PARASITOLOGY - CHAPTER FOUR
NEMATODES (Round Worms)

INTESTINAL HELMINTHS

Intestinal nematodes of importance to man are *Ascaris lumbricoides* (roundworm), *Trichinella spiralis* (trichinosis), *Trichuris trichiura* (whipworm), *Enterobius vermicularis* (pinworm), *Strongyloides stercoralis* (Cochin-china diarrhea), *Ancylostoma duodenale* and *Necator americanus* (hookworms) and *Dracunculus medinensis* (fiery serpents of the Israelites). *E. vermicularis* and *T. trichiura* are exclusively intestinal parasites. Other helminths listed above have both intestinal and tissue phases.

*Ascaris lumbricoides* (Large intestinal roundworm)

![Figure 1. Ascaris Life Cycle](https://example.com/ascaris_life_cycle.png)

Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces. Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed, the larvae hatch, invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs. The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed. Upon reaching the small intestine, they develop into adult worms. Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years.

**Epidemiology**
The annual global morbidity due to ascaris infections is estimated at 1 billion with a mortality of 20,000. Ascariasis can occur at all ages, but it is more prevalent in the 5 to 9 years age group. The incidence is higher in poor rural populations.

**Morphology**
The average female worm measures 30 cm x 5 mm. The male is smaller.

**Life cycle (figure 1)**
The infection occurs by ingestion of food contaminated with infective eggs which hatch in the upper small intestine. The larvae (250 x 15 micrometers) penetrate the intestinal wall and enter the venules or...
A fertilized Ascaris egg, still at the unicellular stage, as they are when passed in stool. Eggs are normally at this stage when passed in the stool (complete development of the larva requires 18 days under favorable conditions). CDC DPDx Parasite Image Library

lymphatics. The larvae pass through the liver, heart and lung to reach alveoli in 1 to 7 days during which period they grow to 1.5 cm. They migrate up the bronchi, ascend the trachea to the glottis, and pass down the esophagus to the small intestine where they mature in 2 to 3 months. A female may live in the intestine for 12 to 18 months and has a capacity of producing 25 million eggs at an average daily output of 200,000 (figure 2). The eggs are excreted in feces, and under suitable conditions (21 to 30 degrees C, moist, aerated environment) infective larvae are formed within the egg. The eggs are resistant to chemical disinfectant and survive for months in sewage, but are killed by heat (40 degrees C for 15 hours). The infection is man to man. Auto infection can occur.

Symptoms
Symptoms are related to the worm burden. Ten to twenty worms may go unnoticed except in a routine stool examination. The commonest complaint is vague abdominal pain. In more severe cases, the patient may experience listlessness, weight loss, anorexia, distended abdomen, intermittent loose stool and occasional vomiting. During the pulmonary stage, there may be a brief period of cough, wheezing, dyspnea and substernal discomfort. Most symptoms are due to the physical presence of the worm.

Diagnosis
Diagnosis is based on identification of eggs (40 to 70 micrometers by 35 to 50 micrometers - figure 2) in the stool.

Treatment and Prevention
Mebendazole, 200 mg, for adults and 100 mg for children, for 3 days is effective. Good hygiene is the best preventive measure.
Trichinellosis is acquired by ingesting meat containing cysts (encysted larvae) of *Trichinella*. After exposure to gastric acid and pepsin, the larvae are released from the cysts and invade the small bowel mucosa where they develop into adult worms (female 2.2 mm in length, males 1.2 mm; life span in the small bowel: 4 weeks). After 1 week, the females release larvae that migrate to the striated muscles where they encyst. *Trichinella pseudospiralis*, however, does not encyst. Encystment is completed in 4 to 5 weeks and the encysted larvae may remain viable for several years. Ingestion of the encysted larvae perpetuates the cycle. Rats and rodents are primarily responsible for maintaining the endemicity of this infection. Carnivorous/omnivorous animals, such as pigs or bears, feed on infected rodents or meat from other animals. Different animal hosts are implicated in the life cycle of the different species of *Trichinella*. Humans are accidentally infected when eating improperly processed meat of *Trichinella spiralis* (Trichinosis)

**Epidemiology**
Trichinosis is related to the quality of pork and consumption of poorly cooked meat. Autopsy surveys indicate about 2 percent of the population is infected. The mortality rate is low.

**Morphology**
The adult female measures 3.5 mm x 60 micrometers. The larvae in the tissue (100 micrometers x 5 micrometers) are coiled in a lemon-shaped capsule.

**Life cycle**
Infection occurs by ingestion of larvae, in poorly cooked meat, which immediately invade intestinal mucosa and sexually differentiate within 18 to 24 hours. The female, after fertilization, burrows deeply in the small intestinal mucosa, whereas the male is dislodged (intestinal stage). On about the 5th day eggs begin to hatch in the female worm and young larvae are deposited in the mucosa from where they reach the lymphatics, lymph nodes and the blood stream (larval migration). Larval dispersion occurs 4 to 16 weeks after infection. The larvae are deposited in muscle fiber and, in striated muscle, they form a capsule which calcifies to form a cyst. In non-striated tissue, such as heart and brain, the larvae do not calcify; they die and disintegrate. The cyst may persist for several years. One female worm produces approximately 1500 larvae. Man is the terminal host. The reservoir includes most carnivorous and omnivorous animals (Figure 3 and 4).

**Symptoms**
Trichinosis symptoms depend on the severity of infection: mild infections may be asymptomatic. A larger bolus of infection produces symptoms according to the severity and stage of infection and organs involved (Table 1).

**Pathology and Immunology**
Trichinella pathogenesis is due the presence of large numbers of larvae in vital muscles and host reaction to larval metabolites. The muscle fibers become enlarged edematous and deformed. The paralyzed muscles are infiltrated with neutrophil, eosinophils and lymphocytes. Splenomegaly is dependent on the degree of infection. The worm induces a strong IgE response which, in association with eosinophils, contributes to parasite death.

**Diagnosis**
Diagnosis is based on symptoms, recent history of eating raw or undercooked meat and laboratory findings (eosinophilia, increased serum creatine phosphokinase and lactate dehydrogenase and antibodies to *T. spiralis*).

**Treatment and Control**
Steroids are used for treatment of inflammatory symptoms and Mebendazole is used to eliminate worms. Elimination of parasite infection in hogs and adequate cooking of meat are the best ways of avoiding infection.
These carnivorous animals (or eating food contaminated with such meat). CDC DPDx Parasite Image Library

**Figure 4**

Encysted larvae of Trichinella in pressed muscle tissue. The coiled larvae can be seen inside the cysts. CDC DPDx Parasite Image Library

Larvae of Trichinella, freed from their cysts, typically coiled; length: .8 to 1 mm. Alaskan bear. CDC DPDx Parasite Image Library

Trichinella spiralis larvae in muscle section (H&E) and muscle press © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

**Table 1**

<table>
<thead>
<tr>
<th>Trichinosis symptomatology</th>
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<tbody>
<tr>
<td><strong>Intestinal mucosa</strong> (24-72 hrs)</td>
</tr>
<tr>
<td>Nausea, vomiting, diarrhea, abdominal pain, headache.</td>
</tr>
<tr>
<td><strong>Circulation and muscle</strong> (10-21 days)</td>
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<tr>
<td>Edema, peri-orbital conjunctivitis, photo phobia, fever, chill, sweating, muscle pain, spasm, eosinophilia.</td>
</tr>
<tr>
<td><strong>Myocardium</strong> (10-21 days)</td>
</tr>
<tr>
<td>Chest pain, tachycardia, EKG changes, edema of extremities, vascular thrombosis.</td>
</tr>
<tr>
<td><strong>Brain and meninges</strong> (14-28 days)</td>
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<tr>
<td>Headache (supraorbital), vertigo, tinnitus, deafness, mental apathy, delirium, coma, loss of reflexes.</td>
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**Trichuris trichiura (whipworm)**

**Epidemiology**
Trichuriasis is a tropical disease of children (5 to 15 yrs) in rural Asia (65% of the 500-700 million cases). It is, however, seen in the two Americas, mostly in the South and is concentrated in families and groups with poorer sanitary habits.

**Morphology**
The female organism is 50 mm long with a slender anterior (100 micrometer diameter) and a thicker (500 micrometers) posterior end. The male is smaller and has a coiled posterior end. The Trichuris
The unembryonated eggs are passed with the stool (1). In the soil, the eggs develop into a 2-cell stage (2), an advanced cleavage stage (3), and then they embryonate (4); eggs become infective in 15 to 30 days. After ingestion (soil-contaminated hands or food), the eggs hatch in the small intestine, and release larvae (5) that mature and establish themselves as adults in the colon (6). The adult worms (approximately 4 cm in length) live in the cecum and ascending colon. The adult worms are fixed in that location, with the anterior portions threaded into the mucosa. The females begin to oviposit 60 to 70 days after infection. Female worms in the cecum shed between 3,000 and 20,000 eggs per day. The life span of the adults is about 1 year. CDC DPDx Parasite Image Library

**Life cycle**

Infection occurs by ingestion of embryonated eggs in soil. The larva escapes the shell in the upper small intestine and penetrates the villus where it remains for 3 to 10 days. Upon reaching adolescence, the larvae pass to the cecum and embed in the mucosa. They reach the ovipositing age in 30 to 90 days from infection, produce 3000 to 10,000 eggs per day and may live as long as 5 to 6 years. Eggs passed in feces embryonate in moist soil within 2 to 3 weeks (Figure 5 and 6). The eggs are less resistant to desiccation, heat and cold than ascaris eggs. The embryo is killed under desiccation at 37 degrees C within 15 minutes. Temperatures of 52 degrees C and -9 degrees C are lethal.

**Symptoms**

Symptoms are determined largely by the worm burden: less than 10 worms are asymptomatic. Heavier infections (e.g., massive infantile trichuriasis) are characterized by chronic profuse mucus and bloody diarrhea with abdominal pains and edematous prolapsed rectum. The infection may result in malnutrition, weight loss and anemia and sometimes death.

**Diagnosis**

Diagnosis is based on symptoms and the presence of eggs in feces (50 to 55 x 20 to 25 micrometers).

**Treatment and Control**

Mebendazole, 200 mg, for adults and 100 mg for children, for 3 days is effective. Accompanying infections must be treated accordingly. Improved hygiene and sanitary eating habits are most effective in control.
**Enterobius vermicularis** (pinworm)

**Epidemiology**
Enterobiasis is by far the commonest helminthic infection in the US (18 million cases at any given time). The worldwide infection is about 210 million. It is an urban disease of children in crowded environment (schools, day care centers, etc.). Adults may get it from their children. The incidence in whites is much higher than in blacks.

**Morphology**
The female worm measures 8 mm x 0.5mm; the male is smaller. Eggs (60 micrometers x 27 micrometers) are ovoid but asymmetrically flat on one side.

**Life cycle**
Infection occurs when embryonated eggs are ingested from the environment, with food or by hand to mouth contact. The embryonic larvae hatch in the duodenum and reach adolescence in jejunum and upper ilium. Adult worms descend into lower ilium, cecum and colon and live there for 7 to 8 weeks. The gravid females, containing more than 10,000 eggs migrate, at night, to the perianal region and deposit their eggs there. Eggs mature in an oxygenated, moist environment and are infectious 3 to 4 hours later. Man-to-man and auto infection are common (Figure 7 and 8). Man is the only host.

**Symptoms**
Enterobiasis is relatively innocuous and rarely produces serious lesions. The most common symptom is perianal, perineal and vaginal irritation caused by the female migration. The itching results in insomnia and restlessness. In some cases gastrointestinal symptoms (pain, nausea, vomiting, etc.) may develop. The conscientious housewife's mental distress, guilt complex, and desire to conceal the infection from her friends and mother-in-law is perhaps the most important trauma of this persistent, pruritic parasite.

**Diagnosis**
Diagnosis is made by finding the adult worm or eggs in the perianal area, particularly at night. Scotch tape or a pinworm paddle is used to obtain eggs.

**Treatment and Control**
Two doses (10 mg/kg; maximum of 1g each) of Pyrantel Pamoate two weeks apart gives a very high cure rate. Mebendazole is an alternative. The whole family should be treated, to avoid reinfection. Bedding and underclothing must be sanitized between the two treatment doses. Personal cleanliness provides the most effective in prevention.
oviposit while crawling on the skin of the perianal area (5). The larvae contained inside the eggs develop (the eggs become infective) in 4 to 6 hours under optimal conditions (1). Retroinfection, or the migration of newly hatched larvae from the anal skin back into the rectum, may occur but the frequency with which this happens is unknown. CDC

**Figure 8**

Enterobius vermicularis adults in section of appendix (H&E) © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

**Figure 9**

The *Strongyloides* life cycle is complex among helminths with its alternation between free-living and parasitic cycles, and its potential for autoinfection and multiplication within the host. Two types of cycles exist:

Free-living cycle: The rhabditiform larvae passed in the stool (1) (see "Parasitic cycle" below) can either molt twice and become infective filariform larvae (direct development) (6)

Parasitic cycle: The infective larvae of *S. stercoralis* penetrate the skin of man, enter the venous circulation and pass through the right heart to lungs, where they penetrate into the alveoli. From there, the adolescent parasites ascended to the glottis, are swallowed, and reach the upper part of the small intestine, where they develop into adults. Ovipositing females develop in 28 days from infection. The eggs in the intestinal mucosa hatch...

### Strongyloides stercoralis (Threadworm)

**Epidemiology**

Threadworm infection, also known as Cochin-China diarrhea, estimated at 50 to 100 million cases worldwide, is an infection of the tropical and subtropical areas with poor sanitation. In the United States, it is prevalent in the South and among Puerto Ricans.

**Morphology**

The size and shape of threadworm varies depending on whether it is parasitic or free-living. The parasitic female is larger (2.2 mm x 45 micrometers) than the free-living worm (1 mm x 60 micrometers) (figure 10). The eggs, when laid are 55 micrometers by 30 micrometers.

**Life cycle** (figure 9)

The infective larvae of *S. stercoralis* penetrate the skin of man, enter the venous circulation and pass through the right heart to lungs, where they penetrate into the alveoli. From there, the adolescent parasites ascend to the glottis, are swallowed, and reach the upper part of the small intestine, where they develop into adults. Ovipositing females develop in 28 days from infection. The eggs in the intestinal mucosa, hatch...
or molt four times and become free living adult males and females (2) that mate and produce eggs (3) from which rhabditiform larvae hatch (4). The latter in turn can either develop (5) into a new generation of free-living adults (as represented in (2)), or into infective filariform larvae (6). The filariform larvae penetrate the human host skin to initiate the parasitic cycle (see below) (6). Parasitic cycle: Filariform larvae in contaminated soil penetrate the human skin (6), and are transported to the lungs where they penetrate the alveolar spaces; they are carried through the bronchial tree to the pharynx, are swallowed and then reach the small intestine (7). In the small intestine they molt twice and become adult female worms (8). The females live threaded in the epithelium of the small intestine and by parthenogenesis produce eggs (9), which yield rhabditiform larvae. The rhabditiform larvae can either be passed in the stool (1) (see "Free-living cycle" above), or can cause autoinfection (10). In autoinfection, the rhabditiform larvae become infective filariform larvae, which can penetrate either the intestinal mucosa (internal autoinfection) or the skin of the perianal area (external autoinfection); in either case, the filariform larvae may follow the previously described

into infective filariform larvae and enter a new host (indirect cycle), or mature into adult worms to repeat the free-living cycle.

**Symptoms**

Light infections are asymptomatic. Skin penetration causes itching and red blotches. During migration, the organisms cause bronchial verminous pneumonia and, in the duodenum, they cause a burning mid-epigastric pain and tenderness accompanied by nausea and vomiting. Diarrhea and constipation may alternate. Heavy, chronic infections result in anemia, weight loss and chronic bloody dysentery. Secondary bacterial infection of damaged mucosa may produce serious complications.

**Diagnosis**

The presence of free rhabditiform larvae (figure 10) in the feces is diagnostic. Culture of stool for 24 hours will produce filariform larvae.

**Treatment and control**

Ivermectin or thiabendazole can be used effectively. Direct and indirect infections are controlled by improved hygiene and auto-infection is controlled by chemotherapy.

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**Figure 10**

*Strongyloides stercoralis*

The esophageal structure is clearly visible in this larva; it consists of a club-shaped anterior portion, a post-median constriction, and a posterior bulbus CDC DPDx Parasite Image Library

*Strongyloides stercoralis* Note the prominent genital primordium in the mid-section of the larva; note also the Entamoeba coli cyst near the tail of the larva. CDC DPDx Parasite Image Library

*Strongyloides stercoralis* rhhabditiform larva © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission
route, being carried successively to the lungs, the bronchial tree, the pharynx, and the small intestine where they mature into adults; or they may disseminate widely in the body. To date, occurrence of autoinfection in humans with helminthic infections is recognized only in *Strongyloides stercoralis* and *Capillaria philippinensis* infections. In the case of *Strongyloides*, autoinfection may explain the possibility of persistent infections for many years in persons who have not been in an endemic area and of hyperinfections in immunodepressed individuals CDC DPDx Parasite Image Library

Figure 11

Hookworm life cycle. Eggs are passed in the stool (1), and under favorable conditions (moisture, warmth, shade), larvae hatch in 1 to 2 days. The released rhabditiform larvae grow in the feces and/or the soil (2), and after 5 to 10 days (and two molts) they become become filariform (third-stage) larvae that are infective (3). These infective larvae can survive 3 to 4 weeks in favorable environmental conditions. On contact with the human host, the larvae penetrate the skin and are carried through the veins to the heart and then to the

**Necator americanes and Ancylostoma duodenale** (Hookworms)

**Epidemiology**
Hookworms parasitize more than 900 million people worldwide and cause daily blood loss of 7 million liters. Ancylostomiasis is the most prevalent hookworm infection and is second only to ascariasis in infections by parasitic worms. *N. americanes* (new world hookworm) is most common in the Americas, central and southern Africa, southern Asia, Indonesia, Australia and Pacific Islands. *A. duodenale* (old world hookworm) is the dominant species in the Mediterranean region and northern Asia.

**Morphology**
Adult female hookworms are about 11 mm x 50 micrometers. Males are smaller. The anterior end of *N. americanes* is armed with a pair of curved cutting plates whereas *A. duodenale* is equipped with one or more pairs of teeth. Hookworm eggs are 60 micrometers x 35 micrometers.

**Life cycle** (figure 11 and 12)
The life cycle of hookworms is identical to that of threadworms, except that hookworms are not capable of a free-living or auto-infectious cycle. Furthermore, *A. duodenale* can infect also by oral route.

**Symptoms**
Symptoms of hookworm infection depend on the site at which the worm is present (Table 2) and the burden of worms. Light infection may not be noticed.
lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed (4). The larvae reach the small intestine, where they reside and mature into adults. Adult worms live in the lumen of the small intestine, where they attach to the intestinal wall with resultant blood loss by the host (5). Most adult worms are eliminated in 1 to 2 years, but longevity records can reach several years. Some *A. duodenale* larvae, following penetration of the host skin, can become dormant (in the intestine or muscle). In addition, infection by *A. duodenale* may probably also occur by the oral and transmammary route. *N. americanus*, however, requires a transpulmonary migration phase. CDC DPDx Parasite Image Library

<table>
<thead>
<tr>
<th>Site</th>
<th>Symptoms</th>
<th>Pathogenesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermal</td>
<td>Local erythema, macules, papules (ground itch)</td>
<td>Cutaneous invasion and subcutaneous migration of larva</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Bronchitis, pneumonitis and, sometimes, eosinophilia</td>
<td>Migration of larvae through lung, bronchi, and trachea</td>
</tr>
<tr>
<td>Gastro-intestinal</td>
<td>Anorexia, epigastric pain and gastro-intestinal hemorrhage</td>
<td>Attachment of adult worms and injury to upper intestinal mucosa</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Iron deficiency, anemia, hypoproteinemia, edema, cardiac failure</td>
<td>Intestinal blood loss</td>
</tr>
</tbody>
</table>

**Diagnosis**
Diagnosis is made by identification of hookworm eggs in fresh or preserved feces. Species of hookworms cannot be distinguished by egg morphology.

**Treatment and control**
Mebendazole, 200 mg, for adults and 100 mg for children, for 3 days is effective. Sanitation is the chief method of control: sanitary disposal of fecal material and avoidance of contact with infected fecal material.

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![Hookworm eggs examined on wet mount (eggs of Ancylostoma duodenale and Necator americanus cannot be distinguished morphologically). Diagnostic characteristics: Size 57-76 μm by 35-47 μm Oval or ellipsoidal shape Thin shell. The embryo in B has begun cellular division and is at an early (gastrula) stage](Image)

![Ancylostoma duodenale adult male and female](Image) Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

![Necator americanus adult female, anterior end](Image) Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

![Hookworm filariform larva](Image) Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

![Necator americanus adult male, posterior end](Image) Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

![Hookworm eggs](Image) Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission
Dracunculus medinensis (Guinea worm; fiery serpent of the Israelites)

**Epidemiology**
Guinea worm is estimated to infect about 50 million people in North, West and Central Africa, southwestern Asia, the West Indies and northeastern South America.

**Morphology**
The adult female worm measures 50-120 cm by 1 mm and the male is half that size.

**Life cycle**
The infection is caused by ingestion of water contaminated with water fleas (Cyclops) infected with larvae. The rhabditiform larvae penetrate the human digestive tract wall, lodge in the loose connective tissues and mature into the adult form in 10 to 12 weeks. In about a year, the gravid female migrates to the subcutaneous tissue of organs that normally come in contact with water and discharges its larvae into the water (figure 13). The larvae are picked up by Cyclops, in which they develop into infective form in 2 to 3 weeks.

**Symptoms**
If the worm does not reach the skin, it dies and causes little reaction. In superficial tissue, it liberates a toxic substance that produces a local inflammatory reaction in the form of a sterile blister with serous exudation. The worm lies in a subcutaneous tunnel with its posterior end beneath the blister, which contains clear yellow fluid. The course of the tunnel is marked with induration and edema. Contamination of the blister produces abscesses, cellulitis, extensive ulceration and necrosis.

**Diagnosis**
Diagnosis is made from the local blister, worm or larvae. The outline of the worm under the skin may be revealed by reflected light.

**Treatment**
Treatment includes the extraction of the adult guinea worm by rolling it a few centimeters per day or preferably by multiple surgical incisions under local anaesthesia. Metronidazole is effective in killing the worm. Protection of drinking water from being contaminated with Cyclops and larvae are effective preventive measures.
Toxocara canis and T. catti (visceral larva migrans)

These are roundworms of dogs and cats but they can infect humans and cause damage of the visceral organs. Eggs from feces of infected animals are swallowed by man and hatch in the intestine. The larvae penetrate the mucosa, enter the circulation and are carried to liver, lungs, eyes and other organs where they cause inflammatory necrosis. Symptoms are due to the inflammatory reaction at the site of infection. The most serious consequence of infection may be loss of sight if the worm localizes in the eye. Treatment includes Mebendazole to eliminate the worm and prednisone for inflammatory symptoms. Avoidance of infected dogs and cats is the best prevention (figure 14 and 15).

Ancylostoma braziliensis (cutaneous larva migrans, creeping eruption)

Creeping eruption is prevalent in many tropical and subtropical countries and in the US especially along the Gulf and southern Atlantic states. The organism is primarily a hookworm of dogs and cats but the filariform larvae in animal feces can infect man and cause skin eruptions. Since the larvae have a tendency to move around, the eruption migrates in the skin around the site of infection. The symptoms last the duration of larval persistence which ranges from 2 to 10 weeks. Light infection can be treated by freezing the involved area. Heavier infections are treated with Mebendazole. Infection can be avoided by keeping away from water and soil contaminated with infected feces (figure 16 and 17).
Hookworm eggs examined on wet mount (eggs of Ancylostoma duodenale and Necator americanus cannot be distinguished morphologically). Diagnostic characteristics: 
Size 57-76 µm by 35-47 µm 
Oval or ellipsoidal shape 
Thin shell 
The embryo in B has begun cellular division and is at an early (gastrula) developmental stage. 

The released rhabditiform larvae grow in the feces and/or the soil, and after 5 to 10 days (and two molts) they become infective (third-stage) larvae that are infective. These infective larvae can survive 3 to 4 weeks in favorable environmental conditions. On contact with the human host, the larvae penetrate the skin and are carried through the veins to the heart and then to the lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed. The larvae reach the small intestine, where they reside and mature into adults. Adult worms live in the lumen of the small intestine, where they attach to the intestinal wall with resultant blood loss by the host. Most adult worms are eliminated in 1 to 2 years, but longevity records can reach several years.

Some A. duodenale larvae, following penetration of the host skin, can become dormant (in the intestine or muscle). In addition, infection by A. duodenale may probably also occur by the oral and transmammary route. N. americanus, however, requires a transpulmonary migration phase.

BLOOD AND TISSUE HELMINTHS

The major blood and tissue parasites of man are microfilaria. These include Wuchereria bancrofti and W. (Brugia) Malayi, Onchocerca volvulus, and Loa loa (eye worm).

Wuchereria bancrofti and W. (Brugia) malayi (elephantiasis)

Epidemiology
W. bancrofti (figure 18) is strictly a human pathogen and is distributed in tropical areas worldwide, whereas B. malayi (figure 19) infects a number of wild and domestic animals and is restricted to South-East Asia. Mosquitoes are vectors for both parasites.

Morphology
These two organisms are very similar in morphology and in the diseases they cause (figure 18 and 19). Adult female W. bancrofti found in lymph nodes and lymphatic channels are 10 cm x 250 micrometers whereas males are only half that size. Microfilaria found in blood are only 260 micrometers x 10 micrometers. Adult B. malayi are only half the size of W. bancrofti but their microfilaria are only slightly
Lymphatic Filariasis- An Introduction
27 min SureStream Movie
WHO-RealVideo

Brazil: The Magician
8 minute SureStream movie
The story of Dr Gerusa Dreyer and her attempts to tackle lymphatic filariasis in Recife
WHO-RealVideo

smaller than *W. bancrofti*.

**Life cycle**
Filariform larvae enter the human body during a mosquito bite and migrate to various tissues. There, they may take up to a year to mature and produce microfilaria which migrate to lymphatics (figure 19) and, at night, enter the blood circulation. Mosquitos are infected during a blood meal. The microfilaria grow 4 to 5 fold in the mosquito in 10 to 14 days and become infective for man.

**Symptoms**
Symptoms include lymphadenitis and recurrent high fever every 8 to 10 weeks, which lasts 3 to 7 days. There is progressive lymphadenitis due to an inflammatory response to the parasite lodged in the lymphatic channels and tissues. As the worm dies, the reaction continues and produces a fibro-proliferative granuloma which obstructs lymph channels and causes lymphedema and elephantiasis (figure 20). The stretched skin is susceptible to traumatic injury and infections. Microfilaria cause eosinophilia and some splenomegaly. Not all infections lead to elephantiasis. Prognosis, in the absence of elephantiasis, is good.

**Diagnosis**
Diagnosis is based on history of mosquito bites in endemic areas, clinical findings and presence of microfilaria in blood samples collected at night.

**Treatment and control**
Diethylcarbamazine quickly kills the adults worms or sterilizes the female. It is given 2 mg/kg orally for 14 days. Steroids help alleviate inflammatory symptoms. Cooler climate reduces the inflammatory reaction.

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**Figure 18**

A Different species of the following genera of mosquitoes are vectors of *W. bancrofti* filariasis depending on geographical distribution. Among them are: *Culex* (*C. annulirostris, C. bitaeniorhynchus, C. quinquefasciatus, and C. pipiens*); *Anopheles* (*A. arabiensis, A. bancroftii, A. farauti, A. funestus, A. gambiae, A. koliensis, A. melas, A. merus, A. punctulatus and A. wellcomei*); *Aedes* (*A. aegypti, A. aquasalis, A. bellator, A. cooki, A. darlingi, A. kochi, A. polynesiensis, A. pseudoscutellaris, A. rotumae, A. scapularis, and A. vigilax*); *Mansonella* (*M. pseudotitillans, M. uniformis*); *Coquillettidia* (*C. juxtamansonia*). During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop in adults that commonly reside in the lymphatics. The female worms measure 80 to 100 mm in length and 0.24 to 0.30 mm in diameter, while the males measure about 40 mm by 1 mm. Adults produce microfilariae measuring 244 to 296 µm by 7.5 to 10 µm, which are sheathed and have nocturnal periodicity, except the South Pacific microfilariae which have the absence of marked periodicity. The microfilariae migrate into lymph and blood channels moving actively through lymph and blood. A mosquito ingests the microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and some of them work their way through the wall of the proventriculus and cardiac portion of the mosquito’s midgut and reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate through the hemocoel to the mosquito's proboscis and can infect another human when the mosquito takes a blood meal.

B Microfilaria of *Wuchereria bancrofti*, from a patient seen

C Microfilaria of *Wuchereria bancrofti* collected by filtration with a nucleopore membrane. Giemsa stain, which does not
in Haiti. Thick blood smears stained with hematoxylin. The microfilaria is sheathed, its body is gently curved, and the tail is tapered to a point. The nuclear column (the cells that constitute the body of the microfilaria) is loosely packed, the cells can be visualized individually and do not extend to the tip of the tail. The sheath is slightly stained with hematoxylin. CDC DPdx Parasite Image Library
demonstrate the sheath of this sheathed species (hematoxylin stain will stain the sheath lightly). The pores of the membrane are visible. CDC DPdx Parasite Image Library

Figure 19

A Microfilaria of Brugia malayi. Thick blood smear, hematoxylin stain. Like Wuchereria bancrofti, this species has a sheath (slightly stained in hematoxylin). Differently from Wuchereria, the microfilariae in this species are more tightly coiled, and the nuclear column is more tightly packed, preventing the visualization of individual cells. CDC DPdx Parasite Image Library

B Detail from the microfilaria of Brugia malayi showing the tapered tail, with a subterminal and a terminal nuclei (seen as swellings at the level of the arrows), separated by a gap without nuclei. This is characteristic of B. malayi. CDC DPdx Parasite Image Library

C Wuchereria bancrofti adults in section of lymph node (H&E) © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

D Wuchereria bancrofti microfilaria in peripheral blood, giemsa stain © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

E The typical vector for Brugia malayi filariasis are mosquito species from the genera Mansonia and Aedes. During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop into adults that commonly reside in the lymphatics. The adult worms resemble those of Wuchereria bancrofti but are smaller. Female worms measure 43 to 55 mm in length by 130 to 170 µm in width, and males measure 13 to 23 mm in length by 70 to 80 µm in width. Adults produce microfilariae, measuring 177 to 230 µm in length and 5 to 7 µm in width, which are sheathed and have nocturnal periodicity. The microfilariae migrate into lymph and enter the blood stream reaching the peripheral blood. A mosquito ingests the microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and work their way through the wall of the proventriculus and cardiac portion of the midgut to reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage larvae. The third-stage larvae migrate through the hemocoel to the mosquito’s proboscis and can infect another human when the mosquito takes a blood meal. CDC DPdx Parasite Image Library

Figure 20

Elephantiasis of leg due to filariasis. Luzon, Philippines.
**Onchocerca volvulus (Blinding filariasis; river blindness)**

**Epidemiology**
Onchocerciasis is prevalent throughout eastern, central and western Africa, where it is the major cause of blindness. In the Americas, it is found in Guatemala, Mexico, Colombia and Venezuela. The disease is confined to neighborhoods of low elevation with rapidly flowing small streams where black flies breed. Man is the only host.

**Morphology**
Adult female onchocerca measure 50 cm by 300 micrometers, male worms are much smaller. Infective larvae of *O. volvulus* are 500 micrometers by 25 micrometers (figure 21).

**Life cycle**
Infective larvae are injected into human skin by the female black fly (*Simulium damnosum*) where they develop into adult worms in 8 to 10 months. The adults usually occur as group of tightly coiled worms (2 to 3 females and 1 to 2 males). The gravid female releases microfilarial larvae, which are usually distributed in the skin. They are picked up by the black fly during a blood meal. The larvae migrate from the gut of the black fly to the thoracic muscle where they develop into infective larvae in 6 to 8 days. These larvae

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<th>CDC</th>
<th>Inguinal lymph nodes enlarged due to filariasis</th>
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<th>Histopathology showing cross section of Dirofilaria worm in eye</th>
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<td></td>
<td>An elderly village chief undresses prior to bathing. He has elephantiasis of the left leg, large hydrocoele, leopard skin and onchocerciasis nodules clearly visible on his torso. WHO/TDR/Crump</td>
<td></td>
<td>An elderly village chief sits bathing himself outside his home with water from a bowl. He has elephantiasis of the left leg, large hydrocoele, leopard skin on the left leg and onchocerciasis nodules. WHO/TDR/Crump</td>
<td></td>
<td>An elderly male with hydrocoele, elephantiasis of the leg, hanging groin, leopard skin and onchocerciasis nodules. WHO/TDR/Crump</td>
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<td></td>
<td>The feet of a male villager showing elephantiasis and skin lesions of the left leg and foot. WHO/TDR/Crump</td>
<td></td>
<td>This lady has elephantiasis of the right leg and oedema in the left. WHO/TDR/Crump</td>
<td></td>
<td>An elderly male with hydrocoele, elephantiasis of the leg, hanging groin and leopard skin. WHO/TDR/Crump</td>
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Steps necessary to establish an effective community-directed treatment system using ivermectin to combat onchocerciasis.

WHO-RealVideo

Steps necessary to establish an effective community-directed treatment system using ivermectin to combat onchocerciasis.

WHO-RealVideo

Symptoms
Onchocerciasis results in nodular and erythematous lesions in the skin and subcutaneous tissue due to a chronic inflammatory response to persistent worm infection. During the incubation period of 10 to 12 months, there is eosinophilia and urticaria. Ocular involvement consists of trapping of microfilaria in the cornea, choroid, iris and anterior chambers, leading to photophobia, lacrimation and blindness (figure 21).

Diagnosis
Diagnosis is based on symptoms, history of exposure to black flies and presence of microfilaria in nodules.

Treatment and control
Diethylcarbamazine is effective in killing the worm. Destruction of microfilaria produces extreme allergic reaction which can be controlled with corticosteroids. Prevention measures include vector control, treatment of infected individuals and avoidance of black fly.

Onchocerca volvulus. CDC/Dr. Lee Moore

Onchocerca volvulus, posterior end. CDC/Dr. Lee Moore

Onchocerca volvulus, from skin snip from a patient seen in Guatemala. Wet preparation. Some important characteristics of the microfilariae of this species are shown here: no sheath present; the tail is tapered and is sharply angled at the end. CDC DPDx Parasite Image Library

Onchocerca volvulus adults in section of tumour (H&E) © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Histopathology of Onchocerca volvulus nodule. Onchocerciasis. CDC/Dr. Mae Melvin

Face of a blind male patient in the onchocerciasis ward. WHO/TDR/Crump

An old man, blinded by onchocerciasis. WHO/TDR/Crump

Life cycle of Onchocerca volvulus.

Onchocerca volvulus. CDC/Dr. Lee Moore

During a blood meal, an infected blackfly (genus Simulium) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. In subcutaneous tissues the larvae develop into adult filariae, which commonly reside in nodules in subcutaneous connective tissues. Adults can live in the nodules for approximately 15 years. Some nodules may contain numerous male and female worms. Females measure 33 to 50 cm in length and 270 to 400 µm in diameter, while males measure 19 to 42 mm by 130 to 210 µm. In the subcutaneous nodules, the female worms are capable of producing microfilariae for approximately 9 years. The microfilariae, measuring 220 to 360 µm by 5 to 9 µm and unshathed, have a life span that may reach 2 years. They are occasionally found in peripheral blood, urine, and sputum but are typically found in the skin and in the lymphatics of connective tissues. A blackfly ingests the microfilariae during a blood meal. After ingestion, the microfilariae migrate from the blackfly's midgut through the hemocoeel to the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the blackfly's proboscis and can infect another human when the fly takes a blood meal. CDC DPDx Parasite Image Library
**Loa loa (eye worm)**

Loasis is limited to the areas of African equatorial rain forest. The incidence in endemic areas varies greatly (8 to 75 percent). The larger, female organisms are 60 mm by 500 micrometers; males are 35mm by 300 micrometers in size (figure 22). The circulating microfilaria are 300 micrometers by 7 micrometers; the infective larvae in the fly are 200 micrometers by 30 micrometers. The life cycle of *Loa loa* (figure 23) is identical to that of onchocerca except that the vector for this worm is the deer fly. The infection results in subcutaneous (Calabar) swelling, measuring 5 to 10 cm in diameter, marked by erythema and angioedema, usually in the extremities. The organism migrates under the skin at a rate of up to an inch every two minutes. Consequently, the swelling appears spontaneously, persists for 4 to 7 days and disappears, and is known as fugitive or Calabar swelling. The worm usually causes no serious problems, except when passing through the orbital conjunctiva or the nose bridge. The diagnosis is based on symptoms, history of deer fly bite and presence of eosinophilia. Recovery of worms from the conjunctiva is confirmatory. Treatment and control are the same as those for onchocerciasis.

**Figure 22**

*Loa loa*, agent of filariasis. Anterior end. CDC/Dr. Lee Moore

*Loa loa*, posterior end. CDC/Dr. Lee Moore

*Loa loa*, agent of filariasis. CDC/Dr. Lee Moore

**Figure 23**

The vector for *Loa loa* filariasis are flies from two species of the genus *Chrysops*, *C. silacea* and *C. dimidiata*. During a blood meal, an infected fly (genus *Chrysops*, day-biting flies) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. The larvae develop into adults that commonly reside in subcutaneous tissue. The female worms measure 40 to 70 mm in length and 0.5 mm in diameter, while the males measure 30 to 34 mm in length and 0.35 to 0.43 mm in diameter. Adults produce microfilariae measuring 250 to 300 µm by 6 to 8 µm, which are sheathed and have diurnal periodicity. Microfilariae have been recovered from spinal fluids, urine, and sputum. During the day they are found in peripheral blood, but during the noncirculation phase, they are found in the lungs. The fly ingests microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and migrate from the fly’s midgut through the hemocoel to the thoracic muscles of the arthropod. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the fly’s proboscis and can infect another human when the fly takes a blood meal. CDC DPDx Parasite Image Library

### Summary

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<th>Treatment</th>
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<td><em>Ascaris lumbricoides</em></td>
<td>Oro-fecal</td>
<td>Abdominal pain, weight loss, distended abdomen</td>
<td>Stool: corticoid oval egg (40-70x35-50 µm)</td>
<td>Mebendazole</td>
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<tr>
<td><strong>Trichinella spiralis</strong></td>
<td>Poorly cooked pork</td>
<td>Depends on worm location and burden: gastroenteritis; edema, muscle pain, spasm; eosinophilia, tachycardia, fever, chill headache, vertigo, delirium, coma, etc.</td>
<td>Medical history, eosinophilia, muscle biopsy, serology</td>
<td>Corticosteroid and Mebendazole</td>
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</tr>
<tr>
<td><strong>Trichuris trichiura</strong></td>
<td>Oro-fecal</td>
<td>Abdominal pain, bloody diarrhea, prolapsed rectum</td>
<td>Stool: lemon-shaped egg (50-55 x 20-25µm)</td>
<td>Mebendazole</td>
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<tr>
<td><strong>Enterobius vermicularis</strong></td>
<td>Oro-fecal</td>
<td>Peri-anal pruritus, rare abdominal pain, nausea vomiting</td>
<td>Stool: embryonated eggs (60x27 µm), flat on one side</td>
<td>Pyrantel pamoate or Mebendazole</td>
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<td><strong>Strongyloides stercoralis</strong></td>
<td>Soil-skin, autoinfection</td>
<td>Itching at infection site, rash due to larval migration, verminous pneumonia, mid-epigastric pain, nausea, vomiting, bloody dysentery, weight loss and anemia</td>
<td>Stool: rhabditiform larvae (250x 20-25µm)</td>
<td>Ivermectin or Thiabendazole</td>
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<tr>
<td><strong>Necator americanus; Ancylostoma duodenale</strong> (Hookworms)</td>
<td>Oro-fecal (egg); skin penetration (larvae)</td>
<td>Maculopapular erythema (ground itch), bronchopneumonitis, epigastic pain, GI hemorrhage, anemia, edema</td>
<td>Stool: oval segmented eggs (60 x 30 20-25µm)</td>
<td>Mebendazole</td>
</tr>
<tr>
<td><strong>Dracunculus medinensis</strong></td>
<td>Oral: cyclops in water</td>
<td>Blistering skin, irritation, inflammation</td>
<td>Physical examination</td>
<td>Mebendazole</td>
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<tr>
<td><strong>Wuchereria bancrofti; W. brugia malayi</strong> (elephantiasis)</td>
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<td>Recurrent fever, lymph-adenitis, splenomegaly, lymphedema, elephantiasis</td>
<td>Medical history, physical examination, microfilaria in blood (night sample)</td>
<td>Mebendazole; Diethylcarbamazine</td>
</tr>
<tr>
<td><strong>Onchocerca volvulus</strong></td>
<td>Black fly bite</td>
<td>Nodular and erythematous dermal lesions, eosinophilia, urticaria, blindness</td>
<td>Medical history, physical examination, microfilaria in nodular aspirate</td>
<td>Mebendazole; Diethylcarbamazine</td>
</tr>
<tr>
<td><strong>Loa loa</strong></td>
<td>Deer fly</td>
<td>As in onchocerciasis</td>
<td>As in onchocerciasis</td>
<td>As above</td>
</tr>
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