PARASITOLOGY 3 & 4 - HELMINTHS

I. INTRODUCTION

Helminths are multicellular organisms responsible for health problems of considerable worldwide importance.

A. LIFE CYCLE:
A series of developmental steps often taking the organism through a number of hosts. These are essential for the parasite to develop.

1. DEFINITIVE HOST:
   Host in which adult develops and reproduces sexually.

2. INTERMEDIATE HOST
   Any animal in which various stages of larval development occur, e.g., insect, crustacean, another vertebrate.

Importance illustrated by the fact that there is no schistosomiasis in North America because the proper snails are not present. Multiplication does not occur in the human host (*Strongyloides stercoralis* and *Hymenolepis nana* are the only exceptions). Therefore the extent of infestation is dependent on the extent of exposure to the infective form. Severity of infection is dependent on the number of worms; the immune status of the host, i.e. some are more severe in the immunocompromised; and the immune response of the host, i.e. tissue reaction may cause symptoms. As a rule these organisms stimulate eosinophils which are important in defending against the infection. Eosinophilia is a helpful diagnostic sign of helminth infestation.

B. DIAGNOSIS OF HELMINTHIC DISEASE

1. Laboratory required since the clinical features of the diseases are non-specific.
2. Parasitologist must be skilled in identification of eggs (ova), larvae or adult worms in stool, urine, blood or tissue.
3. Serological tests are of some benefit.
4. PCR greater role (No longer Parasitology Section in LUMC laboratory)
CLASSIFICATION OF HELMINTHS

Nematodes - Roundworms
Cestodes – Tapeworms
Trematodes - Flukes

II. INTESTINAL NEMATODES

A. Intestinal: Attach to mucosa and obtain nourishment from host tissue or through intestinal lumen. Some may exhibit extensive migratory patterns through man.

B. Life cycles: Asexual reproduction or larval reproduction uncommon. Most have one definitive host.

C. Pathogenicity: Cause disease not only due to presence in intestine but also because of a reaction of the host to larval or adult products, especially during migration. e.g. – asthma with eosinophilia during lung migration.

III. ACQUIRED BY PASSAGE THROUGH THE SKIN

A. STRONGYLOIDES STERCORALIS
2 mm x 45 um female

IV.

1. Epidemiology: Tropical and subtropical climates

2. Life cycle
a. Adult in submucosa of upper small bowel lays eggs. Eggs hatch and rhabditiform (R) larva is passed in feces. R larva develops in soil to infective filariform (F) larva which penetrates skin → lymphatics → bloodstream → lungs and alveoli where coughed and swallowed. Maturation to adult occurs in small bowel and colon.

b. Free-living Cycle: In optimal climate the R larva can develop into adult worm and reproduce in the soil.
c. **Autoinfection Cycle:** *R* larvae can develop into infective *F* larva in intestine of man. *F* larva will penetrate intestinal wall and migrate through lung back to upper small bowel. Adult worm population increases in man and infection maintained > 30 years. Hyperinfection in immunosuppressed.

3. **Symptoms:**
   a. Lung migration - cough, wheezing, fever
   b. Light - asymptomatic.
   c. Moderate to heavy - anemia weight loss, chronic diarrhea and fever.
   d. Hyperinfection syndrome - overwhelming sepsis and multiple organ involvement in immunocompromised host including HTLV-I infection due to autoinfection.
   e. Chronic – creeping eruption.

4. **Diagnosis:**
   a. Marked eosinophilia.
   b. Stool - rhabditiform larva (short buccal cavity - 275um x 16um). (30-50% sensitive). Enhancing methods
      - Duodenal aspirate - egg and larvae.
   c. Serology – ELISA- important as single stool positive 30-50%
   d. PCR not commercially available

5. **Treatment:**
   a. Ivermectin, albendazole (alternative)
   b. **Must eradicate the infection**

B. **HOOKWORM**
   Major species – *Ancylostoma duodenale, Necator americanus*
Morphologic differentiation requires adult worm. 
Adult female 10mm.

1. **Epidemiology**: Worldwide. Tropical and subtropical.

2. **Life cycle**: Same as in Strongyloides except:
   a. Egg passed in feces - hatches in soil
   b. No free-living or autoinfection cycle
   c. Adult life span up to 5 years

3. **Symptoms**:
   a. Ground itch - papular rash with itching at site of skin entry.
   b. Light - asymptomatic.
   c. Moderate to heavy – GI symptoms and iron deficiency anemia and its sequelae.
      Ancylostoma-0.15-0.25 ml blood/day/adult worm.  Necator 0.03 ml blood/day/adult worm).  Dependent on dietary iron.

4. **Diagnosis**:
   a. Should be considered as cause of hypochromic microcytic anemia in endemic area
   b. Eggs in feces - similar for both species

5. **Treatment**
   a. Prevention, Iron, Diet
   b. Albendazole, Mebendazole or Pyrantel pamoate

**ACQUIRED BY INGESTION**

**C. ASCARIASIS - ASCARIS LUMBRICOIDES**

1. **Epidemiology**:
   a. Very common worldwide. This is due to:
      i. Each female lays 200,000 eggs/day for 1 year.
      ii. Eggs have thick coat and are very resistant to dryness, cold, etc.

2. **Life cycle**:
   Eggs ingested → hatch small bowel.  Larva penetrates → carried to lung coughed and swallowed. Adult develops in upper small bowel (female 30 cm; male 20 cm).  Ova require several weeks in warm environment to become infective.

3. **Symptoms**:
   a. Lung - asthma-like
   b. Light - asymptomatic
c. Moderate to heavy - intestinal or biliary tract obstruction occur. Extremely rare recorded cases of worm migration through nose or skin.

4. **Diagnosis:**
   a. Eggs easily found in stool
   b. X-ray – adults ingest barium

5. **Treatment:**
   a. Albendazole, Mebendazole, Ivermectin, Nitazoxanide

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**D. VISCERAL LARVA MIGRANS, OCULAR LM, NEURAL LM**

Human is accidental (not true) host

*Toxocara canis* or *T. catis* ascarid worms of dog or cat

*Baylisascaris procyonis* – ascarid of raccoon.

1. **Epidemiology:**
   a. Worldwide

2. **Life cycle:**
   a. Embryonated eggs are ingested usually by children. The larvae hatch, penetrate intestinal mucosa and wander aimlessly in the viscera - liver, brain, eye, spinal cord, lungs, cardiac muscle,
3. **Pathogenesis:**
a. Wandering second stage larvae produce hemorrhage and chronic inflammation. Eosinophilic infiltration. Granuloma around the dead larva.

4. **Manifestations:** Three Syndromes:
a. Visceral larva migrans – fever, malaise, myalgias, cough, hepatomegaly
   i. Uncommon myocarditis, encephalitis, pneumonia
   ii. Leukocytosis with hypereosinophilia
b. Ocular – retinal damage → visual loss
c. Neural – meningoencephalitis, seizures, motor and cognitive impairment. High mortality

5. **Diagnosis:**
a. Recover or see the larva, e.g. liver biopsy, ophthalmoscope. Serology. ELISA most sensitive and specific. Anti *B. procyonis* antibodies in blood and CSF.
b. Cause of eosinophilic meningitis

6. **Treatment:**
a. Self limited. Albendazole or Mebendazole
b. No treatment proven effective for Baylisascaris
   i. One success with albendazole and corticosteriods
c. Corticosteroids - eye.

E. **ENTEROBIASIS - PINWORM** (no lung migration)
*Enterobius vermicularis*
Adult female 10mm x 0.4mm.

1. **Epidemiology:** Worldwide
2. **Life cycle:** Man ingests egg → hatches in duodenum several larval stages before reaching ileum and cecum. Female migrates to perianal and perineal regions to deposit eggs.
3. **Transmission:**
a. Anus → hand → mouth.
b. Inhalation of dust.
c. Fomites → hand → mouth.
d. Retroinfection from anus eggs hatch and larvae migrate (rare)
4. **Symptoms:**
a. Perianal and perineal itching, incriminated in many childhood psychiatric problems and behavioral disorders. Also said to
cause granulomatous lesions in genitourinary organs but this is most unusual.

5. **Diagnosis:**
   a. Scotch tape - ovum

6. **Treatment:**
   a. Albendazole, Mebendazole or Pyrantel pamoate. Single dose is now preferred over other therapies (with repeat single dose two weeks after the first). Aggressive household disinfection procedures have not proven effective.
   b. Other family members should be tested and all positives treated.

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F. **TRICHURIS TRICHIURA - WHIPWORM** (No lung migration)

1. **Epidemiology:** Very high infection rate. 2.2 million in U.S.

2. **Life Cycle:**

<table>
<thead>
<tr>
<th>Egg (soil)</th>
<th>ingested by man</th>
<th>larva hatches small intestinal villi 3-10 days</th>
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<tbody>
<tr>
<td>↑</td>
<td></td>
<td>↓</td>
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<tr>
<td>feces</td>
<td>migrates to cecum and imbeds 4-6 years</td>
<td></td>
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</tbody>
</table>

   a. Adult - 30-50 mm
   b. Thin whiplike anterior end. Posterior end widens and contains reproductive organs. Anterior attaches to mucosa of cecum and ascending colon.

3. **Symptoms:** Majority asymptomatic
   a. Abdominal symptoms: diarrhea, pain, nausea, vomiting.
b. Dysentery with many worms → rectal prolapse.
c. Anemia - worms obtain blood - but anemia rare.
d. Symptoms occur with larger worm burdens.

4. **Diagnosis:**
   a. Eggs in feces - no concentrations necessary.
   b. Lemon shaped with bile stained polar plugs.

5. **Treatment:**
   a. Mebendazole (preferred), Albendazole, Ivermectin, Oxantel pamoate
   b. Treatment with single drugs less effective
   c. Combination with mebendazole and Oxantel pamoate
   d. Light infection – not necessary

G. **TRICHINELLA SPIRALIS - TRICHINOSIS**

1. **Life cycle** - Trichinosis infects carnivorous animals. It shows little host specificity but rats, swine and bears are commonly infected.

   - Skeletal muscle of man, pig, bear, etc. → Poorly cooked pork → Ingested by man
   - Contains encysted larvae
   - Brain
   - Myocardium etc.
   - Bloodstream → Lymphatics → Larvae penetrate mucosa in intestine
   - Adult worms mate (5 days)

2. **Epidemiology** :
   a. Worldwide - 1941 - 16%
   b. USA autopsy diaphragms - 1968 - 4%
   c. Currently most cases in US from ingestion of wild game

3. **Disease**
      During maturation attachment and reproduction of adults.
      i. Diarrhea - 4%; nausea & vomiting - 10%; abdominal pain - 20%; headache - mild
      ii. Length of intestinal phase days to weeks.
b. Phase II - Migration and encyst. 7-42+ days. Fever, muscle pain and tenderness, headache, periorbital edema, cough and dyspnea, rash, heart, esp. ECG abnormalities.
   i. CNS disease may be severe.
   ii. Deaths occur during this stage.

c. Phase III
   i. Recovery
   ii. Encystment in muscle.

4. Diagnosis:
   a. Eosinophilia
   b. Muscle enzymes (CK, LDH) - during phase II in 50% or more
   c. Serology - 3-4 weeks after infection.
   d. Muscle biopsy - crush preparation

5. Treatment:
   a. Albendazole. Kill adults in intestine and stop production of new larvae
   b. Severe: Albendazole + Corticosteroids

H. EOSINOPHILIC MENINGITIS
1. Agent: Angiostrongylus cantonensis - Rat Lung Worm
2. Life Cycle: Adults in rat lung. Eggs hatch in lung and larvae passed in rodent feces → Larvae infect snails, molt to reach infective stage → Snail, vegetation contaminated with snail, or crab or shrimp that ate snail ingested by human → Released larvae penetrate intestinal vasculature, reach the meninges and die.
3. Disease:
   a. Two to four weeks after ingestion
   b. Headache, fatigue, meningeal signs, hyper or paraesthesias, vomiting, diarrhea, fever
4. Diagnosis:
   a. Eosinophils in spinal fluid
   b. May find larva in CSF
   c. Serology in CSF and serum sensitive and specific
5. Therapy:
   a. No antihelmenthic shown effective
      i. Small trials with albendazole and mebendazole possibly effective
   b. Corticosteroids for increased pressure

III. BLOOD AND TISSUE NEMATODES
Filaria: Infections caused by arthropod transmitted organisms. The adults reside in lymphatic or subcutaneous tissues and females bear live microfilaria which either circulate in blood or move about in subcutaneous tissues. Co-infection (of the nematode) with symbiotic, rickettsia-like Wolbachia recently described (tetracycline now used in treatment).

A. **LYMPHATIC FILARIASIS**

**WUCHERERIA BANCROFTI**
Tropical and subtropical South America, Africa, southern and southeast Asia.

**BRUGIA MALAYI**
Southern and southeast Asia

1. **Vector:** Mosquito-several species
2. **Epidemiology:** Very high infection rate in endemic areas 20-25%.
3. **Symptoms:**
   a. Majority-light asymptomatic infection.
   b. Immune unresponsiveness - specific cellular and humoral factors which actively suppress the immune response have been described in Brugia malayi infection. This presumably protects against immune mediated injury.
   c. Elevated IgG levels with depressed cell mediated immunity against nonfilarial antigens has also been described.
   d. Acute inflammation - lymphangitis and lymphadenitis which subsides and recurs. May get headache, back pain but little fever associated with circulating microfilariae.
   e. Chronic obstructive lesions - Allergic tissue reactions around live or dead adult filariae in lymphatics destroy architecture leading to obstruction, chronic lymphedema and elephantiasis of legs and genitalia most common. Also breast and intraabdominal lymphatics involved.
   f. Tropical pulmonary eosinophilia – nocturnal cough and wheeze
4. **Diagnosis:**
   a. Eosinophilia
   b. Night blood smears. (↑ circulating microfilariae at night)
   c. Serology - very non-specific (cross-reacts)
5. **Treatment:**
   a. Diethylcarbamazine, doxycycline
b. Kills microfilaria. No effect in obstructive disease.
c. Triple drug regimen (DEC, albendazole, ivermectin more effective than dual combinations.

B. **ONCHOCERCA VOLVULUS**

Adult female 30-50 mm  
Adult and microfilariae found in dermis and subcutaneous tissue  
Tropical rain forests of Africa and Central and South America  
18,000,000 in world; 5% blind

1. **Vector:** - Blackfly Simulium species
2. **Life cycle:** - Adults mature in subcutaneous tissue -- form nodules. Microfilariae migrate through subcutaneous tissues and are ingested by fly. Microfilariae rarely found in viscera.

3. **Disease:**

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<th>Signs and symptoms due almost entirely to dead or dying microfilariae</th>
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   a. Nodules. Firm (mm-1cm) painless, non-tender nodules, especially over bony prominences. These contain encapsulated masses of adult worms. Overlying skin moves freely.
   b. Dermatitis. Caused by enzyme release and reaction to antigen products of migrating microfilaria. Pruritic papular lesions which become excoriated. Destruction of supporting subcutaneous tissue gives skin an aged appearance.
   c. Eye. Can affect virtually all eye structures during migration. Keratitis (cornea) and corneal fibrosis common. Also conjunctivitis and iritis. Released *Wolbachia* enhances inflammation in eye structures.

4. **Diagnosis:** Physical findings (incl. eye), skin snips, PCR, serology, eosinophilia.
5. **Treatment:** Screen for *Loa loa* before treating. Ivermectin, doxycycline then ivermectin new drug Moxidectin

C. **LOA LOA**
Adult female 50-70mm
West Central Africa

1. **Vector**: Tabanid flies - Chrysops
2. **Life cycle:**
   a. Adults move in subcutaneous tissue.
   b. Microfilariae circulate.
3. **Disease:**
   a. Calabar swelling. Painful, edematous, non-erythematous swellings in skin, 10-20 cm diameter. May be allergic reaction to worm products or to dead worms. Resolve over several days.
   c. Serology – non specific
      i. IgG4 antibody test
   d. PCR
4. **Treatment:**
   a. Diethylcarbamazine - very effective in *Loa loa*.
      i. Experienced center recommended due to side effects
   b. Surgery

D. **DRACUNCULUS MEDINENSIS - GUINEA WORM** (not a filaria)

Adult female - 2.5 - 4 feet
Africa, Asia (esp. India), West Indies and Northeastern South America
Estimated worldwide prevalence - 50 million

1. **Life cycle**: Gravid female in subcutaneous tissue protrudes uterus into skin blister. Blister forms ulcer from which larvae are discharged into water when immersed. Larvae ingested by copepod of genus Cyclops in which development to infective stage takes place. Copepods accidentally ingested by man with subsequent release of larva in intestine. Larva migrates through intestine to subcutaneous tissue where development to adult occurs.
2. **Disease:**
   a. Secondary bacterial infection of blister ultimately resulting in damaging infections to tendons, joints, bones.
   b. Hypersensitivity phenomenon (urticaria, itching, asthma) just preceding protrusion of gravid female.
3. **Diagnosis**: See worm in skin. Identify larvae in ulcer.
4. **Treatment:**
   a. Surgical removal of worm
IV. CESTODES

A. CESTODES - TAPEWORM

Long, ribbon-like segmented worm
Segment - proglottid contains reproductive apparatus

1. Life cycle: Intermediate host (pig, cattle or fish) ingests eggs from human feces Larval form encysts in muscle of intermediate host. Man acquires infection by eating poorly cooked meat or fish.
   a. Taenia saginata: Beef - man intestine only
   b. Taenia solium: Pork - man can become intermediate host by ingesting eggs passed in human feces and resulting in cysticercosis.
   c. Diphyllobothrium latum: Fish - Scandinavia, Minnesota, and Alaska
   d. Hymenolepis nana: Dwarf tapeworm, intermediate host is flea or beetle; Definitive hosts: human, rat, mouse. Larval development in human or murine intestine (intermediate host not required).

b. No drugs effective—treat secondary bacterial infection.

5. Prevention: Replace step in wells with pump for drinking water.
2. **Symptoms:**
   a. Mild abdominal symptoms with all.
   b. *D. latum* - effectively competes with host for vitamin B12, therefore, pernicious anemia rarely occurs.
   c. *T. saginata* - proglottids are motile and produce perianal sensations on movement.
   d. Ingestion of *T. solium* eggs results in *cysticercosis.* The larvae are carried to multiple tissues where they encyst. Symptoms produced when they *encyst in CNS: epilepsy, meningoencephalitis, coma, death.* This may occur years later when host inflammatory response to the dying parasite causes symptoms.
   e. Human infection with *H. nana* occurs after ingestion of intermediate host (flea or beetle) OR after ingestion of human or mouse feces containing ova. In either case larval development occurs in human intestine. **Thus ova from adult H. nana in human intestine develop into adult worms before fecal passage. This autoinfection cycle may result in a light infection becoming a very heavy one.** Enteritis may accompany heavy infection.
3. **Diagnosis:** Eggs or proglottids in feces

4. **Treatment:**
   a. Praziquantel for Tenia species, *D. latum, H. nana*
   b. Albendazole or Praziquantel plus corticosteroids for cysticercosis
   c. Niclosamide – alternative to praziquantel for all intestinal tapeworms

B. **ECHINOCOCCAL CYST**

1. **ECHINOCOCCUS GRANULOSUS**
   This is a dog tapeworm in which sheep are the usual intermediate host. Man, when closely involved with dogs and sheep, may accidentally ingest egg from dog feces and become intermediate host. The cyst in man develops a germinal layer from which multiple daughter cysts develop.
   a. **Disease:**
      i. Space occupying cystic lesions in multiple organs. Over 2/3 are in liver. Lung is next most common. Skeletal lesions are unusual but can cause symptoms.
      ii. Liver cysts found in investigation of asymptomatic liver enlargement.
      iii. Rarely cause bile duct obstruction.
      iv. Lung - occasional cough.
      v. Bone - pathologic fractures.
   b. **Diagnosis:**
      i. Imaging
      ii. See protoscolices in cyst fluid
      iii. Serology,
   c. **Treatment:**
      i. Surgery or percutaneous drainage + albendazole - extreme caution since spillage may cause an aphylactic reaction.
      ii. PAIR: percutaneous aspiration, injection (formalin), reaspiration
      iii. Albendazole at time or aspiration or surgery or alone when surgery contraindicated.
Echinococcus Multilocularis

Very similar parasite in which foxes, wolves and wild felines are the definitive hosts, but domestic dogs and cats become infected. Intermediate hosts are rodents including mice. It occurs in cold climates. Spreads like malignancy in humans because it lacks a germinal capsule.

V. Trematoda or Flukes

Organisms characterized by complex life cycle in which multiple larval stages pass through water. Involve crustaceans.

Schistosomiasis

S. Haematobium – Africa and Middle East – GU tract
S. Mansoni – Arabia, Africa, S. America, Caribbean
S. Japonicum – Japan, China, Philippines

1. Life cycle:
   a. Free-swimming cercariae (larvae) penetrate human skin → lungs → liver where they mature, mate and pass down into mesenteric or vesical (bladder) venules to begin egg production. Eggs (feces or urine of man) reach fresh water, hatch and larvae (miracidia) penetrate specific snails which after several molts, release the cercariae.

2. Pathobiology:
   a. Adult schistosomes are not eliminated by the immune system.
Host proteins are adsorbed to surface of organism.

b. Intense fibrotic reaction to ova around liver or bladder (ureters) is probably mediated by cytokines and involves T helper lymphocytes.

3. **Disease:**
   a. Katayama fever – hypersensitivity reaction - acute febrile illness lasting 4-8 weeks following heavy exposure. Fever, cough lymphadenopathy, hepatosplenomegaly, eosinophilia. Most common with japonicum.
   b. Chronic - eggs which do not escape in feces are carried back to liver (mansoni and japonicum) where they produce an intense granulomatous fibrotic reaction. This results in periportal fibrosis and portal hypertension. Patients develop ascites and esophageal varices. Because of periportal location, synthetic function of liver is preserved. *S. haematobium* ova may produce obstructive uropathy and genital elephantiasis.
   c. Dermatitis – Exposure to non-human schistosomes in freshwater lakes in N. America. “Swimmer’s Itch”

4. **Diagnosis:**
   a. Eggs - feces or urine
   b. Rectal biopsy - crush preparation
   c. Serology – Important for diagnosis in travelers

5. **Treatment:**
   a. Haematobium - Praziquantel
   b. Mansoni - Praziquantel
   c. Japonicum - Praziquantel

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**B. OPISTHORCHIS (CLONORCHIS) SINENSIS - CHINESE LIVER FLUKE**
1. **Life cycle:**
a. Adult lives in biliary tree of human, dog and cat. Ova passed in feces ingested by fresh water snail ultimately releasing cercaria which enter fish and encyst as infective metacercaria in fish flesh. Released after ingestion by human and adult migrates luminally to biliary tree.

2. **Epidemiology:**

3. **Disease:**
a. Usually asymptomatic
b. Occasionally obstructive jaundice, cholangitis
c. Rarely cirrhosis
d. Associated with cholangiocarcinoma

4. **Diagnosis:**
a. Ova in feces
b. Serology

5. **Treatment:**
a. Praziquantel or Albendazole

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C. **PARAGONIMUS WESTERMANI**

1. **Life cycle:**
a. Adult in cystic cavities of lung. Ova, expectorated and shed in sputum or feces. Miracidia hatch in fresh water and enter snail. Cercariae emerge and enter crab or crayfish which is eaten raw by man. Adolescent worm wanders in body cavity 3 weeks then penetrates diaphragm and enters lung where it initiates reaction and may survive 20 years.

2. **Epidemiology:**
a. Zoonosis affecting fur bearing animals around the world.
b. Most human disease occurs in Orient.
c. Recent description of US outbreak caused by *Paragonimus kellicotti* from eating crayfish in Missouri.

3. **Disease:**
4. **Diagnosis:**
   a. Ova in sputum or feces
   b. Eosinophilia
   c. Serology

5. **Treatment:**
   a. Praziquantel or Triclabendazole
STUDY QUESTIONS

1. Explain why treatment of asymptomatic infection with Trichuris trichiura in a traveler who has returned to the U.S. from Cambodia might not be necessary.

2. Name the nematodes that are acquired through skin penetration and migrate through the lung.

3. Name the nematodes that are acquired by ingestion and migrate through the lung.

4. Name the nematode that can autoinfect (reinfect) the human intestine, complete its life cycle and thus cause lifelong infection without acquiring new organisms from the environment.

5. The clinical parasitology laboratory identifies infection with *Strongyloides stercoralis* by visualizing ______________________________ in the stool specimen.

6. Explain why treatment of infection with Strongyloides is mandatory.

7. The major clinical manifestation in persons with moderate to heavy infection with hookworm is ________________________.

8. The morphologic feature which contributes most to symptoms caused by *Ascaris lumbricoides* is its ________________________________.

9. How does visceral larva migrans basically differ from infection with other intestinal nematodes?

10. Specimens for diagnosis of infection with *Enterobius vermicularis* are best obtained by ________________________________.

11. 0 is the characteristic ova of which intestinal nematode?

12. Name the parasite acquired by eating undercooked, infested pork which causes myositis, cardiac conduction defects and encephalitis in heavily infected patients.

13. Name three filaria species in which microfilaria circulate in the blood.

14. The vector of the filaria infection which causes chronic lymphedema is the ________________________________.
15. Which form (adult or microfilaria) of *Onchocerca volvulus* causes damage to subcutaneous supporting tissues and the eye?

16. Are subcutaneous lesions that occur in infection with *Onchocerca volvulus* painful? What about the subcutaneous lesions that occur with *Loa loa*?

17. How do humans acquire infection with *Dracunculus medinensis*?

18. Which of the human intestinal tapeworms can encyst in the extraintestinal tissues of man if its ova are ingested?

19. Which tapeworm may cause macrocytic anemia?

20. Which tapeworm can complete its life cycle in the intestine of man and thus multiply into many tapeworms?

21. What is the most common clinical manifestations in man if disease results from ingestion of ova from the dog tapeworm?

22. Schistosomiasis (other than swimmers' itch) does not occur in the U.S. because ________________________________.

23. Which schistosome species cause chronic portal hypertension?

24. What morphologic form of the schistosome fluke causes chronic inflammatory portal hypertension?

25. What morphologic form of the schistosome fluke penetrates the skin establishing infection in man?

26. How does one diagnose infection with Schistosoma haematobium?

27. ____________ is the ova of what helminth?

28. Man acquires infection with *Opisthorchis sinensis* by ________________________________.
28. The major site of disease in persons infected with *Paragonimus westermani* is the _________________________________. 
EXAMPLE OF TEST QUESTION

The adult form of this cestode is not found in the human intestine.

A. *Taenia solium*
B. *Taenia saginata*
C. *Hymenolepis nana*
D. *Diphyllobothrium latum*
E. *Echinococcus granulosus*

CORRECT ANSWER TO ABOVE QUESTION: E