Cestodes discussed in this handout:

1. *Taenia solium*—the **pork tapeworm**
2. *Taenia saginata*—the **adult beef tapeworm**
3. *Echinococcus granulosus*—the **minute tapeworm**
4. *Diphyllobothrium latum*—the **broad tapeworm/fish tapeworm**
5. *Dipylidium caninum*—the **double pored dog tapeworm**
6. *Hymenolepis nana*—the **dwarf tapeworm**
7. *Hymenolepis diminuta*—the **rat tapeworm**

**INTRODUCTION**

The cestodes (tapeworms):

- **Phylum:** Platyhelminthes
- **Class:** Cestoda
- The adult tapeworms found in humans all have a **flat** and **ribbon-like body**
  - **Color:** White/Yellowish
  - **Body:**
    - Anteriorly, the attachment organ—**Scolex**
      - The scolex is followed by a series of segments called **Proglottids**
        - **Proglottids** aka strobila
      - The proglottids can be regenerated when the worm is cut and the site where the regeneration starts is the **first proglottid** immediately **posterior** to the scolex
        - Initially, the newly regenerated proglottid segments are considered to be **immature** because their internal structures are not yet developed
        - The **mature proglottid segments** are located in the **middle of the chain** and they are **larger** and each segment may contain:
          - One **male** reproductive system
          - One **female** reproductive system
          - **Both male and female** reproductive systems
        - The **terminal proglottid** of some species may become detached in the intestine and can be seen in the human stool
          - Thus, the detached terminal proglottid can be a key diagnostic modality
• The terminal proglottid (terminal strobila) contains the ripe/gravid segments that is filled with eggs
  o The eggs are enclosed in the uterus
    ▪ The form of the uterus is unique per species and is used to differentiate one species from another

➤ Adult tapeworms inhabit the small intestine where they live attached to the small intestinal mucosa
  o The attachment is possible due to the tapeworms’ scolex
  o ALL human cestodes have 4 muscular, cup-shaped suckers on the scolex EXCEPT in Diphyllobothrium latum and other related species
    ▪ In addition to the suckers, the scolex may have an elongate and protrusible structure—the rostellum
      1 The rostellum is situated in the central portion of the scolex
      2 In some species, the rostellum bears hooks and is referred to as armed rostellum
    ▪ The scolex morphology can be used in making a specific diagnosis

➤ Tapeworms do NOT have a digestive system and their food is absorbed from the host’s intestine

➤ Cestodes that parasitize humans have complex life cycles that generally involve both a definitive and an intermediate host
  o For some species, the human person is the ONLY definitive host—growing to adulthood in the intestine after ingestion of the infective larvae
    ▪ Ex: Diphyllobothrium latum, Taenia saginata, Hymenolepis diminuta
  o For some species, the human person can the definitive host OR the intermediate host
    ▪ Ex: Taenia solium, Hymenolepis nana
  o For some species, the human person is JUST an intermediate host
    ▪ Ex: Echinococcus granulosus

➤ Cestode infection: In general, extraintestinal infection with the larval forms is much more serious than infection with the adult worm.

➤ Unlike the trematodes whose eggs are usually operculate, the operculum is much less in the eggs of the cestodes
  o Cestode eggs vary considerably in the appearance of the external shell as well as in the number and thickness of the embryonic membranes
    ▪ These membranes serve as protective coverings of the embryo, which is called an oncosphere and bears 6 elongate hooks
PARADE OF THE CESTODES

A. *Taenia solium*—the *pork tapeworm*

➔ *T. solium* occurs in places where people eat *cured/undercooked pork*

➔ Prevalent areas: *Mexico, Latin America, Iberian peninsula, Slavic countries, Africa, India, Southeast Asia, and China*

➔ Causative agent for:
  - *Taeniasis* (*Taeniasis solium*)
  - *Cysticercosis*

➔ *Pigs* and *humans* are the usual intermediate hosts of *T. solium* but dogs can also become infected by ingestion of the eggs of this tapeworm
  - It has been reported that the European brown bear (*Ursus arctos*) and the American black bear (*Ursus americanus*) can also become infected
  - Humans are the ONLY known definitive hosts for *T. solium*
    - Kalito noh? Basta humans are the ONLY definitive host + they can be the intermediate host also because they can transmit the infection to the pigs

Morphology

➔ Adult *T. solium* can attain a length of *several meters(!!!)* Usually 2-3 meters but can be as long as 8 meters

➔ The *scolex* is muscular and bears *4 cup-shaped suckers* and a *double crown* of prominent hooks—all of these are used for attachment to the intestinal mucosa
  - The scolex has a conspicuous, rounded *armed rostellum* with double rows of large and small hooks numbering *22-36*

➔ *Cervical region/Neck* is short, about 5-10mm in length and ½ as thick as the scolex

➔ *Proglottid/Segments*
  - *Mature segments/proglottid* are wider than they are long
    - Contains 1 set of male + 1 set of female reproductive organs
    - The shape is nearly square-ish
    - *150-200 follicles* distributed throughout the dorsal plane at the posterior aspect of the proglottid
    - The *uterus*—rises from the *anterior* aspect of the *ootype*
    - The *trilobed ovary*—situated in the *posterior* aspect of the proglottid with an *accessory ovarian lobe*
    - The number of the *testes* are ONLY half as that of the *T. saginata*
    - There are *genital pores* on consecutive segments
  - *Gravid segments/proglottid* are longer than they are wide
    - Contains a *branched uterus* filled with *eggs*
    - A *central uterine stem* extends through the length of the gravid segment
      - From this stem arise *side branches* which project laterally and extend toward the lateral margin of the proglottid
These main branches may have a variable number of smaller secondary branches.

In *T. solium*, the number of main branches on one side of the central stem varies from 7 to 13 in a dendritic/finger-like branching fashion (sabi sa lecture 5-13 daw huhu, ano ba talaga auntie?)

Thus, specific diagnosis of *T. solium* is made by counting the number of uterine side branches, which may be done in living or in stained preparations.

**Egg**

- The **oncosphere** is enclosed in a thick, radially striated coat called the **embryophore** which is usually **dark brown** in color.
  - The **radial striation** is very unique to *Taenia* eggs and must not be confused with eggs of other human helminths.
- The **6-hooked embryo** can be easily seen in living eggs but frequently become shrunken and opaque in preservation process.

**Life Cycle**

![Life Cycle Diagram](image)

Figure 1. Life cycle of *Taenia solium* and how the cycles end to produce a cysticercosis or taeniasis solium.
1—Eggs are eaten by human, which develops to become a **cysticercus** in the
**muscle** or **brain**
   - A cysticercus—thin-walled bladder within which a single scolex develops
2—The eggs are passed out in feces
3—The eggs become embryonated and are ingested by a **pig**
4—The eggs in the pig develop into **infective larval stage** or **cystecerci** in the
**muscles**
   - The larvae are 0.5 cm or more in diameter
5—Humans eat undercooked pork containing the larvae and become infected
   - When humans eat undercooked cysticercus-infected meat, all but the scolex
   portion is digested, and the remnant scolex is able to attach to the small
   intestinal wall to begin to regenerate a chain of proglottids
6—Ulit sa #2

**Diagnosis and Diagnostic Limits**
- Demonstration of eggs in stool
  - DFS
  - Scotch tape swab (Parang dun sa **Enterobius**)
    - Limit: While gravid segments and eggs may be found in stool
      specimens, specific identification cannot be made on the basis of the
      eggs alone but only on examination of gravid proglottids because eggs
      and proglottids of **T. solium** and **T. saginata** are very much alike
- Demonstration of gravid proglottids in the stool
- Recovery of scolex after antihelminthc therapy

**Specific Differentiation**
- **T. solium** vs **T. saginata**
  - Scolex differentiation
    - **T. solium**—has **armed rostellum** with **spines** or **hooklets**
    - **T. saginata**—**NO** well-defined rostellum **nor** spines
  - Segmental differentiation
    - Segments:
      1. **T. solium**—800-1,000 segments
      2. **T. saginata**—1,000-2,000 segments
    - Lateral branches
      1. **T. solium**—has **13 branches** OR LESS
      2. **T. saginata**—has **MORE THAN 15 branches**

**Infectious Process**
- Although the adult tapeworm develops in humans after the ingestion of **infected
  meat**, infection with the **larval stage** or the **cysticercus** occurs after ingestion of
  **eggs**, either from **exogenous sources** or from their own stools. Although not
  proved, it is thought that **reverse peristalsis** may carry intestinal contents with
eggs to the upper portions of the duodenum, where after hatching, the oncocytes penetrate directly into the intestinal wall.
  o Thus, for these reasons, infection with adults of *T. solium* is dangerous both to the patients and to those whom they come in contact.

**Symptoms**

- The adult worms probably cause no symptoms in the majority of the patients. However, the following have been reported:
  - Vague abdominal discomfort—due to the heavy cysticercosis resulting to the regurgitation of gravid segments in the stomach of patients suffering from *taeniasis solium*
  - Hunger pangs
  - Chronic indigestion
  - Moderate eosinophilia
  - Mild transitory intestinal obstruction

**Treatment**

- **Praziquantel**
  - 5-10 mg/kg BW is the drug of choice with cure rates approximating 100%
- **Niclosamide** can also be used
  - Also used in *diphyllobothriasis*

**Taenial Cysticercosis**

- The common larval stage of tapeworms of the genus *Taenia* is known as a cysticercus—or bladder worm
  - When these stages of the worm were first discovered, their relationship to the adult form was not known. Thus, they were taxonomically classified separately with a separate name as *Cysticercus cellulosae*.
    - But today, *Cysticercus cellulosae* = cysticercus/larval stage of *Taenia*
    - And today, the taxonomic name *Cysticercus cellulosae* is not a valid taxonomic name anymore
  - The eggs may remain viable for many weeks in the soil after they are passed out in the feces.
    - Upon the ingestion of these eggs by humans or hogs, the outer shell disintegrates in the small intestines, and the embryo contained in the egg (oncosphere) is able to invade the intestinal wall and enter a blood vessel by means of the 6 hooklets that it has.
      - Now in the bloodstream, it can be carried to any part of the body and may lodge in any tissue, but MOST frequently it develops in the striated muscle.
      - The cysticercus larva develops in about 2 months
        1. It is transparent
2. It is **Opalescent white**
3. It is **Elongately oval**
4. Length: **0.6-1.8 cm** (pero sa lecture 5-10mm in length, ano ba talaga auntie?)
5. It is **fluid-filled** and on one side is a **denser area** containing the **scolex** that is invaginated with **hooks + suckers**

**Humans that harbor the bladder worm**

1. **Hetero-infection**
   - Eggs liberated from disintegrating gravid proglottids passed by one individual get into the mouth of another and are swallowed

2. **External autoinfection**
   - Eggs may be transferred from anus to mouth or unclean fingertips of an individual who has an intestinal infection with *T. solium*

3. **Internal autoinfection**
   - Gravid proglottids in an individual harboring the adult *T. solium* may become detached from the main strobili or regurgitated into the stomach and then return to the duodenal canal where they disintegrate and liberate ripened eggs

**Symptoms and Pathogenesis**

- In humans, cysticerci most commonly come to our attention when they occur in the **CNS**
  - In swine, cysticercal infections are often localized in the muscle. But in humans, skeletal muscle cysticercosis can also occur.

- Light infections with the bladder worm? → **asymptomatic**
- Heavy infections cause systemic effects
  - It can lodge in the **brain (neurocysticercosis, NCC)**
    - There cases where patients with neurocysticercosis are asymptomatic
  - It can lodge in the **spinal cord**
  - It can lodge in the **heart**
  - It can lodge in the **liver**
  - It can lodge in the **eyes**
  - It can lodge in the **muscles**
    - Cysticerci can develop in any skeletal muscle, though most cases are asymptomatic, the following can be seen in **skeletal muscle cysticercosis**:
      1. **Myositis**
      2. **Fever**
      3. **Eosinophilia**
      4. Rarely, **muscular pseudohypertrophy**
        a. Muscular pseudohypertrophy—initially, there is muscle **swelling** then it leads to **atrophy** and then **fibrosis**
• In skeletal muscle cysticercosis, the worms can die and become **calcific** without giving rise to any symptoms

→ The unsuspected presence of the bladder worms in the muscles may have a diagnostic importance, and soft tissue films of the thigh are often useful in the workup of a patient with unexplained seizures

→ Subcutaneous cysts are easily palpated and may resemble **small lipomas**
  o When these subcutaneous cysts are encountered, surgical removal of the cysts are performed together with the accomplishment of the diagnosis of the cysticercosis

1. **General Symptoms** caused by infection of the bladder worm:
  → Cellular reactions
  → Blood cell infiltration
  → Fibrosis
  → Necrosis

2. **For Neurocysticercosis (NCC)**
  → The following symptoms can be noted:
    o Epilepsy/Seizures
    o Behavioral changes
    o Intermittent obstructive hydrocephalus
    o Dysequilibrium
    o Meningoencephalitis
    o Failing vision

  → Worldwide, NCC is the **most common** parasitic disease of the **CNS**
    o The NCC can present itself in many forms, depending on the localization of the cysts and disease activity

  → **Classification:**
    o **Inactive NCC**
      ▪ Dead worms in the brain
      ▪ Classified as inactive NCC are **calcific(!) parenchymal cysts** and **hydrocephalus** that is secondary to **meningeal fibrosis**
    o **Active NCC**
      ▪ Living cysts in the brain
      ▪ Characterized by:
        1. **Arachnoiditis**
        2. **Spinal fluid pleocytosis**
        3. **Increased CSF protein**
        4. + **CSF serologic test for cysticercosis**
      ▪ There can also be:
        1. **Meningitis**
        2. **Obstructive hydrocephalus**
        3. **Cranial nerve involvement**
4 Intracranial hypertension
5 Cerebral arterial thrombosis
6 STROKE

- Cysts located in the brain parenchyma that are non-calcific(!) may cause no symptoms and no changes in the CSF but, sometimes, they can give rise to:
  1 Cerebral edema
  2 Epileptic seizures
  3 Focal deficits
  4 Intracranial hypertension

- Other forms of active NCC include:
  1 Vasculitis with resultant brain infarction
  2 Mass effect produced by large cysts or groups of cysts
  3 Intraventricular cysts
    a) Intraventricular cysts may be asymptomatic, but in the event that they block the flow of CSF, they may cause intermittent or continuously increased intracranial pressure

4 Spinal cysts
  a) Cysts developing within the spinal cord may produce an arachnoiditis or symptoms similar to those resulting from any mass lesion

- In both active or inactive NCC, the following can be observed:
  - Vomiting
    1 The vomiting center in the brainstem is stimulated by the toxins of the worms
  - Epilepsy
  - Headache
  - Papilledema—the swelling of an optic disc that is secondary to elevated intracranial pressure
  - Pyramidal tract signs
    1 Involvement of the corticobulbar + corticospinal tracts causing defects in voluntary movements
  - Intellectual deterioration
  - Ataxic gait
    1 Sign of cerebellar damage
  - Diminished visual acuity
  - Optic atrophy
  - Psychosis
  - Diplopia—double vision
  - Vertigo
  - Cranial nerve palsies
  - Behavioral disturbances
Meningeal cysts
- Characterized by:
  1. Intense arachnoiditis which may lead to obstructive hydrocephalus, cranial nerve involvement, intracranial hypertension, arterial thrombosis, and finally, stroke

Parenchymal cysts may give rise to:
- Cerebral edema
- Epileptic seizures of various types
- Focal deficits
- Intracranial hypertension

All these neurologic symptoms largely depend on the following factors:
- Localization of the cyst
- And if the cyst is alive, dying, dead, and calcific

Parenchymal cysticerci that do NOT come in contact with the subarachnoid space and cysticerci in the ventricles that do NOT obstruct the flow of CSF may cause no symptoms when they are alive BUT an intense inflammatory reaction may develop once these worms die
- It has been suggested that this may be secondary not only to the release of antigenic substances by the dead/dying worms but also to cessation of their active immunosuppression, considered to deplete the complement, suppress lymphocyte activity, reduce eosinophilic activity, and have an active cytotoxic effect

3. For ophthalmic cysticercosis
- The cysticerci may be found within the orbit, in either the anterior or posterior chamber of the eye or in the retinal tissues
  - These can be seen with an ophthalmoscope
- Depending on the location of the cysticerci, they may give rise to visual difficulties that fluctuate with eye position or to a generalized decrease in visual acuity with accompanying retinal edema, hemorrhage + vasculitis, or detachment
- The cysts developing along the optic tracts may give rise to visual field defects

Diagnosis
- Surgical removal of subcutaneous or intracranial cysts, with demonstration of the organisms, radiographic demonstration of calcified cysts in the muscle, or visualization of the cysticercus within the orbit are all diagnostic
- Signs and symptoms of a space-occupying lesion of the CNS may be highly suggestive of cysticercosis in the presence of demonstrable cysticerci elsewhere in the body or of a positive serologic test for cysticercosis
  - Ironically, these serologic tests come out negative (-) when used for the diagnosis of neurocysticercosis
- Plain skull films may reveal calcified cysts within the brain
But CT scan shows both calcified + non-calcified cysts and with the use of a contrast medium may show enhancement indicative of inflammatory changes, which may be used to distinguish active from inactive cysts
  - However, CT scan can NOT show intraventricular cysts
  - For cysts in the ventricles, use MRI instead

- Racemose cysts are aberrant cysticerci, which at times, are found developing in the ventricles or the subarachnoid space
  - Such larvae do NOT form a scolex, and the cyst wall grows in an irregular branching and budding fashion to a diameter of several centimeters
  - In using MRI, these racemose cysts are seen to produce unphysiologic enlargements within the ventricles
    - The cysts here can resemble the larvae of Sparganum proliferum
      - But in section, they can be distinguished by the presence of a cavity within the parenchyma as well as by the demonstration of a specific immune complex

Treatment
- If asymptomatic, it requires no treatment (haha sabi ng book kaloka so go lang more uod pa sa utak dedma ka lang ganon?!?!?)
- Anticonvulsants to relieve seizures
- Corticosteroids, if necessary, to control symptoms secondary to meningitis or cerebral edema
- Surgical intervention
  - For direct excision of ventricular cysts
  - Shunting procedures to relieve hydrocephalus
  - Removal of cysts by means of stereotaxic endoscopy
- There is spontaneous disappearance of parenchymal cysticerci especially in children suggesting the advisability of symptomatic treatment in those for whom it is effective and following the evolution of the lesions with CT or MRI
  - Such treatment includes the administration of dexamethasone (24-32mg/day during the acute stage) for patients who developed an acute cysticercotic encephalitis
    - Albendazole is not beneficial in treatment for acute cysticercotic encephalitis
- For active parenchymal brain cysts and subarachnoid cysts, Albendazole is recommended with the dosage 15mg/kg BW for 8-30 days
- For adults with neurocysticercosis, 800mg albendazole/day + 6mg dexamethasone/day for 10 days is safe and effective in decreasing the parasite burden and in reducing the number of seizures
  - Serum levels of the drugs are increased if taken with fatty meal
  - Side effects:
    - Dizziness/Headache
    - Abdominal pain
Lecture 6: Cestodes
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- Diarrhea
- Nausea
- Vomiting
  - Contraindication:
    - Pregnancy

➤ The alternative drug is praziquantel
  - Less effective than albendazole
  - Dosage: 50mg/kg bw in three divided doses daily for 15 days

➤ The simultaneous administration of steroids (dexamethasone) is considered essential in the treatment of subarachnoid cysts but NOT for parenchymal cysts
  - Dexamethasone administration may result in decreased plasma levels of praziquantel BUT increases plasma levels of albendazole
➤ For ophthalmic cysticercosis, surgical removal is often used as a treatment modality.

Epidemiology
➤ Humans acquire cysticercosis through ingestion of eggs passed in human feces
  - Proper disposal of feces, to avoid food contamination, soil, and water, IS essential
➤ Cysticercosis is seen principally in areas where there is a culture of raw pork consumption so that the prevelance of *T. solium* infection is high, and where poor hygiene allows contamination of food stuff by human feces

Control
➤ Thorough cooking of pork or freezing to -5degC for 4 days, -15degC for 3 days, or -24degC for 1 day kills the larvae and if freezing method is practiced universally, it would eliminate the infection from both humans and pigs

B. *Taenia saginata*
➤ AKA the beef tapeworm
➤ Has a worldwide distribution: Africa, Mexico, Argentina, Middle Europe, USA, Asia-Pacific: Korea, Indonesia, Philippines, and Thailand
➤ The life cycle are somewhat similar to that of *T. solium* but instead of a pig, the *T. saginata* uses cattle as the intermediate host, while humans are hosts only to the adult worms (humans are definitive hosts ONLY)
  - Therefore, cysticercosis due to *Taenia saginata* does NOT occur
➤ The existence of the parasite is successful in areas where there is a culture of liking rare (undercooked/uncooked) steak

Morphology
➤ Adults
  - Length: can grow as long as 25 meters (omg) but they are usually 8-12 meters long (omg pa rin)
**Scolex**
- Has 4 muscular suckers
- Has a small rostellum (unarmed)
  - Thus, the rostellar structure can be used to differentiate *T. solium* from *T. saginata* because the former has an armed rostellum and the latter has an unarmed rostellum
- Scolex serves as attachment organ to the mucosa of the upper ½ of the small intestine

**Mature proglottids**
- Shape: nearly square, broader than longer
- Contains both male and female reproductive organs
- Genital organs are same with *T. solium*
- Contains 2x as many testes as *T. solium*
- Contains a bilobed ovary with NO accessory ovarian lobe

**Gravid proglottids**
- Passed in stool
- Longer than wide
- Gravid proglottids of *T. saginata* is longer than those of *T. solium*
- Contains the uterus
  - Uterus
    1. Very much like that of the *T. solium*
    2. But the number of main side branches on each side of the central stem is 15-20
      - Again, a structure that can be used as a basis to differentiate *T. saginata* from *T. solium*

**Eggs**
- Are indistinguishable with *T. solium*
- Shape: round to slightly oval
- Length: 31-43 microns
- Have thick, striated shell and contain the 6-hooked embryo AKA the oncosphere
- Can remain viable in the soil for days-weeks
- Eggs of *T. saginata* from man can NOT be infective to another man

**Life Cycle**
- 1—Embryonated eggs are passed in the feces
- 2—Eggs are ingested by a cattle and develops into a cysticercus (the infective stage larva) in the cattle’s flesh
- 3—Cysticercus in undercooked beef is eaten by human and it develops into adult worms in the upper ½ of small intestine
- 4—Ulit sa #1
The larval stage of *T. saginata*  
- AKA **Cysticercus bovis**  
- **Hexacant** (six-hooked) larva that will hatch from the egg, and will actively penetrate the small intestine and migrate hematogenously to all organ systems  
- Mostly, they lodge in the SKELETAL MUSCLES, where they encyst in the fascial tissues and develop into cisticercus, the infective stage to man

**Infectious Process of T. saginata**  
1. Adult tapeworm is acquired by ingestion of raw or undercooked beef containing the infective stage larva, the cisticercus, **Cysticercus bovis**  
2. Cysticercus, released from its surrounding muscle tissue by digestion in the small intestine, everts its scolex and attaches to the mucosa where it develops to a fully grown adult tapeworm within 3 months  
3. The developing proglottids extend down the small intestine and can reach the jejunum  
4. The most distal proglottids are gravid and they tend to break off from the rest of the worm  
5. The egg-laden segments then actively migrate through the large intestine, rectum, anus, and to outside  
6. Some eggs are usually expressed from the proglottids as it exits from the body and are deposited in the perianal skin. Large ribbons of gravid proglottids may also be passed out with the feces  
7. If the gravid proglottids come out in the grazing area, they may be ingested by cattle, the proglottids are then digested and the eggs are released  
8. The hexacant larva (**Cysticercus bovis**), actively penetrates the small intestine and migrates by the hematogenous route to all organ systems  
9. Most oncospheres lodge in the skeletal muscles where they encyst in the fascial tissues and develop to become a cisticercus infective to man

**Pathology**  
- Again, infection with *T. saginata* is considered to be less serious than from the infection with *T. solium* because *T. saginata* does NOT cause cysticercosis in man  
- The pathology from *T. saginata* is mainly due to its large size which frequently disturbs the normal function of the GIT  
- Absorbed by-product of the worm may also cause systemic intoxication  
- It does NOT cause obstruction although the large tapeworm occupies a substantial proportion of the lumen of the small intestine due to its flexibility  
- There are NO host responses against the worm and therefore, NO immune reaction  
- Proglottids pass from the patient during the period of sleep and are found in the bedding or clothing in the following morning  
- Patient may pass a large segment of the worm either during:  
  - Defecation
Spontaneously

Diagnosis
- Demonstration or identification of the proglottids passed by the patients
  - Method: Fix in 10% formaldehyde solution and the uterine branches injected with india ink (black)
- Demonstration of eggs in the perianal skin
  - Method: Scotch tape swa
    - Eggs in the stool can be identified only up to the genus level Taenia because the eggs of T. solium and T. saginata look alike

Treatment
- Niclosamide
  - MOA: Damage to the worm to the point of dissolution
    - Therefore, decrease worm burden

Prevention and Control
- Protection of cattle from coming in contact with human excreta
- Thorough cooking of beef

C. Echinococcus granulosus
- Causative agent: Hydatid disease, Unilocular echinococcosis, Echinococcus disease
- In the genus of Echinococcus, there are 3 species for which humans are the host to the larval stage (hydatid)
  - E. granulosus
  - E. multilocularis
  - E. vogeli
    - The first three mentioned species have adults worms that can be found parasitizing members of the Canidae (dogs, foxes, wolves, etc)
  - E. oligarthrus—this 4th species is still being debated as to whether it can parasitize humans. It parasitizes felids (family of cats meow!) though.
- The life cycles of all of these echinococcal species follow that of the Echinococcus granulosus which makes E. granulosus the principal pathogen of the genus Echinococcus
- It is the smallest tapeworm
- Only the larval form of infection is found in the human host
- The infection caused by this organism is serious and can be fatal
- Worldwide in distribution
- Man is an accidental host

Morphology
- Adult
Lecture 6: Cestodes
Parsitology: Cestodes

- Length: 0.6 cm or less (sa lecture, 2.5 mm-9 mm ano ba talaga auntie?)
- Possesses:
  - Scolex
    1. Has a prominent rostellum with a double row of 20-40 large
    2. Has small hooklets
    3. Has cup-like suckers
  - Neck
  - Three proglottids (1 immature, 1 mature, and 1 gravid)
    1. Mature Proglottid
      a. Narrowest among the three proglottids
      b. Contains BOTH male and female reproductive organs
    2. Gravid Proglottid
      a. Broadest and longest among the three proglottids
      b. Consists of hundreds of infective eggs
      c. Can be broken off and disintegrate in the large bowel releasing the infective eggs that pass out in the feces

- Egg
  - Cannot be distinguished from Taenia eggs
  - Contains an oncosphere (the embryo), which is hexacant because it bears 6 (hexa) hooklets
    - The oncosphere is brown and radially striated

- Larval Stage (Oncosphere)
  - Diameter: 20 cm
  - Layers of enveloping membrane:
    - Outer
      1. Laminated
      2. Milky
      3. Opaque
      4. Non-nucleated layer
    - Inner
      1. Nucleated germinal layer which will give rise to the protoscoleces/protoscoleces
      2. From this inner membrane arises buds or brood capsules which:
        a. May retain attached to the inner membrane by stalks
        b. May be set free into the fluid of the cystic cavity
          i. The free brood capsules or free scolices are referred to as the hydatid sand
          - The hydatid sand can be aspirated for diagnostic purposes
Life Cycle

1—Infected viscera is eaten by a **dog**/**any canid**
2—**Protoscolices** from **hydatid cyst** attach to the **small intestine** of the dog and grow into an adult worm
3—Eggs that are passed out in the dog’s feces are ingested by **humans** or **herbivores**
4—**Oncosphere** (the embryo) penetrates the intestinal wall→carried to the circulation→can lodge in:
   - Liver
   - Lungs
   - Brain
5—The herbivores that ingested eggs are left untreated and eventually die and they are eaten by dogs making ulit #1 OR the gravid segment can be disintegrated in the large intestine of man and be passed out in the feces making ulit #3

**Infectious Process** and **Generalities** of **Echinococcus**

Upon ingestion by the intermediate host (the dog, the canidae), the embryos, released from their surrounding membranes by action of gastric acid, **bore actively** into the **intestinal wall** and enter a **blood vessel**

- Thus, they may lodge in any part of the body
  - Usually, in the:
    1. Lungs
    2. Liver—**most common site**
- The embryo develops slowly into a **hydatid cyst** (a **space-occupying lesion**), reaching a diameter of **1 cm** in **5 months** and continue to enlarge steadily so that at the **10th** year (or even more) of infection, the hydatid cyst may contain **liters** of **fluid**
  - By the time the cyst of the **E. granulosus** has reached a diameter of 1 cm, its wall is differentiated into a two:
    1. **Thick outer laminated, non-cellular layer** AKA **limiting membrane** which will cover the **2**
    2. **Thin germinal epithelium/layer**
      a. From the germinal epithelium, masses of cells grow into the cavity of the cyst.
        i. These cells become vacuolated and are known as the **brood capsules**
        - **Protoscolices** bud from the inner wall of the brood capsule
        ii. Occasionally, **daughter cysts** appear within the hydatid cyst and these, in turn, produce **brood capsules**, which may contain **protoscolices**
Lecture 6: Cestodes

- The daughter cysts are replicas in miniature of the complete hydatid possessing both the outer thick limiting membrane and the inner germinal layer
  
  iii Gradually the daughter cyst + brood capsules break down to liberate the developed scolices

- Found in the older cysts is the granular material—hydatid sand which is consist of:
  1 Free protoscolices
  2 Daughter cysts
  3 Amorphous material

- Some cysts may never produce brood capsules OR the brood capsules may fail to produce protoscolices
  1 In other cases, hydatids may become sterile because of secondary bacterial infection
  2 In other cases, the hydatids may die and become calcific

- The growth of the hydatid cysts depends on the area where the hydatid cysts are localized
  1 If the area permits growth, they can become very large and cause impairment of vital structures that can cause death
  2 If the area does NOT permit growth, then, they are unable to expand freely, so maliit lang siya

- When the hydatid cyst is formed in the bone (osseous hydatid cyst), development is markedly abnormal, and the limiting membrane (outer membrane) is NOT formed
  
  o The hydatid develops first in the marrow cavity from which it expands and frequently erodes large areas of bone
    
    ▪ The erosion occurs because the cyst can NOT expand within the bone. Despite that, the cyst continues to grow and eventually erode into the bone.

- The minute tapeworm, E. granulosus, lives as an adult in the intestines of dogs and other canids
  
  o There are many strains of E. granulosus that have been observed but the following are the 2 major strains:
    
    ▪ Pastoral strain
      1 Intermediate host: Herbivores
        a The organs and tissues of these herbivores are sites of localization where the hydatid cysts develop
        b Most important intermediate host is SHEEP but the pastoral strain can also parasitize: goats, swine, cattle, and horses
Thus, there is high incidence of **hydatid disease** in areas where **sheep** is being raised
- Ex: Southern Brazil, Argentina, Uruguay, Chile, Peru, Yugoslavia, Bulgaria, Sardinia, Cyprus, Turkey, Lebanon, Africa, in central **Asia**, northern China, New Zealand, and southern Australia
- Most prevalent in: **Turkana, Kenya + Sudan + Ethiopia + Uganda**

- **Sylvatic strain**
  1. Occurs in wolves and ungulates (moose and reindeer)
  2. Occurs in Alaska, Canada, Scandinavia, and northern Eurasia

- Other strains recognized:
  1. **Sheep-dog strain**
  2. **Horse-dog strain**
     - AKA **Echinococcus granulosus equines**
     - Does NOT parasitize humans
  3. **Swiss cattle-dog strain**
  4. **Wallaby-dingo strain**
     - Endemic in Australia

› For many years, **E. multilocularis** was considered to be an abnormal variant of **E. granulosus**
  - But now, it is a valid taxonomic entity in itself
  - Definitive host: **Foxes**
  - Intermediate hosts: **Voles, lemmings, shrews, mice**
    - Can also parasitize dogs/cats in rural communities which domestic cycles occur
     1. In this domestic setting where people pet dogs and cats, people can become infected

› **E. granulosus vs. E. multilocularis**

<table>
<thead>
<tr>
<th>Point of contrast</th>
<th>E. granulosus</th>
<th>E. multilocularis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outer membrane of embryo (the limiting membrane)</td>
<td>Thick</td>
<td>Thin</td>
</tr>
<tr>
<td>Inner membrane (the germinal epithelium)</td>
<td>Does NOT metastasize</td>
<td>May bud externally to proliferate in any direction or even metastasize; it is because of their appearance that they are called <strong>alveolar/multilocular cyst</strong></td>
</tr>
</tbody>
</table>
| Localization of hydatid cyst | Lodge on:
1. Liver
2. Lungs
3. Brain | Lodge on liver usually THEN metastasize to lungs, brain as the cyst grow into the **vena cava/portal vein** |
**E. vogeli**
- Occurs in: Central and South America, Panama, Colombia, Brazil, Peru, and Ecuador
- Parasitizes: Bush dog
- The polycystic laval stage of *E. vogeli* can be found in rodents (pacas and spiny rats)
- The germinal membrane of the hydatid proliferates both:
  - **Inward** → to the original cyst that will form septations that divide it into many sections
  - **Outward** → to form new cysts
- The vesicles forming a polycystic hydatid are relatively large and fluid-filled
- In human infections, the hydatids of *E. vogeli* can localize in the following:
  - Liver—most common site of localization
  - Lungs
  - Pleura
  - Pericardium
  - Heart
  - Intercostal muscles + Diaphragm
  - Stomach + Omentum + Mesenteries
- *Therefore, infection by the hydatids of *E. vogeli* is MORE DISSEMINATED* than infection by hydatids of *E. granulosus* and *E. multilocularis*
- Human infections by *E. vogeli* have been reported from Suriname

**E. oligarthrus**
- Found in: Central and South America
- The adult stage of the tapeworm is found in wild felids (definitive hosts)
- Like *E. vogeli*, the polycystic hydatid cysts in the rodents (intermediate hosts)
- Human infections by *E. oligarthrus* have been reported from Suriname

**Diagnosis**
- Unilocular hydatid cysts are diagnosed ONLY after they have grown to a tremendous size
  - Baket?
Because when the cysts are still small, the patient is **asymptomatic**. Therefore, the patient will not seek consult because there is nothing bothers the normal physiology of the patient.

But when the cysts grow big enough to **impede vital functions** of the organs where the cysts are localized, that is the time when they seek consult.

**Diagnostic modalities for hydatid disease:**

- **X-ray**
  - In x-ray, the cysts show a **sharp outline** and **fluid levels** can sometimes be detected within it.

- **Photoscans**
  - Can indicate the presence of **space-occupying lesions** of the liver/spleen.

- **Utrasound/CT**

- **Exploratory cyst puncture**

- **Immunologic tests**
  - ELISA is satisfactory.

- **Intradermal test**

- **Precipitin test**

- **Complement fixation test**

- **Hemagglutination test**
  - Indirect HemAgglutination test (IHA) is satisfactory.

- **Bentonite latex slide agglutination test**

- **Fluorescent antibody test**

If the cyst is large and abdominal, a characteristic “**thrill**” can sometimes be elicited.

After surgical exposure of a presumed hydatid cyst, aspiration of a portion of its contents can be performed, and examination of fluid removed in this manner may reveal **hydatid sand**.

- It should be remembered that some cysts are **sterile**—they do **NOT** contain **hydatid sand**.

Serologic tests can be confusing as it hydatid disease is cross-reactive with the serologic test for cysticercosis giving a **false positive result**.

**Symptoms**

- Symptoms of the hydatid disease vary according to the location of the cyst.
  - If, as is most commonly the case, the cyst is lodged in the liver, it may cause no symptoms and will be noted only when its increasing size calls attention to its presence.
    - **Hepatic cysts** may cause early symptoms if the location in the liver is such that their **expansion** produces **pressure** on a **major bile duct/blood vessel** or if **intrabiliary rupture occurs**.
    - **Pulmonary cysts** ordinarily are **asymptomatic** until become large enough to give rise to the following:
      - **Coughing**
2 Shortness of breath
3 Chest pains
   - CNS cysts produce serious damage and can elicit neurologic symptoms
     1 Symptoms are very similar to those seen in ocular sparganosis

Pathogenesis

- Generally, the pathology produced by the hydatid cyst in the human body is both mechanical and toxic
  - Mechanical pathology
    - Tremendous size of cyst can interfere with the vital functions of the organs where the cyst are located
    - Infection may become fatal due to growing cyst which can also cause organ obstruction
  - Toxic pathology
    - Rupturing of cyst can elicit an allergic/anaphylactic shock
      1 Anaphylactic shock—a multiorgan system reaction caused by the release of chemical mediators from mast cells and basophils

- The expanding hydatid cyst causes pressure necrosis of surrounding tissues, though cystic growth is slow
  - Therefore, the pressure necrosis can be arrested during the slow growth of the cyst before any vital structures are compromised
  - This pathology is still dependent on the localization of the cyst

- The slow leakage of hydatid fluid from the cyst sensitizes the patient and elicits eosinophilia

- Rupture of an abdominal cyst, either through trauma or in the course of surgery, carries with it both the:
  - Grave risk of anaphylactic shock
  - And the possibility of spread of infection through seeding the peritoneal cavity with hydatid sands or bits of the germinal epithelium—which are capable of producing new cysts

- Rupture of a pulmonary cyst into the bronchus may be marked by severe allergic symptoms + coughing with the production of blood-flecked fluid
  - The blood-flecked fluid may contain hydatid tissue
  - At times, the rupture of a pulmonary cyst may result to spontaneous cure
    - But! Secondary infection may lead to chronic lung abscess

- Circulating immune complexes have been detected and membranous nephropathy demonstrated in patients with hydatid disease

Treatment

- Surgical
Precautionary measures should be in place to prevent the spillage of the cyst fluid or hydatid sand into body cavities which can lead to new cystic infections.

**Pharmacologic**
- **Albendazole**—400mg/2x/day for 4 week
  - Albendazole penetrates well into the hydatid cysts and is therefore more preferred than mebendazole

**PAIR**
- AKA Percutaneous Aspiration, Injection of hypertonic saline or other scolicidal fluid and Reaspiration
  - Praziquantel has some protoscolicidal effect

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**Epidemiology, Prevention, and Control**
- Echinococcal infection is very rare in the Philippines
  - Only 2 cases have been reported
    - 1 case involved the kidney
    - 1 case involved the lung

As discussed earlier, areas of prevalence are those areas where sheep is being raised: Europe, China, Japan, Vietnam, Canada, Alaska, USA, Australia, New Zealand
- Personal hygiene is important in its prevention
- Prevent dogs from eating carcasses of sheep, cattle, and hogs

**D. Diphyllobothrium latum**
- Causative agent for: Diphyllobothriasis, fish tapeworm infection, broad tapeworm infection
- Worldwide in distribution
  - Occurring in northern temperate areas where pickled/insufficiently cooked freshwater fish are prominent in the diet
  - Common in: Northern Europe, Russia, North America, Manchuria, Japan, and South America, Alaska, Canada, occurred once in the Philippines and USA

Infection with *D. latum* is relatively harmless and in some persons, a condition develops that resemble to *pernicious anemia*
- Pernicious anemia (ang lakas maka-physio beh I forgot already)
  - A type of megaloblastic anemia (although NOT all megaloblastic anemia are “pernicious”) due to lack of VitB12 which is required for the production of intrinsic factor (IF) and the IF is required for normal development of RBCs
  - One of the reasons for the condition to mimic pernicious anemia is the relatively high content of VitB12 in the adult worm which the worm “steals” from the host’s diet before it gets absorbed in the small intestine making the host VitB12 deficient
- The life cycle of *D. latum* requires 2 intermediate hosts
  - A copepod
o A freshwater fish

Morphology

⇒ The adult worm

o May be several meters in length (3-10 meters, haha sige after 3 worms di na ako surprised na ganito sila kahaba)
o Color: Ivory or grayish yellow
o Number of proglottids: 3,000

⇒ Scolex

- Spatulate, elongated almond-shaped (sabi ng book: spoon-shaped ano ba)
- Dimensions: 2-3 mm x 1 mm
- **NO** rostellum **nor** hooklets
- With **two (2) longitudinal (dorsoventral sectorial) grooves** called the **bothria**

⇒ Neck

- Unsegmented, several times the length of the scolex

⇒ Mature proglottid

- Wider than they are long
- Contains **BOTH male and female** reproductive organs
- Characteristic feature: **Dark rosette-like coiled uterus** at the center

⇒ Ripe/Gravid proglottid

- Wider than they are long

⇒ The egg

- Ripe eggs escape through the **uterine pore** and are discharge into the intestine
- **BOTH eggs** and **gravid proglottids** may be found in the stool
- Shape: Ovoid
- Has an **operculum** for the escape of the embryo
- Length: 70 microns
- Width: 50 microns
- Shell:
  - **Smooth**
  - **Moderate thickness**
  - **Yellowish brown**
- Contains a lot of **yolk cells**
- Has a **small, knob-like thickening** at one end
- Immature when oviposited

Life Cycle

⇒ 1—Eggs are passed out in the human feces and comes in contact with water
⇒ 2—In water, the eggs complete their embryonation
3—The ciliated coracidium embryo is liberated from the egg is initially planktonic but is eventually ingested by a copepod
   o Copepods from genus *Diaptomus* or *Cyclops*
4—In the copepod, the coracidial embryo develops to become the first larval stage (L1) AKA the procercoid
5—The copepod with the procercoid larva is eaten by a freshwater fish
6—In the freshwater fish, the procercoid larva develops to become second larval stage (L2) AKA the plerocercoid
   o The L1 becomes L2 in the flesh of the freshwater fish
   o The freshwater fish that ingested the copepod is small, therefore, is NOT a usual prey to other marine animals
   o If the small freshwater fish is indeed ingested by a larger fish, the larger fish will not support the continuity of the development of the L2
      ▪ L2 does NOT become adult worm in the larger fish
      ▪ The larger fish is therefore, by definition = paratenic/transport host
7—Man ingests the undercooked freshwater fish (either the small or the larger fish) containing the plerocercoid larva to become adult worm inside the human host's small intestine
8—Ulit sa #1

Diagnosis

Infer from the clinical picture
   o Tapeworm appetite?
   o Abdominal pain?
   o Anemia?

Laboratory
   o Demonstration of the characteristic egg in the stool using acid ether concentration technique
      ▪ This technique will discriminate against eggs of *Diplogonoporus grandis*

Symptoms

The presence of the adult worms in the intestinal tract causes no symptoms in most infected persons
   o There can be vague GIT symptoms and the patient cannot localize where it hurts (naks ang hugot ay I kennat)
   o However, if the worm attaches to the proximal part of the jejunum, clinical VitB12 deficiency develops in a small percentage of those infected
      ▪ This can produce an anemia which resembles pernicious anemia and if diagnosis of the presence of the worm has been made, this is termed to be tapeworm anemia
Lecture 6: Cestodes
Parasitology: Cestodes

- This VitB12-related anemia is MOST common in infected people in Finland because Finnish people are genetically predisposed to pernicious anemia
  1. This suggests that total VitB12 deprivation caused by diphyllobothriasis is NOT enough to cause an anemia in MOST cases

→ Due to the adult worm, the following symptomatologies may occur:
  - **Systemic toxemia**
    - Due to the by-products of the adult worm which is absorbed by the small intestines of the host
  - **Mechanical obstruction**
    - Due to large numbers of worms especially when they become entangled together

Treatment

→ **Praziquantel** is the drug of choice for treatment of diphyllobothriasis in a single oral dose of 5-10mg/kg BW
  - Curative rate is at 95%
→ For persons unable to take praziquantel (coz the patient is allergic to it, etc), **Niclosamide** is an effective alternative but can cause some side effects in some patients including:
  - Abdominal cramps
  - Diarrhea
  - Nausea
  - Vomiting
  - Niclosamide is administered orally, after light breakfast at dosage:
    - Adult=2 grams
    - Children weighing more than 34kg = 1.5 grams
    - Children weighing 11-34kg = 1 gram
→ As the worms are seldom passed spontaneously after administration of either drug, a saline purge may be given 1-2 hrs later to expel them in a more or less intact condition

Epidemiology

→ Various species of freshwater fish and ones that live in brackish waters may be infected with plerocercoid larvae, as may salmonids that spawn in freshwater
→ Both humans and a variety of fish-eating mammals, such as wild and domestic members of the dog and cat families, bears, minks, pigs, walruses, and seals, may become infected
→ In places where there is a culture of eating fish that is raw exhibit a higher prevalence of diphyllobothriasis
  - Such raw fish-eating culture: Jewish, Russian, Finnish, Scandinavians, and Japanese
Prevention
- Thorough cooking of all freshwater fish used for human consumption
- Freezing of fish for 48 hours at -10°C
- Tasting of raw freshwater fish while it is being prepared should NOT be practiced
- Proper treatment and disposal of sewage

E. Dipylidium caninum
- AKA double pored dog tapeworm
- Causative agent for: Dipylidiasis, dog tapeworm infection
- Common in cats and dogs all over the world
  - Occasionally, it is found in humans, particularly, small children
- Human infections occur in: USA, Argentina, Rhodesia, China, and the Philippines
- The larvae of *D. caninum* is called cysticercoids

Morphology
- Adult worm
  - Color: Pale reddish
  - Length: 15-70 cm (sabi sa book, hanggang 80 cm daw hehe so kayo na bahala how to account for the discrepancies between the lecture vs book)
  - Strobila: a chain of melon-shaped proglottids
  - Number of proglottids: 60-175
  - Scolex
    - Shape: Rhomboidal
    - Retractile conical armed rostellum with 30-150 rose thorn-shaped hooklets arranged in transverse rows
    - With four (4) prominent oval suckers
  - Neck
    - Short and slender
  - Immature proglottid
    - Broader than long when very young
    - Assumes as square shape as it matures
  - Mature proglottid
    - Vase-shaped, melon seed-shaped, or pumpkin seed-shaped (haha kaloka ang shapes)
    - With BOTH male and female reproductive organs
    - Genital atrium on each side of the segment
  - Gravid proglottid
    - Vase-, melon seed-, or pumpkin seed-shaped
    - Longer than they are wide
    - The uterus is located in the gravid proglottid
      1. Initially, it appears in the form of a network.
And as the proglottid becomes **gravid**, the uterus breaks up into discrete units called **egg packets** (or **egg capsules**)

- The egg packet is **polygonal** in shape
- Each egg packet may contain **5-30 eggs** (sabi sa lecture 8-15 eggs daw! 😊)

- The gravid proglottids possess a remarkable degree of motility and may migrate actively from the anus
  - They contract and expand vigorously upon reaching the external environment and may remain attached to the fur surrounding the **perianal area** of the dog/cat
  - These contractions are believed to function in the release of the eggs ➔ which are THEN ingested by the flea larvae

** Egg **
- Shape: Spherical
- Covering: **Thick** and **albuminous**
- Contains the **hexacant embryo** with **3 pairs (6)** of **lancet-shaped hooklets**

** Life Cycle **

- **1**—Egg packets are passed out in human feces
- **2**—Eggs hatch when ingested by dog or cat larval fleas producing **cysticercoid larvae**
  - The cysticercoid larvae remain inside the larval fleas until these larval fleas become adult fleas
- **3**—When these fleas are ingested by a (1) dog, (2) cat, or (3) humans, the cysticercoid larvae develop to become **adult worms** in the **small intestines**
- **4**—Ulitsa #1 😊

** Diagnosis **

- Diagnosis of this species is made on finding in the stool the characteristic **barrel-like gravid proglottid**
  - Or rarely, the finding of the **egg packets**
- The following can be demonstrated, too:
  - **Embryonated egg**
- Clinical presentation of infection with *D. caninum* can is non-specific and therefore, making a diagnosis based on just the symptomatology is difficult

** Symptomatology **

- Light infections are **asymptomatic**
  - However, the following can be observed in some infected individuals:
    - Epigastric pain
Lecture 6: Cestodes

- Slight intestinal discomfort
- Diarrhea
- Allergic reactions
- Anal pruritus

Treatment

➤ Praziquantel
  - Is very effective
  - Dosage: Oral intake of 5-10 mg/kg BW

➤ Niclosamide
  - Can be used as an alternative
  - 4 tables (2 grams) chewed thoroughly in a single dose after a light meal

➤ Paromycin
  - 1 g every 4 hours for 4 doses

➤ Quinacrine HCl
  - 0.8 grams given over a half hour interval

Epidemiology

➤ Definitive hosts are dogs and cats and wild carnivores
  - Man is only an accidental host for *D. caninum*
  - Intermediate hosts are larval fleas of dog/cats/human beings
    - Dog flea: *Ctenocephalides canis*
    - Cat flea: *Ctenocephalides felis*
    - Human flea: *Pulex irritans*
    - Dog louse: *Trichodectes canis*
      - These four fleas have larval stages which can be used by the *D. caninum* as its intermediate host
      - Thus, eradication of these fleas through proper pet hygiene greatly reduces the possibility of infection by *D. caninum*

➤ Usually infected are those with pets especially children because they play with pets and mindlessly put their hands in their mouth—oral route of infection

Control and Prevention

➤ Periodic deworming of infected dogs and cats and control of fleas so that the animals do not become reinfected are essential
迦 Niclosamide is also available for veterinary use
迦 Education of children so that they interact with their pets wisely

F. *Hymenolepis nana*

迦 AKA the dwarf tapeworm
迦 Parasitizes the common house mouse
迦 Causative agent for: *Hymenolepiasis nana, Dwarf tapeworm infection*
Mostly infects children but can also infect adults
Usually occurs in: Central Europe, Latin America, India, parts of the old USSR, Hawaii, South and Southwest Pacific, Southeastern USA and in areas with warm climates
Intermediate host: Grain Beetles

Morphology
Adult worm
- Length: 25-40 mm
- Number of proglottids: 200
- Scolex
  - Small and globular
  - Bears a short retractile armed rosetellum with a single ring of 20-30 minute hooklets
  - With 4 cup-shaped suckers
- Neck
  - Long and slender
- Immature Proglottid
  - Undifferentiated
- Mature Proglottid
  - Shape: Trapezoidal
    1 Wider than long
  - Has a single genital pore on its left side towards the anterior border
  - Has BOTH male and female structure: 3 round testes + 1 bilobed ovary
- Gravid Proglottid
  - Wider than long
  - Contains a sac-like uterus filled with eggs
    1 The eggs are usually liberated from the gravid proglottids before the gravid proglottids are detached from the worm body

Eggs
- Color: Grayish Hyaline
- Shape: Nearly spherical or broadly ovoid
- Diameter: 20-40 microns (book: 30-47 microns)
- With 2 thin membranous shells
- They contain the hexacant embryo with 6 hooklets within a rigid membrane
  - This membrane has two thickenings (or knobs) from which project 4-8 long, thin filaments called polar filaments
    1 The presence of the polar filaments is unique AND diagnostic for H. nana
      a The other hymenolepid worm, Hymenolepis diminuta do NOT have these filamentous structures
Life Cycle
- 1—**Embryonated egg** (infective stage) is ingested
- 2—Oncosphere hatches and **cysticercoid larva** develops in the intestinal villus (in the small intestines)
  - The cysticercoid larva has a **single scolex** and has **NO bladder**
    - The presence of the bladder is characteristic of **cysticercus** (bladder worms)
- 3—Cysticercoid larva breaks out from the villus to develop and become an **adult worm** in the small intestine
- 4—The **gravid proglottids** of the adult worm **disintegrate** and is passed out in feces
- 5—Ulit sa #1 😊

Eksena ng **grain beetles** sa life cycle: When grain beetles are ingested accidentally with contaminated **grain products**, it contains the **infective cysticercoids** and thus, ingested grain beetles can be a source of infection
- **Grain Beetles** carry in them **infective cysticercoids** of the **Murine strain** (*Hymenolepis nana* var. *fraterna*)

**Diagnosis**
- Recovery of the eggs in the stool
- Light cases with the aid of acid-ether concentration technique

**Epidemiology**
- *H. nana* has **two strains:**
  - **Human strain**
    - The human strain of *H. nana* is the ONLY tapeworm that does **NOT** require an intermediate host for the completion of its life cycle
    - Man is the **natural final host**
    - Infective stage is the **embryonated ova** transmitted to man through the agency of **food/drink**, particularly **raw leafy vegetables** eaten as **raw salad**
    - It is more common in **children**
    - Humans are the **chief source** of the **human strain**
  - **Murine strain**
    - AKA *Hymenolepis nana* var. *fraterna*
    - Man is **accidental final host**
    - **Intermediate hosts:**
      1. **Fleas**
      2. **Grain Beetles**
    - Man is infected by ingestion of intermediate host harboring the **cysticercoid larva**
- Humans infected through the ingested of contaminated grain products do NOT possess the relative tissue immunity conferred by harboring cysticercoids
  - The eggs produced by this murine strain that hatch in the intestines encounter less immunity and may give rise to the syndrome of hyperinfection

**Modes of Transmission**

- Direct hand to mouth
- Less frequently, by contaminated food or water
- Possibly, by indirect intermediate hosts

**Symptoms**

- Light infections are asymptomatic
- However, some infected individuals may present with:
  - Diarrhea
  - Anorexia
  - Vomiting
  - Insomnia
  - Loss of appetite and weight
  - Irritability
  - Pruritus of anus and nose
  - Urticaria
  - **Choreiform symptoms**

- Heavy infections is invariably pathogenic with the symptoms of:
  - Moderate to profuse diarrhea
  - Abdominal pain
  - Anorexia and exaggerated disorders
  - Extreme apathy
  - Epileptiform seizures

**Treatment**

- **Praziquantel**
  - The drug of choice for hymenolepiasis nana
  - Dosage: 25 mg/kg BW

- **Niclosamide**
  - Must be given daily for 5 days because of the tissue phase of infection
    - Daily dose for adults: **2 grams**
    - Daily dose for children weighing 34kg and above: **1.5 g**
    - Daily dose for children weighing 11kg-34kg: **1 g**

- **Paromycin**
  - Dosage: 45 mg/kg BW daily, given in four doses at hour intervals for a period of 5 days

- **Nitazoxanide**
FDA-approved for cryptosporidiosis and giardiasis but is now being investigated for hymenolepiasis.

Prevention
- For the human strain
  - Avoid ingestion of eggs by not eating raw vegetable salads
  - Personal hygiene
  - Cleanliness of toilet seats
- For the murine strain
  - Eradicate the rats and mice around the house
  - Residual spraying with insecticides
  - Protection of cooked food from arthropods

G. Hymenolepis diminuta
- AKA Rat tapeworm
- Causative agent for: Hymenolepiasis diminuta, Rat tapeworm infection
- Geographic distribution
  - Cosmopolitan parasite of rats, mice, and other rodents
  - Has been reported from human hosts usually from children in: India, Indonesia, USSR, Japan, Philippines, Southern Europe, Latin America (Argentina, Cuba, Mexico), and from several parts of the USA
- More than 20 species of insects are suitable intermediate hosts for H. diminuta but the most important insect hosts are Flour Moths/Flour Beetles

Morphology
- Despite its name, H. diminuta is not minute and is larger than H. nana
- Adult
  - Dimensions: 10-60 cm (book: 20-50 cm) by 3-5 cm
  - Number of proglottids: 800-1,000
  - Scolex
    - Knob-like and club-shaped
    - Provided with a rudimentary apical UNarmed rostellum or a deep apical sectorial pocket without rostral hooklets
    - With 4 small cup-shaped suckers
  - Neck
    - Short and stout
  - Immature proglottid
    - Undifferentiated
  - Mature proglottid
    - Dimensions: 0.8mm by 2.5mm
    - Same as H. nana, only that the segments are larger
  - Ripe or gravid proglottid
    - Sacculate uterus filled with egg mases
Eggs
- Hyaline with straw-colored brown hue
- Shape: Broadly ovoidal or subspherical
- Dimensions: 58 by 86 microns
- Have two egg membranes—the hexacant embryo is enclosed between the two membranes
  - Outer membrane
  - Inner membrane
    - With two polar thickenings but with NO filaments

Life Cycle
1—Embryonated egg (diagnostic stage) are passed out in the feces
2—The embryonated egg is ingested by insects (flour moths/flour beetles)
   - The oncosphere hatches and penetrates the intestinal wall of the insect
3—Now, the embryo grows to become a cysticercoid in the body cavity of the insect
4—The insect is ingested by man/rat
   - The insects are in the flour which are accidentally ingested by man
5—The scolex attaches to the intestine and there, the cysticercoid develops to become an adult worm
6—Gravid proglottids containing the eggs disintegrate
7—Ulitsa #1 😊

Diagnosis
- Recovery of the eggs in the stool

Symptoms
- Light infections are asymptomatic
- Occasionally, patients can present with:
  - Mild GIT problems
  - Nausea
  - Anorexia
  - Abdominal pains
  - Diarrhea

Treatment
- Same as H. nana
  - Praziquantel
  - Niclosamide
  - Paromycin
  - Nitazoxanide

Epidemiology and Control
Disease caused by *H. diminuta* is primarily a **zoonosis**
- Human infections can be prevented by rodent control measures
  - By protection from rodents and their droppings
- Human infections can be prevented by insect control measures
  - Protect cereals, grains, and other food stuff from insect contact

- Many is only an **accidental final host**
- Rats and other murines are the **natural final hosts**

- **Intermediate hosts** are the
  - Mouse fleas
  - Adult mealworm beetle
  - Myriapods
  - Cockroaches
  - Beetles
  - Lepidopterans

- Infective stage to the final host is the **cysticercoid larva** within the **arthropod host**
### Table 2. Summary of the cestodes

<table>
<thead>
<tr>
<th>Scientific name</th>
<th>Common Name</th>
<th>Disease caused</th>
<th>Name of Larva</th>
<th>Intermediate Hosts</th>
<th>Definitive hosts</th>
<th>Treatment</th>
</tr>
</thead>
</table>

References
1. Markell and Voge’s Medical Parasitology (9th edition)
2. Lecture notes by RAsuriano from the lecturer

Downloadable for free at: [www.theelusivedoktora.wordpress.com](http://www.theelusivedoktora.wordpress.com)
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-END-

THANKS-
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