CESTODES (TAPEWORMS)

I. GENERAL
1. The tapeworms are hermaphroditic worms, which as adults parasitize the gastrointestinal tract of vertebrates.
2. They are segmented / flat shape

A. TAPEWORM ANATOMY:
   1. HEAD or SCOLEX, with adhesive organs, at anterior end of worm. Attachment to the intestinal mucosa is accomplished by the scolex.

2. PROGLOTTIDS, the multiple, hermaphroditic, egg-producing units. These are the flattened segments of the worm body.

Medically important species of cestode.

   a. *Diphyllobothrium latum* has a SCOLEX with elongated, slit-like attachment organs → Fish tape worm
   b. *Taenia saginata* has four muscular SUCKERS → Beef tape worm.
   c. *Taenia solium* has similar muscular SUCKERS → Pork tape worm.
   d. *Hymenolepis nana* → Dwarf tape worm.
   e. *Echinococcus granulosis* → Dog tape worm.

B. TAPEWORM LIFECYCLE:

The DEFINITIVE HOST ingests the larval form. Worms mature from larval forms in the intestine of definitive host. The definitive host harbors ADULT WORMS in the intestine. EGGS are passed in the stool.

Eggs are ingested by the INTERMEDIATE HOST. LARVAE develop from eggs in the intermediate host and penetrate the host intestinal mucosa. Larvae develop into ENCYSTED FORMS in tissues of intermediate host. The CYSTICERCUS is the encysted form of the *Taenia* species. The HYDATID is the encysted form of the *Echinococcus*. 
NOTE: *Diphyllobothrium latum* has two intermediate hosts.

C. **HUMAN DISEASE CAUSED BY TAPEWORMS**

1. **ADULT (WORM) STAGE:**
   a. *Taenia saginata, Taenia solium, Diphyllobothrium latum.*
   b. The presence of adult tapeworms in the human GI tract only rarely causes symptomatic disease. People usually only become aware of the infection when proglottids are passed in the feces.

   NOTE: *T. solium* causes human disease in both adult and larval stages.

2. **LARVAL (CYST) STAGE** (i.e., Cysticercosis):
   a. *Echinococcus granulosus, Echinococcus multilocularis, Taenia solium*
   b. The presence of the cyst stage of the tapeworm in extraintestinal tissues causes signs and symptoms relative to the site of the expanding cyst.
D. **LABORATORY IDENTIFICATION OF TAPEWORMS:**

Morphology of the proglottid (degree of branching, configuration, size) is important in distinguishing *T. solium*, *T. saginata*, and sometimes *D. latum*.

Eggs of *T. solium* and *T. saginata* are morphologically indistinguishable, but the eggs of *D. latum* are operculated.

II. **Taenia saginata**:

A. **Epidemiology:** Humans are the only definitive host. Cows and certain other herbivores are intermediate hosts. Disease is very rare in the U.S., because of industrialization of beef production and meat inspection.

B. **Mode of transmission:** Ingestion of poorly cooked beef containing larvae. Larvae mature in human jejunum to mature worms; worms release eggs that pass in the stool. Cattle ingest fodder contaminated with eggs. Embryos mature into larvae within bovine muscle. Macroscopically visible larval cysts in meat give it a "measled" appearance.

C. **Clinical manifestations:** Usually minimal symptomatology.

D. **Pathology:** Minimal. Extremely rare: gastrointestinal obstructions.

E. **Laboratory diagnosis:** Examination of STOOL for proglottids or eggs. Eggs of *Taenia* species are indistinguishable, so definitive diagnosis requires examination of proglottids. The treatment for intestinal *Taenia saginata* and *T. solium* are the same, so definitive diagnosis is not necessary for treatment.

F. **Treatment:** Niclosamide. Praziquantel is used in the treatment of cysticercosis. Surgical excision of cysts may be necessary.

III. **Taenia solium: TAENIASIS**

A. **Epidemiology:** Disease is very rare in the U.S., although it is prevalent through much of the third world, including Mexico.

B. **Mode of transmission:** Infection by ingestion of poorly cooked pork containing encysted larvae.

C. **Clinical manifestations:** Usually minimal symptoms.

D. **Pathology:** Adult worm inhabits the human jejunum and sheds eggs which pass in the stool. Pigs ingest the eggs which release embryos in the GI tract. The embryos travel to systemic tissue where they transform into encysted larvae (i.e., cysticerci). Humans are infected by eating undercooked meat containing the cysticerci.

E. **Laboratory diagnosis:** Examine STOOL for proglottids or eggs; *T. solium* can be distinguished from *T. saginata* by the proglottid branching. *T. solium* have proglottids with 5-10 primary uterine branches, but *T. saginata* proglottids have 15-20.
F. **Treatment:** Niclosamide or praziquantel.

IV. *Taenia solium: Cysticercosis*

A. **Epidemiology:** A period of months to years may elapse between exposure to the parasite and development of symptoms. Therefore, cysticercosis must be considered in the United States in a patient suspicious symptomatology and a history including prior residence in endemic regions.

B. **Mode of transmission:** Humans accidentally become the intermediate host by ingestion of fecally contaminated food or water (most common) or autoinfection (eggs from anus to hand to mouth) or reverse peristalsis (rare).

C. **Clinical manifestations:** Clinical manifestations reflect the organ system affected by the cyst(s). The most common clinical manifestation results from CNS involvement. Symptoms include headache, seizures, paresis. (Cysticercosis is a common cause of childhood seizures in Mexico.)

D. **Pathology:** Embryos emerge from the ingested egg and travel through the human body where cysticerci develop. Common sites of encystment include the CNS, eye, heart, muscle, and skin. Symptoms result from mass effects of the expanding cyst(s) or from the host inflammatory response to degenerating cysts.

E. **Laboratory diagnosis:** Radiological tests (especially CT or NMR of the head with CNS symptoms shows ring-enhancing lesions), serologic tests, sometimes examination of cyst at surgery.

F. **Treatment:** Treatment is based on the site of cysts and the resulting symptomatology. Therapy can include surgical excision and treatment with praziquantel.

V. *Diphyllolobothrium latum:*

A. **Epidemiology:** Worldwide. Disease occurs wherever undercooked, salted, or pickled freshwater fish is eaten by humans. Human feces release eggs into the freshwater thereby infecting the fish.

B. **Mode of transmission:** Humans are infected by eating fresh-water fish containing the infectious larval forms.

C. **Clinical manifestations:** Disease is only rarely symptomatic. Intestinal obstruction can occur.
D. **Pathology:** In the human intestine, the larva matures into an adult worm, which attaches to the small bowel mucosa. Eggs shed into freshwater release larva which pass through two intermediate hosts. There is minimal direct pathology due to the adult worm.

**NOTE:** The adult worm can split the vitamin B12-intrinsic factor complex and prevent absorption of B12 by the human host. Symptomatic B12 deficiency is very rare, but this is always a test question.

E. **Laboratory diagnosis:** Examine STOOL for characteristic operculated egg & proglottids.

F. **Treatment:** Niclosamide or praziquantel.

VI. *Echinococcus granulosus*:

A. **Epidemiology:** Humans are aberrant hosts. Disease occurs in people (e.g., herders or hunters) who have close contact with dogs that may feed contaminated wild herd animals.

B. **Mode of transmission:** Disease has a zoonotic pattern of transmission. Humans ingest eggs shed by dogs that have fed on infected domestic or wild herbivores. The pastoral form is endemic in the American southwest, Australia, and New Zealand. The sylvatic form, maintained by wolves or coyotes feeding on moose, caribou, or deer, is endemic in Alaska, western Canada, and California.

C. **Clinical manifestations:** Disease is due to the mass effect of enlarging cysts, with symptoms that depend on site of encystment. In the pastoral form of the disease, cysts in the liver present as tender palpable hepatic masses. Cysts in the lung may present as an incidental finding on chest x-ray or as cough and hemoptysis.

D. **Pathology:** Dogs & other canines are definitive hosts. The adult worm in dogs is very small, with only three (3) proglottids. Eggs are shed in feces. Sheep and cattle (and wild herbivores in sylvatic form) are intermediate hosts which ingest the eggs and harbor the cysts. In humans, the larvae penetrate the intestinal mucosa, invade submucosal venules, and are distributed to tissue where HYDATID forms. Hydatid cysts enlarge at 1 cm/yr. Common hydatid sites: liver, lung, bone. Hydatid cysts contain multiple BROOD CAPSULES containing SCOLICES. If cysts rupture either spontaneously or during surgical removal, ANAPHYLAXIS can occur.

E. **Laboratory diagnosis:** The diagnosis is usually suggested by the patient history or by radiological findings (e.g., large hepatic cyst). Patients usually have eosinophilia. Diagnosis usually confirmed serology and/or examination of material removed at surgery.
F. Treatment: Surgical excision is the treatment of choice. Mebendazole may be used when surgery is contraindicated or if cysts spill at surgery.

VII. *Echinococcus multilocularis*: Etiologic agent of only minor importance.
   A. Epidemiology: USSR, Canada, and parts of Alaska. Foxes and dogs are definitive hosts. Rodents are usually the intermediate host. Humans are aberrant intermediate hosts who acquire disease usually by eating wild berries contaminated with fox or dog feces.
   B. Pathology: Larvae typically localize in the liver. Cysts have no capsule, so honeycombed masses of vesicles form. Prognosis is poor.

VIII. *Hymenolepis nana*: The dwarf tapeworm (2-4 cm in length).
   A. Epidemiology: *H. nana* occurs worldwide in humans and is also a common murine parasite, occasionally developing its cysticercoid stage in beetles. It is the MOST COMMON tapeworm infection in North America.
   B. Mode of transmission: Ingestion of contaminated grain and flour with beetles. Autoinfection very common in children in day-care centers. Children are especially at risk of infection.
   C. Clinical manifestations: In cases with low intestinal parasite burden, there are no symptoms. In heavy infections, especially if autoinfection and hyperinfection occur, patients can experience diarrhea, abdominal pain, headache, anorexia, and other vague complications.
   D. Pathology: Life cycle is simple and does not require an intermediate. Embryonated eggs are ingested and develop in the intestinal villi into a larval cysticercoid stage. This larva attaches its four muscular suckers and crown of hooklets to the small intestine and the adult worm produces a strobila of egg-laden proglottids. Eggs passed in the feces are then immediately and directly infective, initiating another cycle. Infection may also be acquired by ingesting infected insect intermediate hosts. AUTOINFECTION (reinfection of the small intestine when eggs are released internally) and HYPERINFECTION also occur, causing subsequent increases in worm burden. Eggs hatch in the intestine, develop into a cysticercoid larvae, and then grow into adult worms without leaving the host.
   E. Laboratory diagnosis: The diagnosis is usually suggested by the presence of characteristic *H. nana* eggs upon stool examination (8-10 polar filaments lying between the membrane of the 6-hooked embryo and the shell).
   F. Treatment: Praziquantel is the drug of choice-, an alternative is niclosamide.