked beef • Frequent infections occur in the US • Life cycle similar to T. solium **Infective stage** → Cysticercus bovis **Definitive host** → Man **Intermediate host** → Cow **Stage in man** → Adult **Habitat** → Small intestine
ADULT WORM	• Whitish or creamy white, tape-like worm • 2-5 m but can reach up to 10 m	• White and semi-transparent • 5-10 m but can reach up to 25 m

		
SCOLEX	• Globular with 4 cup-like suckers • 1mm in diameter • Rostellum armed with 25-30 hooklets 	• Quadrate with 4 cup-like suckers • 1-2 mm in diameter • No rostellum 
PROGLOTTIDS • Mature • Gravid • Genital pore	• 800-1000 • Longer than broad, 12 mm x 6 mm • 7-13 lateral uterine branches • Lateral alternate sides ,irregular • Apolysis- in chains (30-50 K eggs) 	• 1000-2000 • Longer than broad • 15-30 lateral uterine branches • Lateral alternate sides ,irregular • Apolysis- singly (80K eggs) 
Ovum	• spherical, yellow to brown in color with a **thick radially striated embryonic shell** • oncosphere with 3 pairs of hooklets (Hexacanth) • 31-43 um in diameter	• resembles T. solium ova • **Indistinguishable from each other**

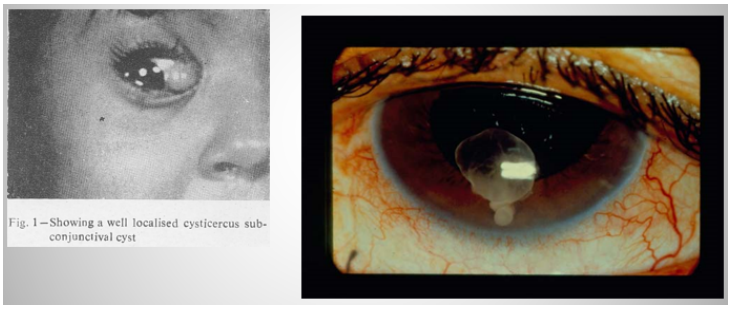
	  
Cysticercoid larva	Eggs ingested → penetrate IW and enter blood stream(hooklets) → lodge in any tissue-voluntary muscles (CNS-brain, eyes) ● Complete its development in about 2 months ● Semitransparent, opalescent white, and elongate in shape, measures 8-10mm x 5mm in diameter ● Scolex bears suckers and hooklets

Cysticercus cellulosae - **Bladder worm**

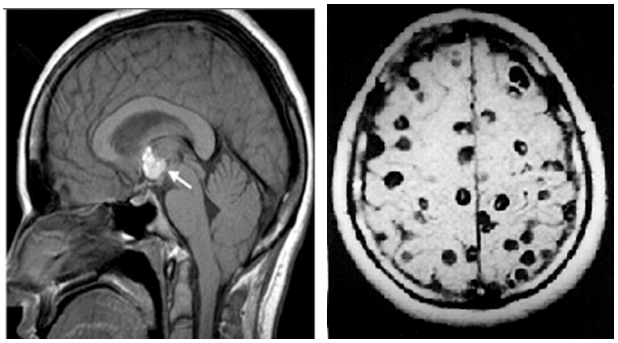




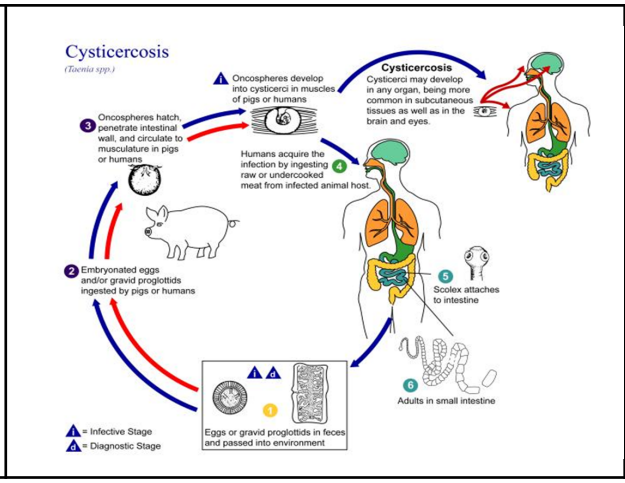
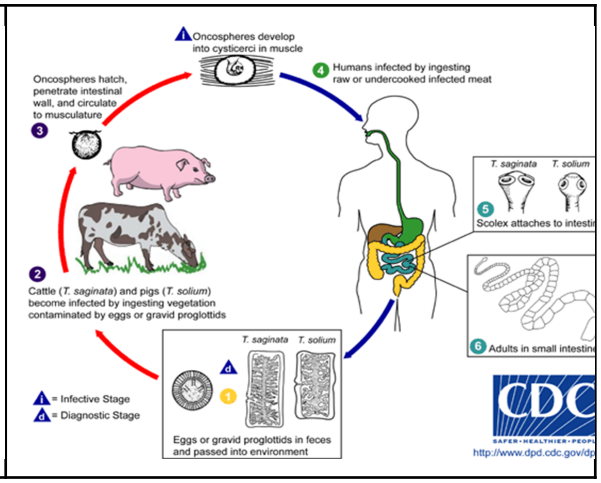
C. cellulosae in different parts of the body



Orbit of the eyes



Subarachnoid & Brain

Life Cycle		

Pathology of Taenia spp.

DISEASE	• Taeniasis
MODE OF TRANSMISSION	• Ingestion encysted larva in muscle **tissue of pork/beef** ○ Criteria for successful TX= recovery of scolex+ negative stool sample 3 months after Tx. • Cysticercosis → ingestion of eggs ○ Only for T. solium
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • Vague abdominal discomfort- abdominal pain, hunger pangs, chronic indigestion • anorexia, nausea • Reverse peristalsis- carry egg in the upper portion of duodenum • Bladder worm- encystment of the larva in the muscles, CNS-brain, eyes, subcutaneous tissue • Seizures,Hydrocephalus, visual problems
DIAGNOSTIC TEST	• DIRECT FECAL SMEAR (DFS) → demonstration of embryonated eggs in stool (oncosphere) or proglottids • X-ray, CT scan, MRI
TREATMENT/ PREVENTION/ CARE	• Albendazole w/ Dexamethasone increase the effectivity in subarachnoid cyst (brain) • Praziquantel- parenchymal cyst • surgical removal → proper cooking of meat → freezing -5°C for 4 days, -15°C for 3 days or -24°C for 1 day kills the larvae → proper disposal of feces to prevent contamination of food, water, soil

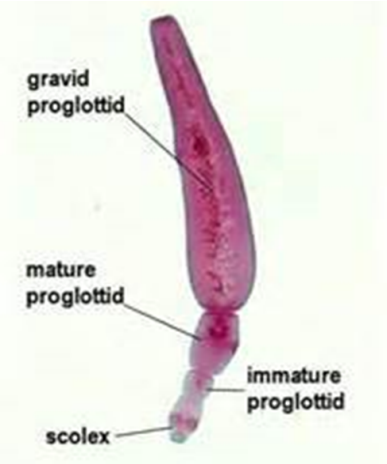
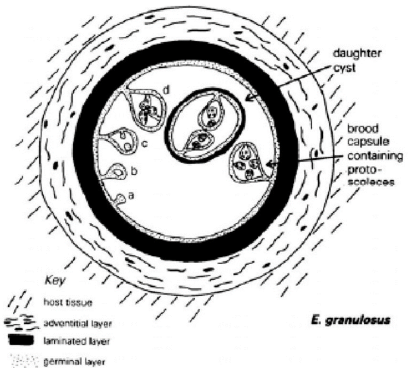
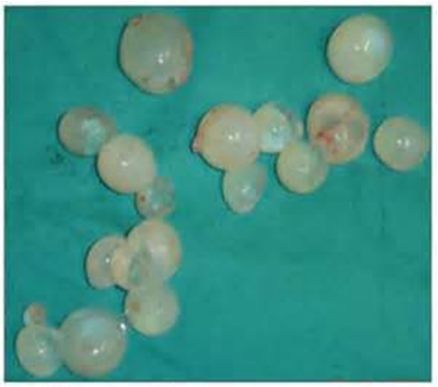
Taenia asiatica

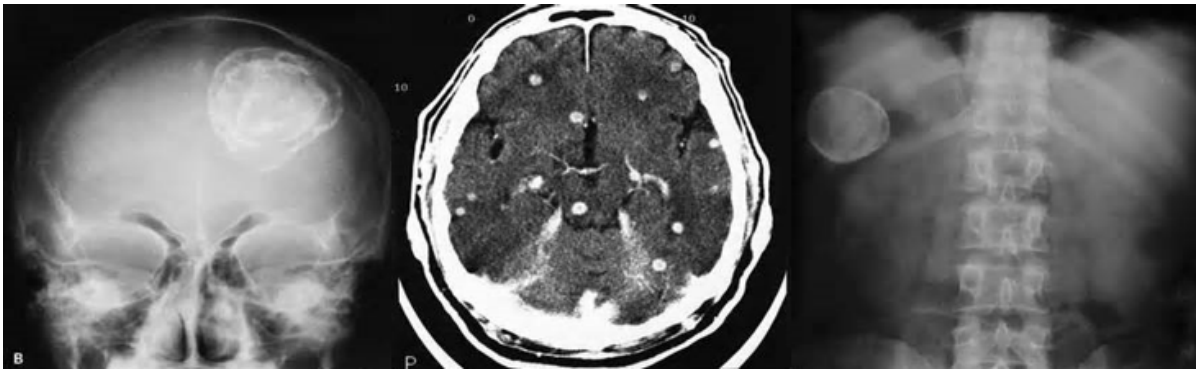
• First reported in Taiwan in 1980. Korea, China, Philippines, Thailand , Malaysia, and Indonesia • Raw or undercooked pork • Does not produce cysticercosis in humans. • Morphologically and genetically similar to T. saginata • Cysticerci develop in the liver and other viscera of pigs and not in the muscle	• **Definitive host** → Man • **Intermediate host** → Goats, Monkey, Wild Boar, Swine, Cattle
 *Scolex*	 *Proglottid*

Echinococcus granulosus

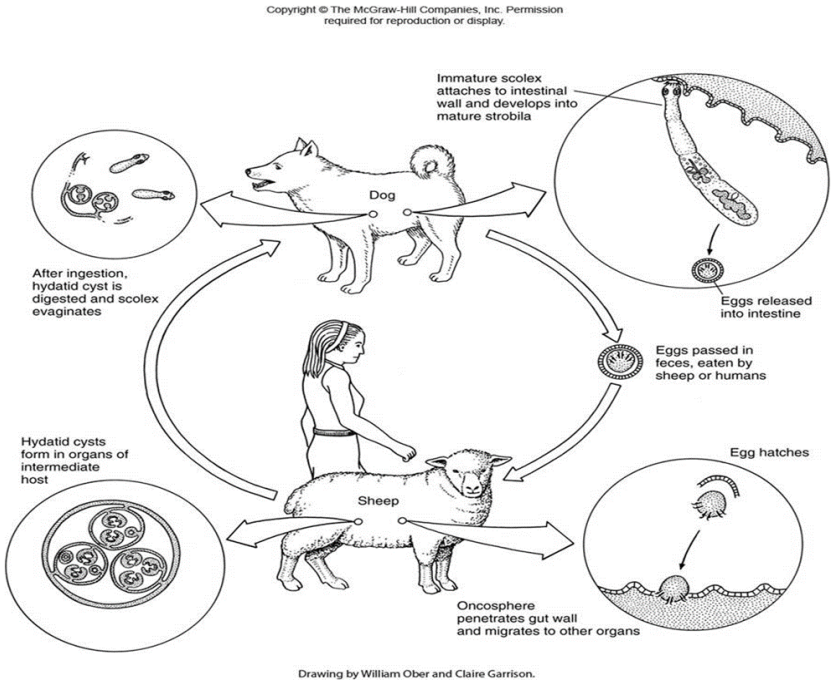
• "Hydatid worm" • Smallest tapeworm - scolex, a short neck and 3 proglottids • Causes **Hydatid cyst** that develops in the different organs and tissues of the host • 2 different life cycles - **PASTORAL** and **SYLVATIC** a serious infection in sheep-raising areas of Australia, New Zealand, North and South America, Europe, Asia, and Africa	• Goats, camels, reindeer, and pigs, together with dogs maintain the cycle in various parts of the world. • Humans are infected when they accidentally ingest the eggs, usually as a result of fondling dogs. **Definitive Host** → Dogs, Wolves or other carnivores **Intermediate Host** → Man, Sheep, horses, goats, cattle, pigs **Stage in man** → Larva **Location in man** → Various organs

Morphology of Echinococcus granulosus

ADULT WORM	HYDATID CYST	OVA
Description → small tapeworm; has only 3 segments Length → 3-6 mm in length ***SCOLEX*** → **globular- 0.6 mm in width with 4 cup like suckers. Rostellum bears a double crown of 28-40 hooklets**  ***PROGLOTTIDS*** • **Immature** - longer than broad with immature reproductive organs • **Mature** - longer than broad. Consist of 43-65 pyriform testes with irregular ovaries at the posterior portion • **Gravid** - 2-3 mm in L X 0.6mm in width- biggest segment. Median uterus w/ 12-15 lateral pouches filled w/ ova	**INFECTIVE LARVA** • embryo develops slowly into a cyst reaching a diameter of 1cm. in 5 mos. Reach 20 cms. in diameter Spherical in shape - **external laminated cuticle *(ectocyst)***. **Inner germinal layer *(endocyst)*** –masses of cells grow into the cavity of the cyst. Become vacuolated- Brood capsules containing Protoscolices bud from the inner wall. Daughter cyst appear w/in the hydatid. • **HYDATID SAND** - found in older cyst that contains granular materials of free protoscolices, daughter cyst.  	Similar to Taenia spp. ova   *Fig. 2. Hydatid cysts removed.* *Hydatid Cysts*



Radiograph slides



Life Cycle of E. granulosus

Pathology of Hymenolepis spp.

DISEASE	• Echinococcosis *(hydatid disease)*
MODE OF TRANSMISSION	• Ingestion of Eggs
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Effects may not become apparent for many years after infection bec. of its usual slow growth. (20 yrs.) • Primarily affects the liver, lungs, brain, bones, and other organs
DIAGNOSTIC TEST	• X-ray, Ct scan, MRI • Serologic test- ELISA & IHA
TREATMENT/ PREVENTION/ CARE	• Albendazole • Praziquantel • ivermectin • Surgical Removal • Aspiration of hydatid fluid → Maintenance of personal hygiene → Avoid close contact with dogs → Proper disposal of feces

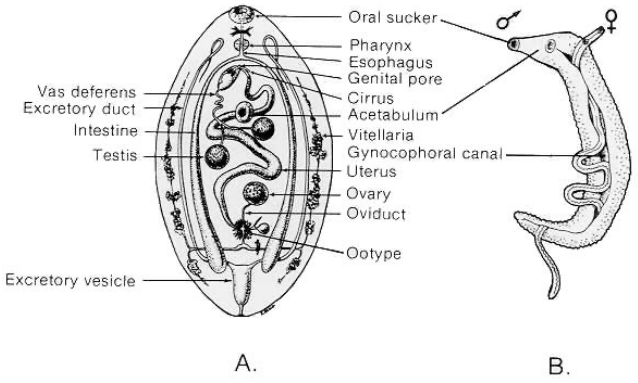
CLASS TREMATODA

- **DIGENETIC parasites**- are among the most common and abundant of parasitic worms
- Second only to nematodes in their distribution
- They are parasites of all classes of vertebrates, especially marine fishes, and nearly every organ of the vertebrate body can be parasitized by some kind of trematode, as adult or juvenile

GENERAL CHARACTERISTICS

- Flat, unsegmented worms
- Leaf-like organisms
- Incomplete digestive tract
- Oral sucker (nourishment)
- Ventral sucker or acetabulum (for attachment)
- Most are hermaphrodite, except Schistosoma
- **Oviparous worms** - operculated that requires aquatic environment for embryonation. **except Schistosoma**
- **requires 2 Intermediate host**, **except Schistosoma**
- **Infective stage- Metacercaria. except Schistosoma**

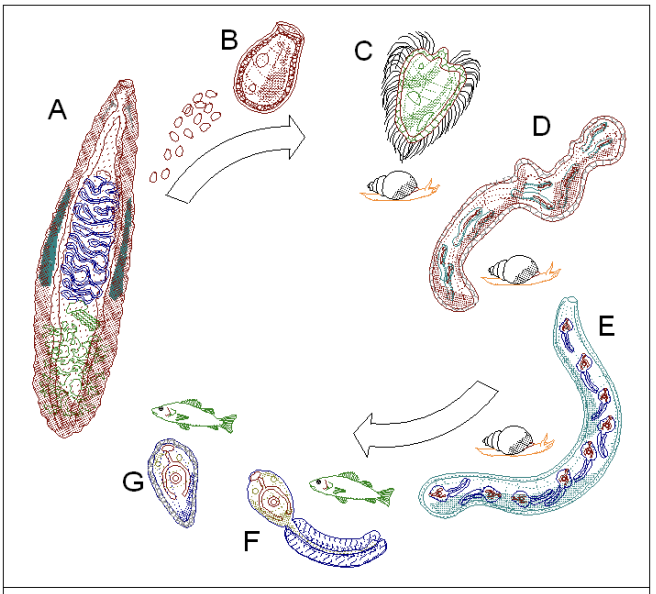
GENERAL MORPHOLOGY OF TREMATODES



LIFE CYCLE

EGG STAGE
LARVAL STAGE • Miracidium • sporocyst • redia • cerceria • metacercaria
ADULT STAGE

DEFINITIVE HOST → Man
1st INTERMEDIATE HOST → Freshwater mollusk
2nd INTERMEDIATE HOST → Fish, crustacean, snail, plants may be required for encystment



A) ADULT
B) EGGS
C) MIRACIDIUM
D) SPOROCYST
E) REDIA
F) CERCARIA
G) METACERCARIA

CLASSIFICATION ACCORDING TO HABITAT IN MAN

HABITAT	TREMATODES INVOLVED
PORTAL BLOODSTREAM OF VERTEBRATES (BLOOD FLUKES)	1. Schistosoma japonicum a. skin penetration of cercaria b. Schistosomiasis japonicum or Oriental schistosomiasis 2. Schistosoma mansoni 3. Schistosoma haematobium
LIVER OF VERTEBRATES (LIVER FLUKES)	1. Fasciola hepatica (Sheep-liver fluke) a. Ingestion of metacercaria on waterplants 2. Clonorchis sinensis (Chinese-Liver Fluke) a. Ingestion of metarcercaria in fish (Clonorchiasis) 3. Opisthorchis felineus (Cat liver fluke) a. Ingestion of metacercaria in fish (Opisthorchiasis)
SMALL INTESTINE OF VERTEBRATES (INTESTINAL FLUKE)	1. Fasciolopsis buski (Giant intestinal fluke) a. Ingestion of metacercaria on waterplants 2. Echinostoma ilocanum a. Ingestion of metacercaria from snails 3. Heterophyid Group

	→ Ingestion of metarcercaria from fish (Heterophyidiasis) a. Heterophyes heterophyes b. Metagonimus yokogawai c. Haplorchis yokogawai d. Haplorchis taichu
LUNGS OF VERTEBRATES (LUNG FLUKES)	1. Paragonimus westermani (Oriental lung fluke) a. Ingestion of metarcercaria from crabs (Paragonimiaisis)

CLASSIFICATION OF OVA

MATURE	IMMATURE
CLONORCHIS **H**ETEROPHYIDS **O**PITHORCHIS **S**CHISTOSOMA	**F**ASCIOLA **F**ASCIOLOPSIS **E**CHINOSTOMA **P**ARAGONIMUS

Schistosoma spp.

- diecious (separate sexes) with two sexes that are **dissimilar** in appearance
- Female worms LEAVE TEMPORARILY the males to deposit the eggs in small venules close to the lumen of the intestine or bladder

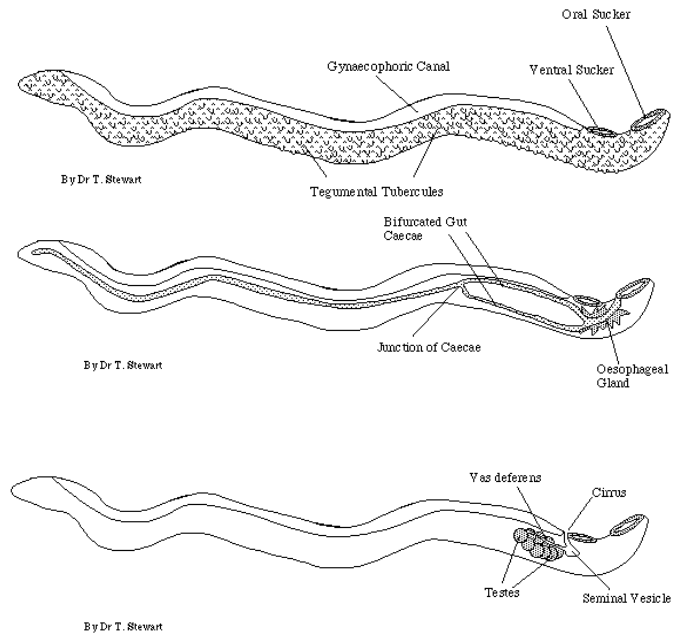
japonicum	***mansoni***	***haematobium***	***intercalatum***	***mekongi***
"oriental blood fluke"	"Intestinal blood fluke"	"Urinary blood fluke"	"African blood fluke"	"Mekong river blood fluke"
China, Taiwan, Japan, Philippines, Indonesia	Africa, Arabian Peninsula and Malagasy, Brazil, Surinam, Venezuela, Puerto Rico, and parts of Carribean	African continent Endemic in the Nile River banks and delta, Jordan Asia minor, Cyprus, Southern Portugal	Western and Central Africa	Mekong river basin in southern Laos and Cambodia
Found in **all mammals** exposed to infected water	Spread in the Western Hemisphere due to African slave trade		resembles **haematobium**	resembles **japonicum**

Morphology of Schistosoma spp.

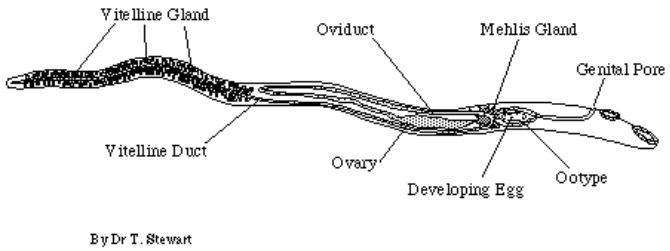
spp.	MALE	FEMALE	OVUM
japonicum	• elongate, cylindrical in shape, measuring **12-20mm x 0.5- 0.55 mm** in diameter • integument is nontuberculate • gynecophoric canal is present • oral and ventral suckers lie close to each other • alimentary system consist of the mouth sorrounded by oral suckers, esophagus, intestine that bifurcates in front of the ventral suckers and joins again in the last	• slender, delicate, and smooth measuring 26 mm x 0.3 mm • ovary is in the middle of the body, with genital pore behind the ventral suckers • Uterus- **50-100 eggs at a time** • Lodge in the gynecophoral canal of the male worm • Location- **Mesenteric veins**	• ovoidal to round in shape measuring 70-100 um × 50- 65 um • thin shelled and frequently has a rough beaded outer surface due to cellular debris attach to it • Lateral, short, sometimes curved spinose knoblike process • embryonated when laid • **LOCATION**- **Veins of Intestine and rectum** • exit from the host=**FECES**
mansoni	• **6-14 mm × 1.1 mm** • Integument- grossly tuberculate • Intestine long • Testes- 8-9 and in zigzag arrangement.	• slender, delicate, and smooth measuring 14 mm x 0.25 mm • ovary is in anterior half of the body • Uterus- **1-3 eggs at a time**	• Elongate, oval measuring 155um x 60 um • yellowish brown, transparent shell • Lateral long spine • embryonated when laid

		• Lodge in the gynecophoral canal of the male worm • Location- **Mesenteric veins**	• LOCATION- **Veins of colon, rectum, mesenteric lymph glands, occasionally bladder** • exit form the host= **FECES rarely URINE**
haematobium	• 19-15 mm x 0.8-1 mm in diameter • • Integument- Fine tuberculate • Intestine long • Testes- groups of 4-5 in number, just behind the ventral suckers	• slender, delicate, and smooth measuring 20 mm x 0.25 mm • ovary is in posterior half of the body • Uterus- **20-30 eggs at a time** • Lodge in the gynecophoral canal of the male worm • Location-**Pelvic veins**	• Elongate, oval/ spindle (rounded ant., Conical pos.) measuring 112-170 um × 50 um • light yellowish brown transparent shell • Terminal, delicate spine • Partly mature when laid with miracidium • LOCATION- **Veins of bladder. occassionaly rectum, pelvic, lymph glands** • exit from host= **URINE, rarely FECES**

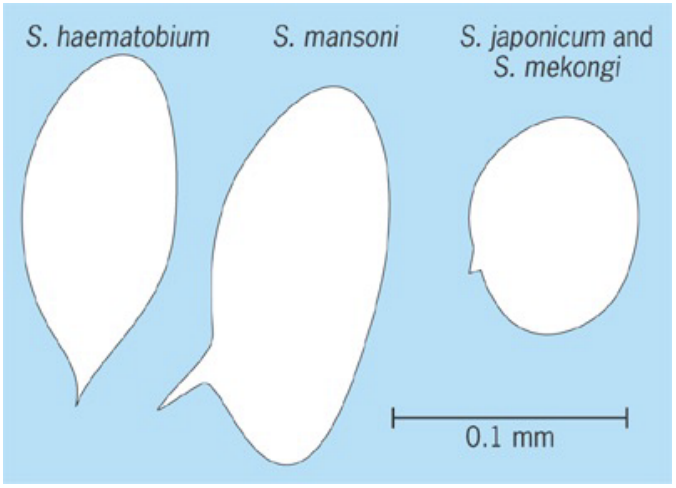
MALE WORM

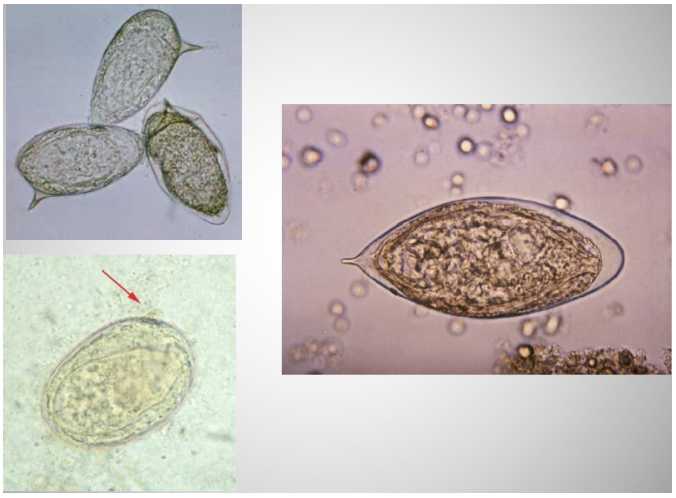


FEMALE WORM



Ova of schistosoma spp.

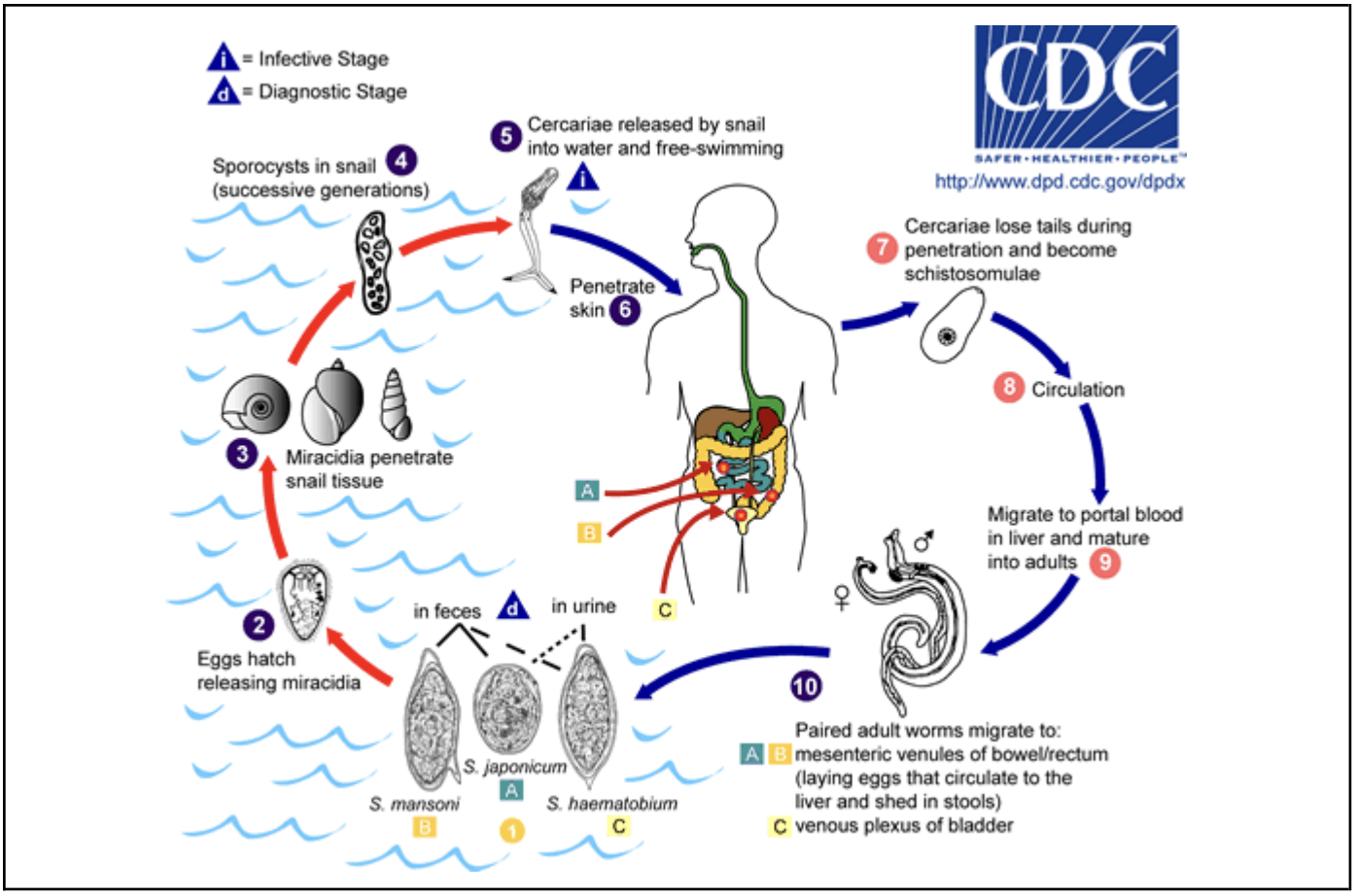




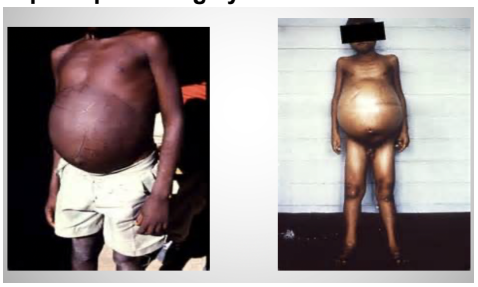
Infective stage – CERCARIA (SKIN PENETRATION)

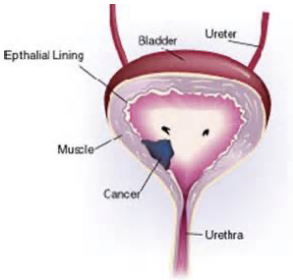
- Elongate pear shape body with rounded end and a long tail that is bifid
- 175-240um × 55-100um in diameter
- body and tail is covered with cuticle and minute spines.
- released into the water 25-30 days after the snail has been infected

Parasite	***Definitive Host***	***Intermediate host***	
Schistosoma haematobium	Man, monkey	Bulinus spp.	
Schistosoma mansoni	Man, monkey	Biomphalaria spp.	
Schistosoma japonicum	Man, dog, cat, cattle, horses & hogs	Oncomelania spp.	



Pathology of Schistosoma spp.

DISEASE	• Schistosoma (bilharziasis)
MODE OF TRANSMISSION	• Penetration of skin by larva (Cercaria) in water
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• SKIN PENETRATION → dermatitis, erythema, pruritus, papules and become intense win 48- 72 hrs. exposure to contaminated waters) ○ malaise, fever, hives, abdominal pain ○ **Katayama fever**- S. japonicum ○ **Hepatosplenomegaly**  ○ **Intestinal**- confined to LI. Leading to bloody diarrhea with intestinal polyposis- marked weight loss and anemia. S. japonicum & manson ○ **Pulmonary involvement-** common in all forms but most common in S.haemotobium. ○ **Cough**-hemoptysis, exertional dyspnea with enlargement of the heart ○ **Urinary involvement**- urinary frequency, dysuria, hematuria, Bladder cancer 

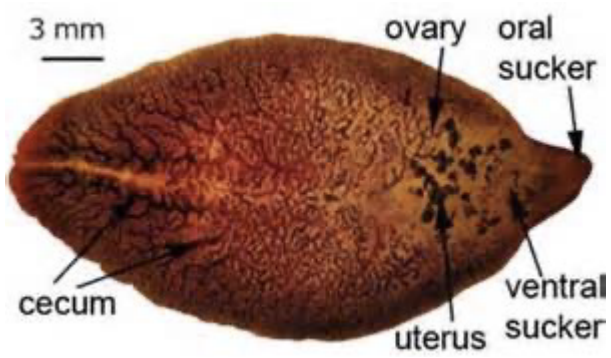
	 Schistosoma haematobium – formation of bladder cancer ○ CNS-common in all forms of Schistosoma. ■ Brain- S. japonicum. ■ Spinal cord- transverse myelitis in the lumbar area- flaccid paralysis of the lower limbs. S. mansoni & haematobium
DIAGNOSTIC TEST	• DFS- recovery of ova in stool • Uinalysis- S. haematobium • sputum exam- S. haematobium • CSF exam
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Oxamniquine • Proper disposal of urine & feces • wearing of protective gears in potentially snail-infested waters • eradication of I.Host

Fasciola hepatica

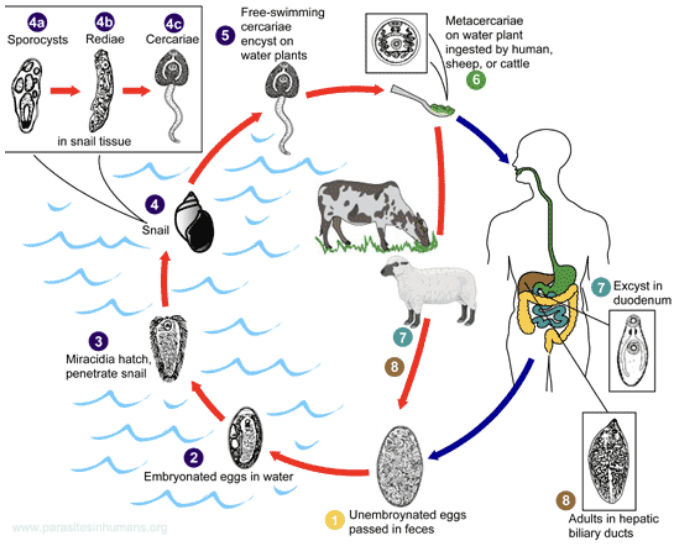
• "sheep liver fluke" - "liver rot" • Common parasite of herbivores and cosmopolitan in distribution • Human infection occurs in sheep-raising countries- South America, Bolivia, Uraguay, Argentina, Peru, Cuba, Southern France, Algeria	• **Definitive Host:** Man, sheep, cattle • **1st Intermediate Host:** snails • **2nd Intermediate Host:** Aquatic/water plants (e.g. Watercess) • **Mode of Transmission:** Ingestion of infected aquatic plants • Worms can live as long as **11 years**
1st Intermediate Host – Lymnaea truncatula 	2nd Intermediate host – Watercress • Watercress (Nasturtium officinale), is a fast-growing, aquatic or semi- aquatic, perennial plant native to Europe and Asia, and one of the oldest known leaf vegetables consumed by humans. It is a member of the family Brassicaceae, botanically related to garden cress, mustard and radish - all noteworthy for a peppery, tangy flavour.  • The hollow stems of watercress are floating, and the leaves are pinnately compound. Small, white and green flowers are produced in clusters.

Morphology of Fasciola hepatica

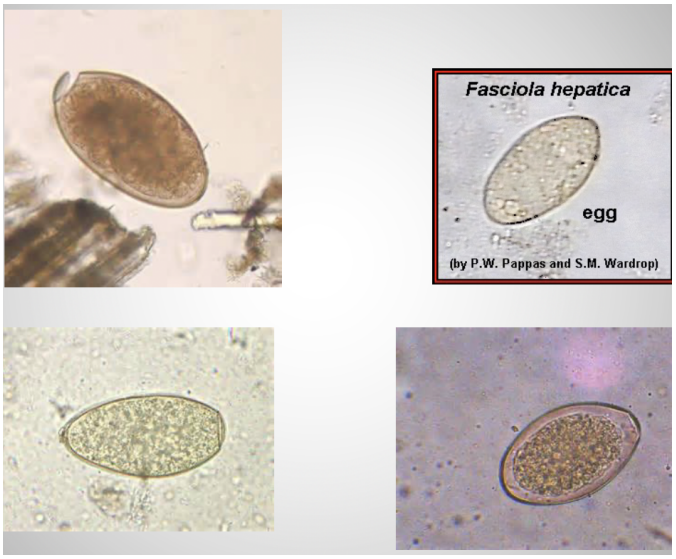
ADULT WORM	OVA



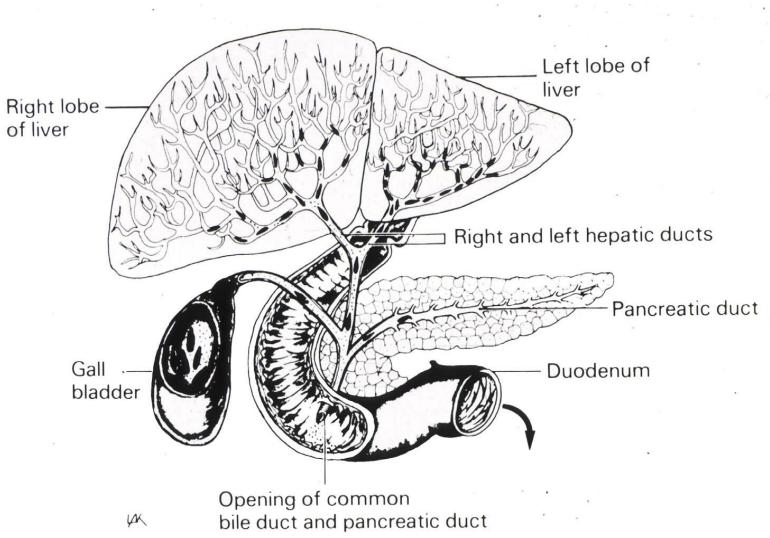
- Large, Flat, brownish, leaf- shaped flukes
- 3cm x 1.5 cm
- has 2 suckers
- Anterior end with cephalic cone (shouldered appearance)
- Integument is covered with scales
- Alimentary system has an inverted "y "appearance
- hermaphrodites



- Large, operculated, ovoid in shape
- 140-150um × 65-90um
- Light yellow in color
- Transparent shell w/ thin double walls
- Contains large mass of lecithin granules when freshly voided



Anatomic Structure of the Billiary system



Pathology of Fasciola hepatica

DISEASE	• Fascioliasis  
MODE OF TRANSMISSION	• Ingestion of encysted larvae (metacercaria) on aquatic plants • Ingestion of raw animal liver infected with adult worm
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• fever, chills • **cholangitis**- bile ducts • Rt. Upper quadrant pain that radiates thru the scapula • Hepatomegaly • eosinophilia • jaundice • portal cirrhosis • **HALZOUN –** pharyngeal dse. Pain, bleeding, edema that sometimes interfere w/ respiration
DIAGNOSTIC TEST	• DFS- recovery of ova in stool large operculate eggs) • serologic test
TREATMENT/ PREVENTION/ CARE	• Bithionol • Triclabendazole- veterinary use • wash and cooked aquatic plants properly • proper disposal of animal human feces • avoid eating improperly cooked liver • eradication of the 1st I.Host

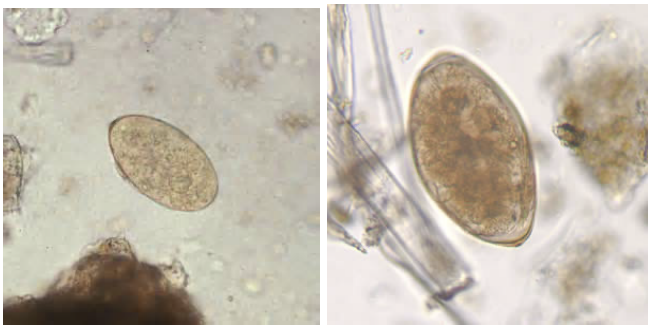
Fasciola gigantica

- "Giant liver fluke"
- Longer and more slender but otherwise very similar to F. hepatica.
- May attain a length of 7.4 cm.
- A parasite of herbivores particularly camels, cattle and water buffalo.
- Found in Africa, Asia, Hawaii
- Morphology, biology, and Pathology is similar to F. hepatica

Adult Worm

a. Fasciola gigantica
b. Fasciola hepatica

- Ova is large , measuring 150-190 um x 70-90 um

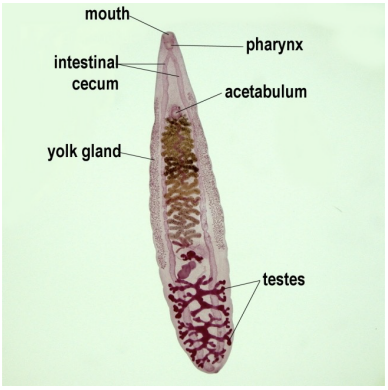


Clonorchis sinensis

- "Chinese liver fluke"
- Was first discovered in the bile passages of a Chinese carpenter in Calcutta in 1875.
- Widely distributed in Japan, Korea, China, Taiwan, Vietnam.
- Adult worms live and localize in the bile ducts and occupy the bile passages in massive infections
- they can also invade the gallbladder and pancreatic duct
- Worms can live as long as 30 years. Can lay 2400 eggs/day/worm

- **Definitive Host:** Man, dog, cats, hogs - R
- **1st Intermediate Host:** Snails – Paraforssarulus manchouricus
- **2nd Intermediate Host:** Freshwater fish – Ctenopharyngodon idellus
- **Mode of Transmission:** Infection is acquired from consumption of freshwater fish containing he encysted larva
    - Fish may be eaten raw, pickled, smoked, or dried
    - Disease have been reported in Hawaii as a result of consumption of infected fish imported from the Orient.

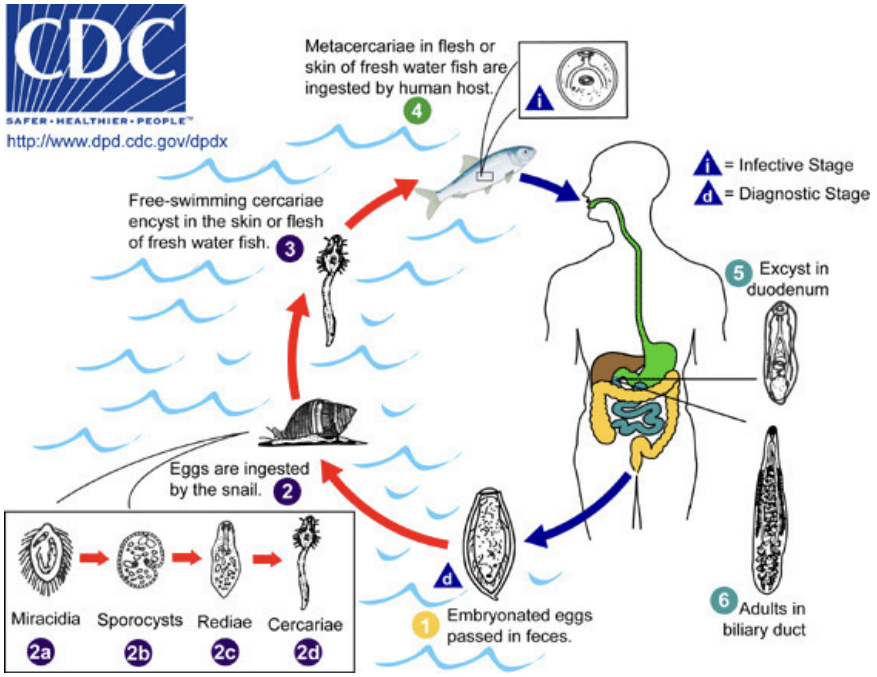
ADULT WORM



- Narrow, oblong, flat, tapering anteriorly and rounded posteriorly- leaf-like
- 10-25mm × 2-3mm
- Oral sucker is larger than the ventral sucker
- large testes which are branched in tandem near the posterior end

OVUM

- Flask shape, yellowish brown, operculated
- 35um × 20 um
- There is a small knob or curved spine on the terminal end.
- fully embryonated when laid



Pathology of Clonorchis sinensis

DISEASE	• Clonorchiasis
MODE OF TRANSMISSION	• Ingestion of encysted larvae (cercaria) in freshwater fish
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• fever • abdominal pain • diarrhea • cholangitis- bile ducts • cholecystitis- gall bladder cholelithiasis • Hepatomegaly • eosinophilia • jaundice • carcinoma of biliary tract- cholangiocarcinoma
DIAGNOSTIC TEST	• DFS- recovery of ova in stool (small, flask shape, operculated eggs) • Duodenal aspirate • Entero test • serologic test/ compliment fixation test • intradermal test
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Albendazole • cooked fresh- water fish properly • proper disposal of animal human feces • eradication of the 1st I.Host

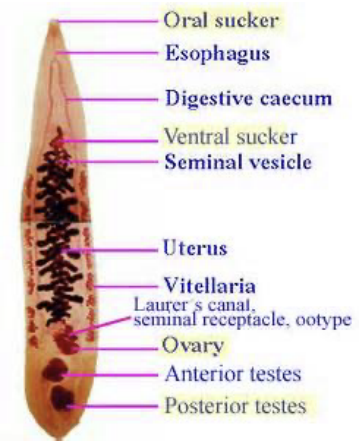
Opisthorchis viverrini

- Family Opisthorchiidae-Clonorchis & Opisthorchis spp.
- "Southeast Asian liver fluke" or "Fish-liver fluke"
- a major health problem in Northern Thailand and Laos and about 7 million are infected in Thailand
- prevalence is 80-90% among rural people, and 55% in urban dwellers increasing with age up to 10 years old

- Pathology and epidemiology is similar to C. sinensis and O. felineus
- **Mode of Transmission:** consumption of uncooked fresh-water fish
- **Definitive Host:** Man, (cats, dogs)-R
- **1st Intermediate Host:** snail
- **2nd Intermediate Host:** fresh-water fish
- Life cycle similar to C.sinensis
- infection is highly correlated with incidence of cholangiocarcinoma-Opisthorchiasis

ADULT WORM

- Flat , elongated, tapering anteriorly and rounded posteriorly
- 5-10 mm x1mm
- Testes are lobate located in the posterior part of the body



OVUM

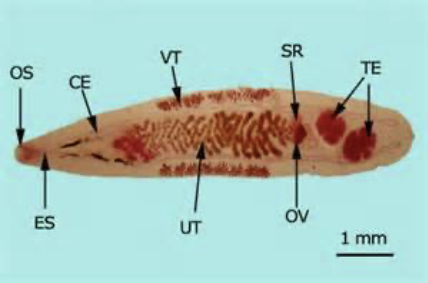
- Relatively short and broad, operculated
- 28 um × 16 um
- may not posses terminal tubercle-like knob
- Fully embryonated when laid

Opisthorchis felineus

- "cat liver fluke"
- prevalent in the liver of both cats and dogs
- 1884 in the liver of cats by Sebastiano Rivolta of Italy
- 1891 a Russian scientist K.N. Vinogradov found it in humans.
- Occurs in Europe, Siberia, Asia- Philippines, Korea, Japan North Vietnam and India
- 1.5 million infection in Russia

- **Mode of Transmission:** consumption of uncooked or raw, slightly salted, frozen fish
- **Definitive host:** cats, dogs, Man
- **1st Intermediate host:** snails
- **2nd Intermediate host:** Freshwater fish
- Life cycle similar to C. sinensis
- Infection can lead to Liver cancer(cirrhosis) but, maybe asymptomatic in children, severe anemia.

ADULT WORM



- Almost the same with the other opisthorchiids
- More similar to C. sinensis

OVUM

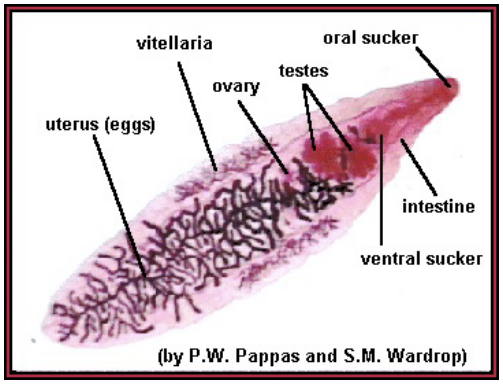
- Ova is narrower, operculated
- 30um × 11-12um
- Otherwise indistinguishable from C. sinensis

Dicroceolium dendriticum

- "lancet liver fluke" bladelike shape
- A common parasite of the biliary tree of herbivores (sheep, cattle, goats, pigs, etc.)
- It is common throughout most of Europe and Asia. Discovered in 1819 by Rudolphi
- They are morphologically similar to C.sinensis with slight differences.
- Human infection is uncommon
- "brainworms"- they alter ant's behaviour increasing it's probability of ingestion by the D.H

- **Mode of Transmission:** Ingestion of ants (Drabick et al. 1988, case report)
- **Definitive host:** sheep, cattle, hogs, goats, Man (accidental host) etc.
- **1st Intermediate Host:** snail
- **2nd Intermediate Host:** ants
  - Anthropod – common brown ant, Formica fusca
- Numerous human infections were false infxn. Eggs detected in stool are actually part of the liver eaten awhile ago.

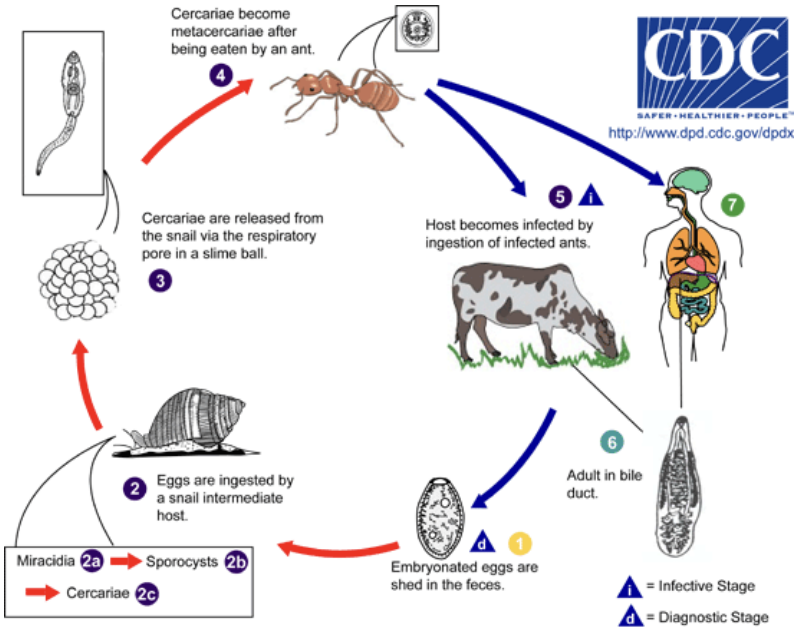
ADULT WORM



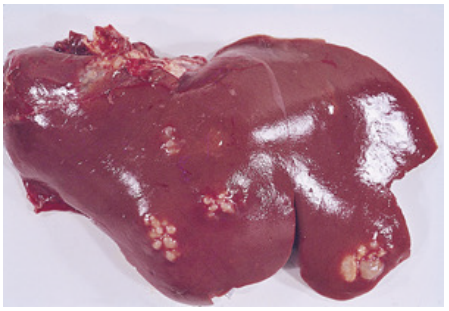
- Bladelike shape
- 6-10mm × 1.5-2.5mm
- Both ends are pointed
- Lobate testes that lie in the anterior third of the body

OVUM

- Eggs passed in feces are dark brown in color
- Thick shelled and with a large operculum
- 38-45 um × 22-30 um



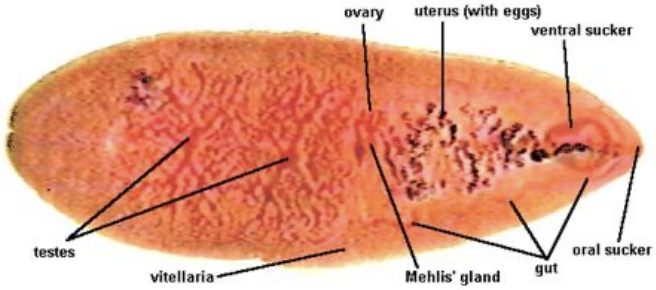
Pathology of Dicrocelium dendriticum

DISEASE	• Dicroceoliasis
MODE OF TRANSMISSION	• Ingestion of larvae encysted (metacercaria) in ants.
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • biliary dysfunction with digestive disturbances • enlargement of bile ducts-cholangitis • fibrosis • cirrhosis 
DIAGNOSTIC TEST	• DFS- demonstration of ova in stool
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Inspect food before consuming • proper disposal of animal / human feces • eradication of the 1st I.Host

Fasciolopsis buski

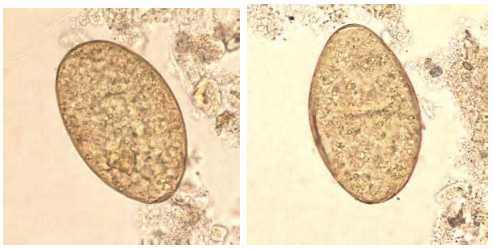
• Family Fasciolidae • "Giant intestinal fluke" • Found in China, Taiwan, Vietnam, Thailand, Indonesia, Malaysia, India. • It was first described by George Busk, in London in 1843 • Adults can lay 25,000 eggs/day • In heavy infection they are found throughout the intestinal tract attached to intestinal wall (SI)	• **Mode of Transmission:** Ingestion of raw or peeled aquatic plants or vegetation containing the metacercariae • **Definitive host:** Man (pigs, dogs, rabbits = R) • **1st Intermediate Host:** snail - Segmentina and Hppeutis • **2nd Intermediate Host:** water chestnuts, water caltrops, lotus, bamboo shots, and other edible plants • Life cycle similar to F. hepatica • Life span of worm is 6 months and an estimated 10 million infection occur annually

ADULT WORM

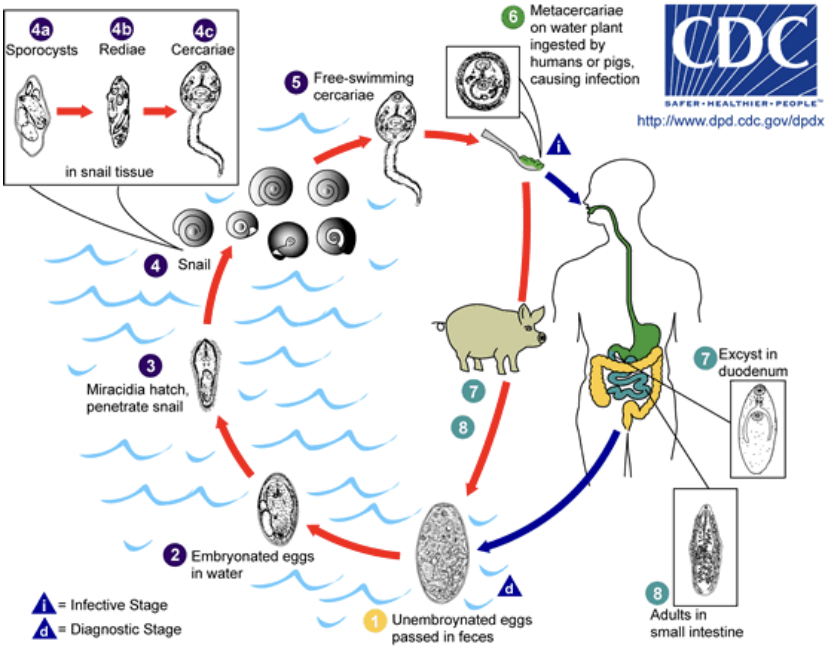


- Adult worms are seen after purgation
- Fleshy worms, elongated and oval, ant. End narrower than post. End. Acetabulum is larger than the oral suckers
- 2-7.5 cm x 0.8-2 cm
- Resembles F. hepatica but does not possess cephalic cone

OVUM



- Yellowish brown, ellipsoidal
- 130-140um × 80-85um
- Has a clear thick shell w/ a small operculum
- Similar to F. hepatica
- Unembryonated when laid-(7wks to mature and hatch at 27°C - 32°C.)



Pathology of Fasciolopsis buski

DISEASE	• Fasciolopsiasis
MODE OF TRANSMISSION	• Ingestion of larvae encysted (metacercaria) in aquatic plants
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Abdominal pain- ulcers w/ hemorrhages • diarrhea • edema &ascites- worm toxins • Intestinal obstruction • eosinophilia • stool become profuse, light w/ much undigested food -malabsorptive process
DIAGNOSTIC TEST	• DFS- demonstration of ova in stool

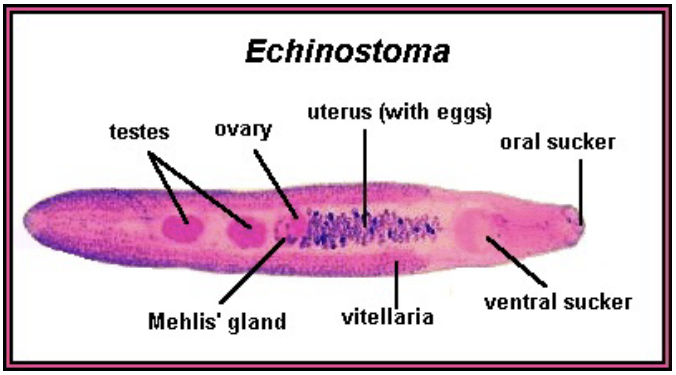
TREATMENT/ PREVENTION/ CARE	• Praziquantel • cooked aquatic plants before consuming • proper disposal of animal / human feces • eradication of the 1st I.Host

Echinostoma ilocanum

- A common medium-size intestinal fluke that occurs in the Philippines
- First seen in the stool of a prisoner in Manila in 1907
- Life cycle involves 2 snail intermediate host

- **Mode of Transmission:** Ingestion of raw snails
- **Definitive Host:** Man, aquatic birds

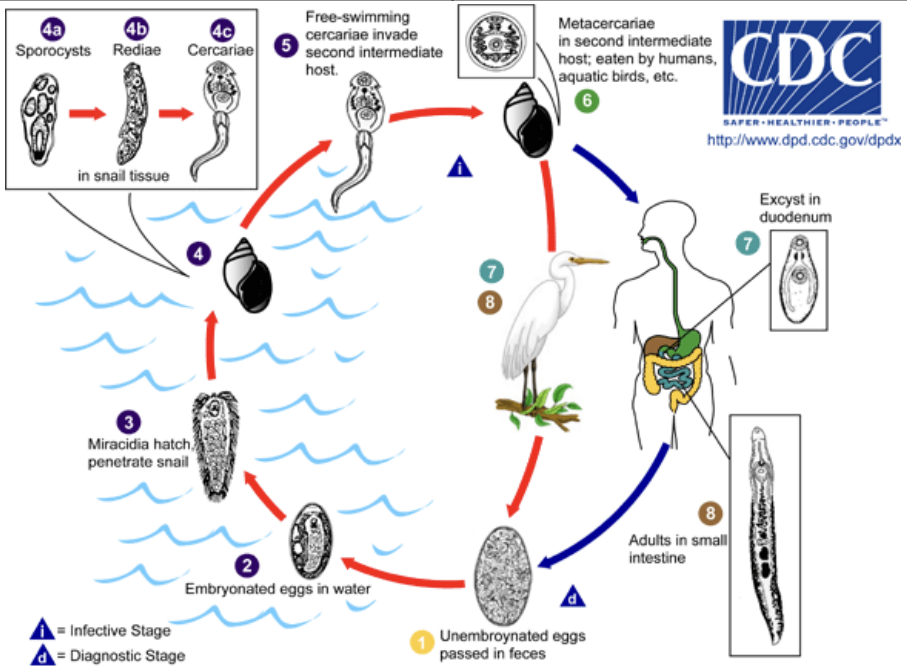
ADULT WORM



- Adult worms are seen after purgation
- Circumoral spine arrange like a crown numbering 49-51
- 25-0.65cm x 0.1-0.13
- Integument is covered with plaque-like scales
- testes are deeply lobate

OVUM

- Ovoid in shape, straw colored, operculated
- 83-116um × 58-69um
- Unembryonated when laid
- Similar to Fasciolopsis

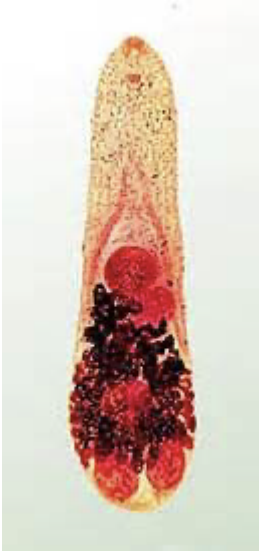
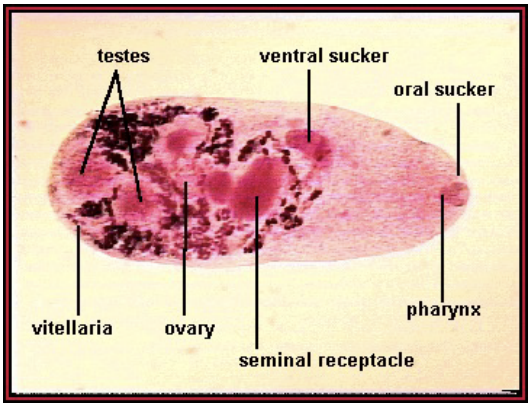


Pathology of Echinostoma ilocanum

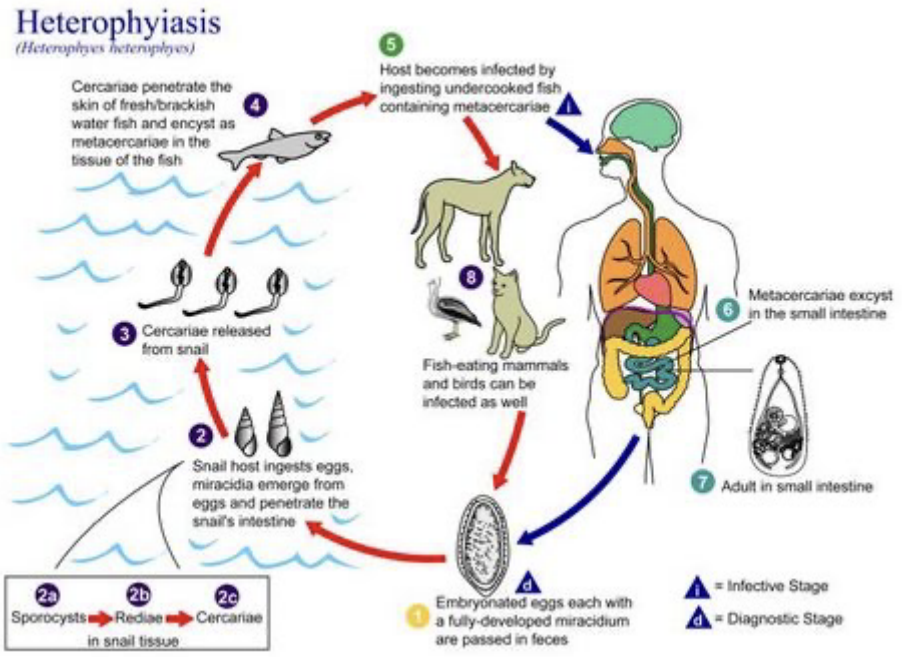
DISEASE	• Echinostomiasis
MODE OF TRANSMISSION	• Ingestion of larvae encysted (metacercaria) in snail
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • Abdominal pain- ulcers w/ inflammation • diarrhea
DIAGNOSTIC TEST	• DFS- demonstration of ova in stoo
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Tetrachloroethylene • avoid consuming raw snails • proper disposal of animal human feces • eradication of the 1st I. Host

The Heterophyids - minute flukes

- Tiny, teardrop-shaped flukes, usually maturing in the small intestine of fish-eating birds and mammals
- Heterophyes heterophyes
- Metagonimus yokogawai
- Occur in Japan, Korea, China, Taiwan, Philippines, and Western India
- Heterophyes- Egypt (Cairo 1851), and Israel
- Metagonimus- Balkans, Spain, Israel, Russia, Indonesia
- **Mode of Transmission:** ingestion of raw or pickled fresh-water fish
- **Definitive Host:** Mammals, fish-eating birds
- 1st Intermediate Host: Snail
    - Heterophyes heterophyes → Pironella conica (Egypt), Cerithidia cingula (Japan) [left]
    - Metagonimus yokogawai → Semisulcospira spp. [right]

	Heterophyes heterophyes	**Metagonimus yokogawai**
ADULT WORM seen after purgation	• Elongate, pyriform, grayish fluke • 1-1.7 mm x 0.3-0.4 mm • The entire body is covered w/ slender scales • 2 oval testes lie side by side near posterior end • Contains a genital sucker • Ovaries are small 	• Small, pyriform in shape with rounded posterior and tapering anterior end • 1-2.5 mm x 0.4-0.75 mm • Ventral suckers is deflected to the right of the midline • 2 ova testes are ocated at the posterior third of the body • Ovary is found at the junction of the middle and lower third of the body 

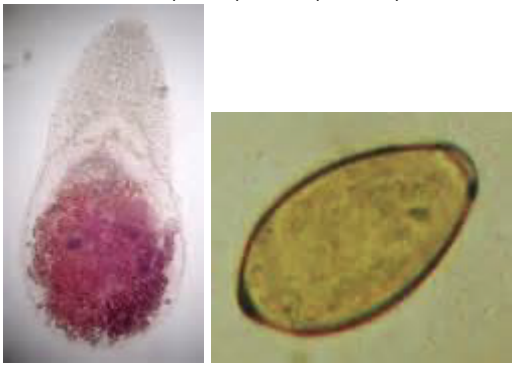
OVUM closely resembles ova of Clonorchis and Opisthorchis	• Minute, operculate, ovoidal, light brown in color • 28-30um × 15-17um • The shell has a slight shoulder at the rim of the operculum and sometimes a knob at the posterior end • Fully embryonated when laid  	• Minute, ovoidal, operculate • Slight shoulder at the rim • 26-28um × 15-17um • Fully embryonated when laid 



Pathology of Heterophyids

DISEASE	• Heterophyiasis
MODE OF TRANSMISSION	• ingestion of larvae encysted (cercaria) in fresh-water fish
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• Asymptomatic • abdominal pain • diarrhea • nausea • Occasionally worms invade the mucosa and deposits eggs in tissue- gain access to circulation and invades the brain, spinal cord- seizures neurologic deficits • heart- cardiac insufficiency
DIAGNOSTIC TEST	• DFS- demonstration of ova in stool
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Tetrachloroethylene • cooked fresh water- fish properly • proper disposal of animal human feces • eradication of the 1st I.Host

Haplorchis taichui

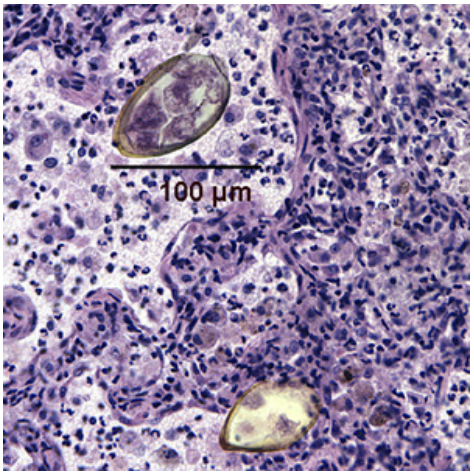
• Heterophyid trematode- Intestinal fluke • reported in Mindanao Island, Southern Philippines (Belizario et al., 2004) • Cases have been reported in Thailand and Philippines • **Mode of Transmission**- ingestion of raw or pickled fresh-water fish • **Definitive Host:** Man, dogs, cats, and hogs • Life cycle similar to Heterophyes	ADULT WORM (LEFT), OVA (RIGHT) 

Pathology of Haplorchis taichui

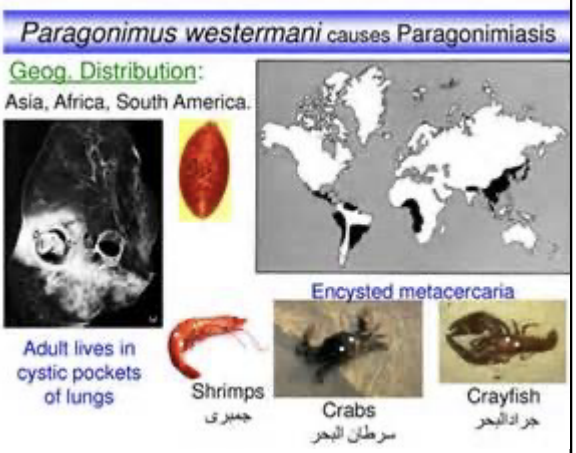
DISEASE	• Haplorchiasis
MODE OF TRANSMISSION	• Ingestion of larvae encysted (cercaria) in fresh-water fish
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• abdominal pain • diarrhea • nausea • borborygmi
DIAGNOSTIC TEST	• DFS- demonstration of ova in stool
TREATMENT/ PREVENTION/ CARE	• Praziquantel • cooked fresh water fish properly • proper disposal of animal-human feces • eradication of the 1st I. Host

Paragonimus westermani

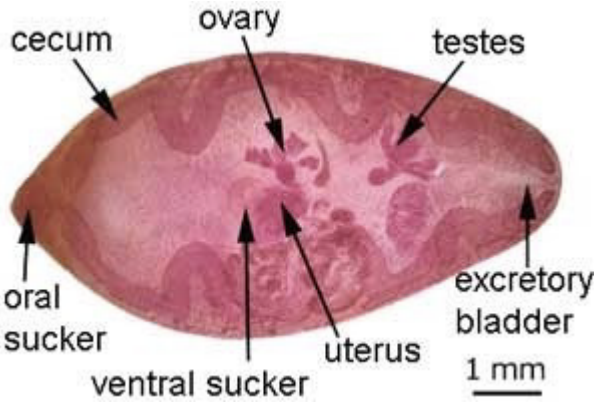
• "Oriental lung fluke" • First described from 2 Bengal tigers that died in the zoos in Europe in 1878. • Most widely prevalent species. It was discovered in humans 2 years after in Formosa Taiwan). It was quickly found in the lungs, brain, and viscera of humans in Japan, Korea, and the Philippines. • There at least 8 species known to infect man under genus Paragonimus. (28 spp. that are worldwide distributed)	• They are primarily a parasite of wild felines- tigers, lions, wolves, leopards, dogs, etc.. • Mammals like wild pigs, badgers, mongoose, racoons, minks and others that eat fresh-water crabs and crayfish • Human are accidental host • **MODE OF TRANSMISSION –** ingestion of raw or insufficiently cooked infected crustacean • **DEFINITIVE HOST –** carnivores, Man • **1ST INTERMEDIATE HOST** – snail • **2ND INTERMEDIATE HOST** – crustacean



Histological examination of a human lung



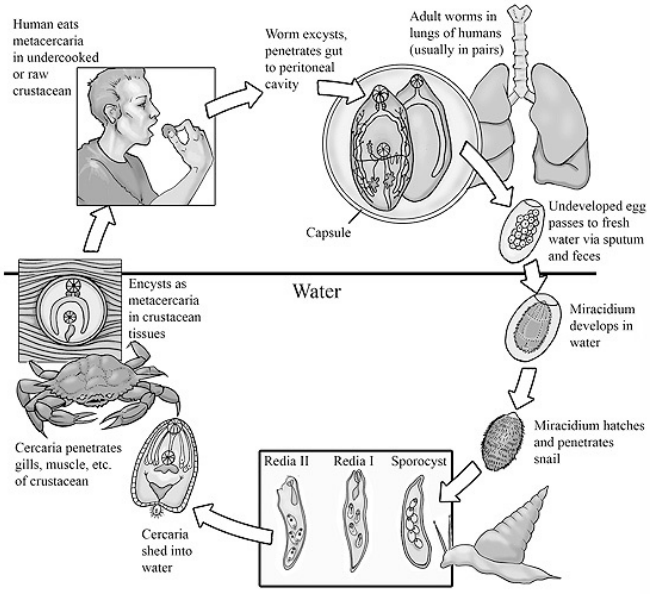
ADULT WORM



- Thick, fleshy and egg-shaped, reddish brown
- 0.8- 1.2 cm x 0.4-0.6 cm
- tegument is densely covered with scale like spines
- Testes is irregularly lobed, placed oblique to each other in the posterior third of the body
- Ovary is lobed located anterior to the testes
- Excretory bladder is large and at the center dividing the body into 2 equal parts

OVUM

- Oval shape, golden brown
- With a flattened operculum and has a thick shell
- 80-120 um × 48-60um
- Unembryonated when laid



Pathology of Paragonimus westermani

DISEASE	• Paragonimiasis
MODE OF TRANSMISSION	• Ingestion of larvae encysted fresh- water crabs or crayfish • Accidental Host
CLINICAL FEATURES (SIGNS AND SYMPTOMS)	• clinical picture resembles that of P. tuberculosis (Chronic) • **productive cough, fever, haemoptysis, eosinophilia** • **Chest pain, dyspnea, chronic bronchitis, pleural effusion** • typically the worm are encapsulated in cystic structures adjacent to the bronchi. (granuloma) • • the eggs are discharge into the bronchi or bronchioles; they may be expectorated or if swallowed , appear in the feces. • Chest film may show patchy infiltrate with nodular cystic shadows or calcification. • worms can enter other parts of the body • **Brain-** cerebral migration- fever, headache, nausea, vomiting, convulsive seizures • **Spinal cord-** motor and sensory disturbances • **Liver-** fibrosis-cirrhosis
DIAGNOSTIC TEST	• DFS- demonstration of ova in stool • Sputum analysis- demonstration of ova in sputum- is frequently blood tinge and may contain numerous dark brown eggs and Charcot-Leyden crystals. • Pleural fluid aspirate • Serologic test- ELISA/ EIA
TREATMENT/ PREVENTION/ CARE	• Praziquantel • Bithionol • cooked fresh-water crabs and crayfish properly • washed hands and utensils properly • eradication of the 1st I.Host

Charcot Leyden crystals- 50um; enzyme-lysophospholipase. (trichrome stain)