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Reading: Medical Microbiology, Murray *et al.* (6th ed.), chapter 83

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Centers for Disease Control (CDC)

TEACHING OBJECTIVES
Epidemiology, morbidity and mortality
Morphology of the organism
Life cycle, hosts and vectors
Disease, symptoms, pathogenesis and site
Diagnosis
Prevention and control

WEB RESOURCES

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CDC

PARASITOLOGY - CHAPTER FOUR

NEMATODES (Round Worms)

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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 americanes* (hookworms)
- *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.

Figure 1
Ascaris Life Cycle
Adult worms (1) live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces (2). Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks (3), depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed (4), the larvae hatch (5), invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs (6). 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 (7). Upon reaching the small intestine, they develop into adult worms (1). 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.
CDC

Ascaris lumbricoides (Large intestinal roundworm)

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 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.

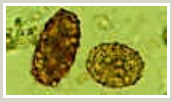
Symptoms
Symptoms are related to the worm burden. Ten to twenty worms may go unnoticed except in a routine stool examination. The commonest complaint is vague abdominal pain. In more severe cases, the patient may experience listlessness, weight loss, anorexia, distended abdomen, intermittent loose stool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

Figure 2


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

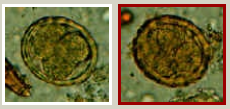

Eggs, unfertilized (left) and fertilized (right). Patient seen in Haiti. CDC DPDx Parasite Image Library


Egg containing a larva, which will be infective if ingested. Patient seen in Léogane, Haiti. CDC DPDx Parasite Image Library


Unfertilized egg. Prominent mamillations of outer layer. Ten-year old boy seen in Cherokee, North Carolina. CDC DPDx Parasite Image Library


Fertilized egg. The embryo can be distinguished inside the egg. Ten-year old boy seen in Cherokee, North Carolina. CDC DPDx Parasite Image Library

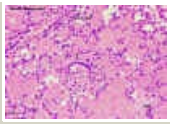

Unfertilized egg with no outer mamillated layer (decorticated). Patient seen during a survey in Bolivia. CDC DPDx Parasite Image Library


Two fertilized eggs from the same patient, where embryos have begun to develop (this happens when the stool sample is not processed for several days without refrigeration). The embryos in early stage of division (4-6 cells) can be clearly seen. Note that the egg on the left has a very thin mamillated outer layer. CDC DPDx Parasite Image Library


Larva hatching from an egg. CDC DPDx Parasite Image Library


An adult Ascaris worm. Diagnostic characteristics: tapered ends; length 15-35 cm (the females tend to be the larger ones). This worm is a female, as evidenced by the size and genital girdle (the dark circular groove at bottom area of image). Worm passed by a female child in Florida. CDC DPDx Parasite Image Library


Ascaris lumbricoides adult male and female © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission



Ascaris lumbricoides larva in section of lung (H&E) © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Figure 3

Trichinellosis is acquired by ingesting meat containing cysts (encysted larvae) 1 of *Trichinella*. After exposure to gastric acid and pepsin, the larvae are released 2 from the cysts and invade the small bowel mucosa where they develop into adult worms 3 (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 4 that migrate to the striated muscles where they encyst 5. *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 these carnivorous animals (or eating food contaminated with such meat). CDC DPDx Parasite Image Library

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.

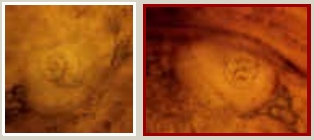
Diagnosis

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

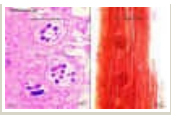
Steroids are use for treatment of inflammatory symptoms and Mebendazole is used to eliminate worms. Elimination of parasite infection in hogs and adequate cooking of meat are the best ways of avoiding infection.

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

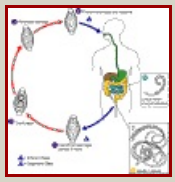


Trichinella spiralis larvae in muscle section (H&E) and muscle press © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Table 1 Trichinosis symptomatology			
Intestinal mucosa (24-72 hrs)	**Circulation and muscle (10-21 days)**	**Myocardium (10-21 days)**	**Brain and meninges (14-28 days)**
Nausea, vomiting diarrhea, abdominal pain, headache.	Edema, peri-orbital conjunctivitis, photo phobia, fever, chill, sweating, muscle pain, spasm, eosinophilia.	Chest pain, tachycardia, EKG changes, edema of extremities, vascular thrombosis.	Headache (supraorbital), vertigo, tinnitus, deafness, mental apathy, delirium, coma, loss of reflexes.

Trichuris trichiura (whipworm)

Figure 5



Life cycle of Trichuris trichiura

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

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 dia,eter) and a thicker (500 micrometers diameter) posterior end. The male is smaller and has a coiled posterior end. The Trichuris eggs are lemon or football shaped and have terminal plugs at both ends.

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.

Figure 6


Egg of Trichuris trichuria as seen on wet mount. The diagnostic characteristics are: a typical barrel shape two polar plugs, that are unstained size: 50-54 µm by 22-23 µm. The external layer of the shell of the egg is yellow-brown (in contrast to the clear polar plugs). The egg is unembryonated, as eggs are when passed with the stool. CDC DPDx Parasite Image Library


Trichuris trichiura adult male and female © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

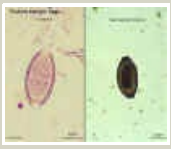
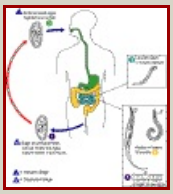

Trichuris trichiura eggs, unstained and haematoxylin stained © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Figure 7


Life cycle of enterobius vermicularis

Eggs are deposited on perianal folds (1). Self-infection occurs by transferring infective eggs to the mouth with hands that have scratched the perianal area (2). Person-to-person transmission can also occur through handling of contaminated clothes or bed linens. Enterobiasis may also be acquired through surfaces in the environment that are contaminated with pinworm eggs (e.g., curtains, carpeting). Some small number of eggs may become airborne and inhaled. These would be swallowed and follow the same development as ingested eggs. Following ingestion of infective eggs, the larvae hatch in the small intestine (3) and the adults establish themselves in the colon (4). The time interval from ingestion of infective eggs to oviposition by the adult females is about one month. The life span of the adults is about two months. Gravid females migrate nocturnally outside the anus and 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

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 Pyrental 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.

Figure 8

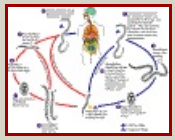
Enterobius vermicularis adult male and female © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

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

A  B  C 

Three eggs of Enterobius vermicularis collected from the same patient on a Swube tube (paddle coated with adhesive material), examined directly on bright field. The diagnostic characteristics are: size 50-60 µm by 20-32 µm; typical elongated shape, with one convex side and one flattened side; colorless shell (here seen as a halo around the egg). The egg in A contains an embryo, while those in B and C contain more differentiated larvae, which are typically coiled. CDC DPDx Parasite Image Library

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) 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

***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 and develop into rhabditiform larvae in man. These larvae can penetrate through the mucosa and cycle back into the blood circulation, lung, glottis and duodenum and jejunum; thus they continue the auto infection cycle. Alternatively, they are passed in the feces, develop into infective filariform larvae and enter another host to complete the direct cycle. If no suitable host is found, the larvae mature into free-living worm and lay eggs in the soil. The eggs hatch in the soil and produce rhabditiform larvae which develop into infective filariform larvae and enter a new host (indirect cycle), or mature into adult worms to repeat the free-living cycle.

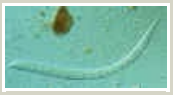
Symptoms
Light infections are asymptomatic. Skin penetration causes itching and red blotches. During migration, the organisms cause bronchial verminous pneumonia and, in the duodenum, they cause a burning mid-epigastric pain and tenderness accompanied by nausea and vomiting. Diarrhea and constipation 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 thiabendozole can be used effectively. Direct and indirect infections are controlled by improved hygiene and auto-infection is controlled by chemotherapy.

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 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 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 rhabditiform larva © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

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.

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 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

Table 2. Clinical features of hookworm disease

Site	Symptoms	Pathogenesis
Dermal	Local erythema, macules, papules (ground itch)	Cutaneous invasion and subcutaneous migration of larva
Pulmonary	Bronchitis, pneumonitis and, sometimes, eosinophilia	Migration of larvae through lung, bronchi, and trachea

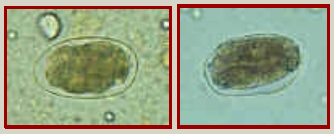
to 2 years, but longevity records can reach several years.
Some *A. duodenale* larvae, following penetration of the host skin, can become dormant (in the intestine or muscle). In addition, infection by *A. duodenale* may probably also occur by the oral and transmammary route. *N. americanus*, however, requires a transpulmonary migration phase. CDC DPDx Parasite Image Library

Gastro-intestinal	Anorexia, epigastric pain and gastro-intestinal hemorrhage	Attachment of adult worms and injury to upper intestinal mucosa
Hematologic	Iron deficiency, anemia, hypoproteinemia, edema, cardiac failure	Intestinal blood loss

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.

Figure 12



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. CDC DPDx Parasite Image Library

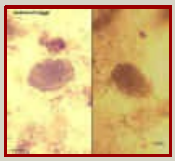
Ancylostoma duodenale adult male and female © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Necator americanus adult female, anterior end © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Necator americanus adult female, anterior and posterior ends © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Hookworm filariform larvae © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Necator americanus adult male, posterior end © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission



Hookworm eggs © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

Dracunculus medinensis (Guinea worm; Fiery serpent)

Dracunculiasis comes from the Latin: *affliction with little dragons*. The common name "Guinea worm" results from the first observation of this parasite by Europeans in the Guinea coast of West Africa in the 17th century. Infection causes a burning, painful sensation leading to the disease being called *the fiery serpent*.

Epidemiology

There have been dramatic efforts to eradicate *Dracunculus*. CDC estimated that in 1986 there were 3.5 million cases worldwide. However, at the end of 2007, there were fewer than 10,000 reported cases in five nations in Africa: Sudan, Ghana, Nigeria, Niger, and Mali, and as of June 2008, cases had been reduced by more than 50 percent compared to the same period of 2007. Guinea worm disease is expected to be the next disease after smallpox to be eradicated and presently there may be as few as 1000 cases worldwide.

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 rhabtidiform 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 13A). 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.

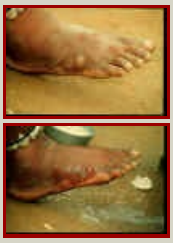
Treatment

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. No drug is effective in killing the worm. Protection of drinking water from being contaminated with Cyclops and larvae are effective preventive measures.

WEB RESOURCES

Drancunculis Guinea Worm - CDC

Figure 13



A, B: The female guinea worm induces a painful blister (A); after rupture of the blister, the worm emerges as a whitish filament (B) in the center of a painful ulcer which is often secondarily infected. (Images contributed by Global 2000/The Carter Center, Atlanta, Georgia). CDC

C *Dracunculus medinensis* worm wound around matchstick.This helminth is gradually withdrawn from the body by winding the stick CDC/Dr. Myron Schultz

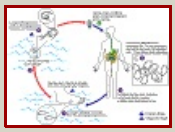


Figure 13A

Humans become infected by drinking unfiltered water containing copepods (small crustaceans) which are infected with larvae of *D. medinensis* 1. Following ingestion, the copepods die and release the larvae, which penetrate the host stomach and intestinal wall and enter the abdominal cavity and retroperitoneal space 2. After maturation into adults and copulation, the male worms die and the females (length: 70 to 120 cm) migrate in the subcutaneous tissues towards the skin surface 3. Approximately one year after infection, the female worm induces a blister on the skin, generally on the distal lower extremity, which ruptures. When this lesion comes into contact with water, a contact that the patient seeks to relieve the local discomfort, the female worm emerges and releases larvae 4. The larvae are ingested by a copepod 5 and after two weeks (and two molts) have developed into infective larvae 6. Ingestion of the copepods closes the cycle 1 CDC DPDx Parasite Image Library

Toxocara canis and *T. catti* (visceral larva migrans)

Figurre 14

Eggs of Toxocara canis. These eggs are passed in dog feces,

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

especially puppies' feces. Humans do not produce or excrete eggs, and therefore eggs are not a diagnostic finding in human toxocariasis! The egg to the left is fertilized but not yet embryonated, while the egg to the right contains a well developed larva. The latter egg would be infective if ingested by a human (frequently, a child). CDC DPDx Parasite Image Library


Toxocara canis (Dog Roundworm) egg, embryonated © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

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).

Figure 15

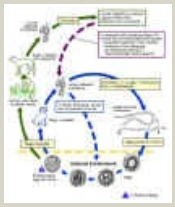

Toxocara Life Cycle
Toxocara canis accomplishes its life cycle in dogs, with humans acquiring the infection as accidental hosts. Following ingestion by dogs, the infective eggs yield larvae that penetrate the gut wall and migrate into various tissues, where they encyst if the dog is older than 5 weeks. In younger dogs, the larvae migrate through the lungs, bronchial tree, and esophagus; adult worms develop and oviposit in the small intestine. In the older dogs, the encysted stages are reactivated during pregnancy, and infect by the transplacental and transmammary routes the puppies, in whose small intestine adult worms become established. Thus, infective eggs are excreted by lactating bitches and puppies. Humans are paratenic hosts who become infected by ingesting infective eggs in contaminated soil. After ingestion, the eggs yield larvae that penetrate the intestinal wall and are carried by the circulation to a wide variety of tissues (liver, heart, lungs, brain, muscle, eyes). While the larvae do not undergo any further development in these sites, they can cause severe local reactions that are the basis of toxocariasis. CDC

Figure 16


Ancylostoma brasiliense adult male and female © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission

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).


A


B
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. CDC DPDx Parasite Image Library

Figure 17


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 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

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 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.


Figure 20E
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

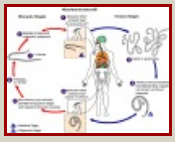


Figure 18A
Different species of the following genera of mosquitoes are vectors of *W. bancrofti* filariasis depending on geographical distribution. Among them are: *Culex* (*C. annulirostris*, *C. bitaeniorhynchus*, *C. quinquefasciatus*, and *C. pipiens*); *Anopheles* (*A. arabinensis*, *A. bancroftii*, *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*); *Mansonia* (*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 ❶. They develop in adults that commonly reside in the lymphatics ❷. 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 ❸. A mosquito ingests the microfilariae during a blood meal ❹. 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 ❺. There the microfilariae develop into first-stage larvae ❻ and subsequently into third-stage infective larvae ❼. The third-stage infective larvae migrate through the hemocoel to the mosquito's prosbocis ❽ and can infect another human when the mosquito takes a blood meal ❶. CDC DPDx Parasite Image Library

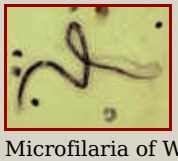
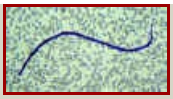


Figure 18B
Microfilaria of Wuchereria bancrofti, from a patient seen in Haiti. Thick blood smears stained with hematoxylin. The microfilaria is sheathed, its body is gently curved, and the tail is tapered to a point. The nuclear column (the cells that constitute the body of themicrofilaria) is loosely 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

 Figure 18C

Microfilaria of Wuchereria bancrofti collected by filtration with a nucleopore membrane. Giemsa stain, which does not 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 19A

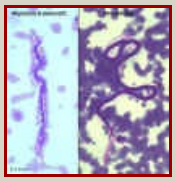
Microfilaria of Brugia malayi. Thick blood smear, hematoxylin stain. Like Wuchereria bancrofti, this species has a sheath (slightly stained in hematoxylin). Differently from Wuchereria, the microfilariae in this species are more tightly coiled, and the nuclear column is more tightly packed, preventing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

 Figure 19B

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

 Figure 19C

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

 Figure 19D

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

 Figure 19E

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 ❶. They develop into adults that commonly reside in the lymphatics ❷. 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 ❸. A mosquito ingests the microfilariae during a blood meal ❹. 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 ❺. There the microfilariae develop into first-stage larvae ❻ and subsequently into third-stage larvae ❼. The third-stage larvae migrate through the hemocoel to the mosquito's prosbocis ❽ and can infect another human when the mosquito takes a blood meal ❶. CDC DPDx Parasite Image Library

 Figure 20A

Scrotal lymphangitis due to filariasis CDC

 Figure 20B

Inguinal lymph nodes enlarged due to filariasis. CDC

 Figure 20C
Histopathology showing cross section of Dirofilaria worm in eye. CDC

 Figure 20D
An elderly village chief undresses prior to bathing. He has elephantiasis of the left leg, large hydrocoele, leopard skin and onchocerciasis nodules, one clearly visible on his torso. WHO/TDR/Crump

 Figure 20 F
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

 Figure 20G
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

 Figure 20H
An elderly male with hydrocoele, elephantiasis of the leg, hanging groin, leopard skin and onchocerciasis nodules. WHO/TDR/Crump

 Figure 20I
An elderly male with hydrocoele, elephantiasis of the leg, hanging groin and leopard skin. WHO/TDR/Crump

 Figure 20J
The feet of a male villager showing elephantiasis and skin lesions of the left leg and foot. WHO/TDR/Crump

 Figure 20K
This lady has elephantiasis of the right leg and oedema in the left. WHO/TDR/Crump

 Figure 20L
Elephantiasis of leg due to filariasis. Luzon, Philippines. CDC

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 migrate to the head of the fly and then are transmitted to a second host.

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
Ivermectin is effective in killing the larvae, but does not affect the adult worm. Preventive measures include vector control, treatment of infected individuals and avoidance of black fly.

Figure 21


Figure 21A
Microfilaria of 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


Figure 21B
Onchocerca volvulus. CDC/Dr. Lee Moore


Figure 21C
Onchocerca volvulus, posterior end. CDC/Dr. Lee Moore


Figure 21D
Face of a blind male patient in the onchocerciasis ward. WHO/TDR/Crump


Figure 21E
Onchocerca volvulus adults in section of tumour (H&E) © Dr Peter Darben, Queensland University of Technology clinical parasitology collection. Used with permission


Figure 21F
Histopathology of Onchocerca volvulus nodule. Onchocerciasis. CDC/Dr. Mae Melvin


Figure 21G

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

 Figure 21H
Life cycle of Onchocerca volvulus.
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 ❶. In subcutaneous tissues the larvae ❷ develop into adult filariae, which commonly reside in nodules in subcutaneous connective tissues ❸. 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 unsheathed, 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 ❹. A blackfly ingests the microfilariae during a blood meal ❺. After ingestion, the microfilariae migrate from the blackfly's midgut through the hemocoel to the thoracic muscles ❻. There the microfilariae develop into first-stage larvae ❼ and subsequently into third-stage infective larvae ❽. The third-stage infective larvae migrate to the blackfly's proboscis ❾ and can infect another human when the fly takes a blood meal ❶. 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 achieved with diethylcarbamazine..

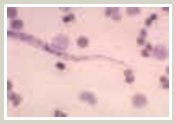
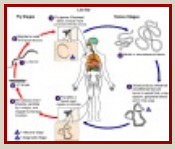
 Figure 22A
Loa loa, posterior end. CDC/Dr. Lee Moore

 Figure 22B
Loa loa, agent of filariasis. Anterior end. CDC/Dr. Lee Moore

 Figure 22C
Microfilariae of Loa loa (right) and Mansonella perstans (left). Patient seen in Cameroon. Thick blood smear stained with hematoxylin. Loa loa is sheathed, with a relatively dense nuclear column; its tail tapers and is frequently coiled, and nuclei extend to the end of the tail. Mansonella perstans is smaller, has no sheath, and has a blunt tail with nuclei extending to the end of the tail. CDC

 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 ❶. The larvae develop into adults that commonly reside in subcutaneous tissue ❷. 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 ❸. The fly ingests microfilariae during a blood meal ❹. After ingestion, the microfilariae lose their sheaths and migrate from the fly's midgut through the hemocoel to the thoracic muscles of the arthropod ❺. There the microfilariae develop into first-stage larvae ❻ and subsequently into third-stage infective larvae ❼. The third-stage infective larvae migrate to the fly's proboscis ❽ and can infect another human when the fly takes a blood meal ❶. CDC DPDx Parasite Image Library

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

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