

# Enteric Nematodes of Humans

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## General Concepts

*Ascaris Lumbricoides*

### Clinical Manifestations

Symptoms correlate with worm load: light loads are asymptomatic; heavier loads cause abdominal symptoms, diarrhea, and sometimes malnutrition. A bolus of worms may obstruct the intestine. Migrating larvae can cause pneumonitis and eosinophilia.

### Structure

*Ascaris lumbricoides* is the largest intestinal nematode of humans. Females are up to 30 cm long; males are smaller. Three types of eggs may appear in feces: fertilized, unfertilized, and decorticated.

### Multiplication and Life Cycle

Adults in the small intestine produce eggs that pass in feces, embryonate in soil, are ingested, and hatch. The larvae migrate from the intestine to the lung and back to the intestine, where they mature.

### Pathogenesis

Migrating larvae cause eosinophilia and sometimes allergic reactions. Erratic adult worms may invade other organs. Heavy infections can impair nutrition.

### Host Defenses

Resistance increases with age; the mechanism is not clear.

### Epidemiology

Egg viability is supported by warm, moist soil. Transmission is favored by unsanitary disposal of feces. Prevalence is highest in children.

## **Diagnosis**

Diagnosis is made most often by identifying eggs in stool; occasionally, erratic adults emerge from body orifices.

## **Control**

Control is by sanitary disposal of feces and by education and treatment.

## **Hookworms**

### **Clinical Manifestations**

Itching may occur where larvae enter skin ("ground itch"). Pneumonitis, cough, dyspnea and hemoptysis may mark the migration of larvae through the lungs. Depending on the adult worm load, intestinal infection can cause anorexia, fever, diarrhea, weight loss, and anemia.

### **Structure**

Two species of hookworms infect humans: *Ancylostoma duodenale* and *Necator americanus*. They are distinguished by the morphology of the mouth parts and male bursa. Females are larger. Eggs are oval, thin-shelled, and transparent. Eggs hatch to release rhabditiform larvae, which mature into filariform (infective stage) larvae.

### **Multiplication and Life Cycle**

Adults attach to the mucosa of the small intestine. Eggs passed in feces embryonate and hatch in soil; mature larvae penetrate the skin and migrate first to the lungs, and then to the intestine, where they mature into the adult stage.

### **Pathogenesis**

Larvae entering skin often cause an erythematous reaction. Larvae in the lung may cause small hemorrhages, eosinophilic infiltration, and pneumonitis. Blood loss from sites of intestinal attachment may cause iron-deficiency anemia.

### **Host Defenses**

Spontaneous self-cure may represent a hypersensitivity reaction. Infection induces high levels of IgE.

### **Epidemiology**

Transmission is favored by poor sanitation and warm moist soil. Prevalence rises with age.

## **Diagnosis**

Diagnosis is by detection of eggs and (sometimes) larvae in stool. Low levels of hemoglobin are suggestive.

### **Control**

Control is by sanitary disposal of feces and by education and treatment.

## ***Strongyloides Stercoralis***

### **Clinical Manifestations**

Ground itch may occur where larvae penetrate the skin. Pneumonitis, epigastric pain, mucous diarrhea, and eosinophilia may occur. In immunocompromised individuals, worms may disseminate to other organs.

### **Structure**

Males are free-living; females may be free-living or parasitic. Eggs develop into rhabditiform and then filariform (infectious) larvae.

### **Multiplication and Life Cycle**

Parasitic females parthenogenetically produce embryonated eggs, which hatch in the intestine. Rhabditiform larvae pass in the feces, mature to the infective filariform stage in soil, penetrate the skin, and migrate to the lungs and other organs, then the intestine. Autoinfection also occurs. Free-living worms reproduce sexually in soil.

### **Pathogenesis**

Worms cause inflammation and ulceration of the intestines. Migrating larvae cause cutaneous pruritus and pneumonitis. Hyperinfection causes sloughing of mucosa, and disseminated infection occasionally leads to pulmonary hemorrhage, pneumonia, or meningitis and death.

### **Host Defenses**

Immunity is not well understood. Infection induces elevated IgE and eosinophilia. Impairment of cell-mediated immunity favors disseminated disease and autoinfection.

### **Epidemiology**

Prevalence is usually low; the infection is more common in tropical countries with poor sanitation, especially Southeast Asia and parts of Africa. Dogs occasionally serve as a reservoir.

### **Diagnosis**

Epigastric pain, eosinophilia, and mucous diarrhea are suggestive; diagnosis is confirmed by detecting rhabditiform larvae in feces, duodenal aspirates, or sputum. Fecal cultures and serology may be helpful.

## **Control**

Control is by sanitary disposal of feces and by education and treatment.

### ***Trichuris Trichura***

## **Clinical Manifestations**

Diarrhea, anemia, weight loss, abdominal pain, nausea, vomiting, eosinophilia, tenesmus, rectal prolapse, stunted growth and finger clubbing may occur.

## **Structure**

Adults are whip-shaped, slender anteriorly and broader posteriorly. Males are shorter than females and have a coiled posterior. The unembryonated eggs are barrel-shaped with bipolar plugs.

## **Multiplication and Life Cycle**

Adults in the large intestine lay eggs which pass in feces and embryonate in soil. Eggs that are ingested hatch and larvae mature to adults in the gut.

## **Pathogenesis**

Adults prefer the cecum but will also colonize the large intestine. Worms cause mucosal inflammation, eosinophilic infiltration, and minor blood loss; heavy infections may lead to anemia and nutritional deficiency.

## **Host Defenses**

Defenses are little understood; resistance does not increase with age.

## **Epidemiology**

Transmission is favored by poor sanitation and warm soil.

## **Diagnosis**

Diagnosis is by detection of eggs in feces.

## **Control**

Control is by sanitary disposal of feces and by education and treatment.

### ***Entrobious Vermicularis***

## **Clinical Manifestations**

Enterobiasis is most common in children, who usually manifest pruritus ani and sometimes insomnia, abdominal pain, anorexia, and pallor. Genitourinary infection may occur in females.

### **Structure**

Worms are white and spindle-shaped with a large, bulbar esophagus. Males are smaller and have a curved posterior. Eggs are ovoid, thin-shelled, and flat on one side.

### **Multiplication and Life Cycle**

Females usually migrate out the anus at night and deposit eggs on the perianal skin. The eggs embryonate quickly and, if ingested, hatch and mature in the intestines.

### **Pathogenesis**

Intestinal lesions are rare; extraintestinal infection may lead to complications.

### **Host Defenses**

The defenses are little known. Most infections occur in children.

### **Epidemiology**

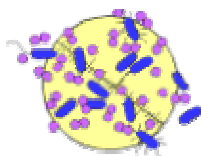
*Enterobius vermicularis* is the most common helminth in the United States. Household and institutional epidemics occur, usually in children. Transmission is usually by hand to mouth transfer of infective eggs.

### **Diagnosis**

Eggs are rare in feces but are readily collected by Scotch-tape perianal swabs.

### **Control**

Control is by anthelmintic treatment and by improved personal hygiene, including washing the perianal region and changing nightclothes.



## **INTRODUCTION**

Enteric nematodes are among the most common and widely distributed animal parasites of humans. In his classic address to the American Society of Parasitologists in 1946, entitled "This Wormy World," Stoll estimated 2.3 billion helminthic infections in a human population of 2.2 billion. Since 1946, the world population has doubled and, by all indications, enteric nematode infections of humans have kept pace. The most

common intestinal roundworms are those transmitted through contact with the soil (for example *Ascaris lumbricoides*, *Trichuris trichiura*, the hookworms, and *Strongyloides stercoralis*). In Stoll's estimate, these worms, with *Enterobius vermicularis*, accounted for three-quarters of all helminthic infections.

Most enteric nematodes have established a well-balanced host-parasite relationship with the human host; humans tolerate these parasites well. Little disease is associated with light infection, but when the worm load increases, a corresponding increase in disease usually occurs. The worms may irritate the intestinal mucosa, causing inflammation and ulceration. Some produce "toxic" substances. The larger worms may become entangled and block the intestinal tract. Larval worms that migrate through the tissue to complete their life cycle may lose their way, end up in the wrong organ, and cause severe disease. Nutritional problems occasionally are associated with the intestinal parasitosis, and persons with deficient diets often suffer from polyparasitism.

Diagnosis usually is based on microscopic examination of feces for eggs and larvae, except in the case of pinworm infections, which are diagnosed by examining samples taken with perianal swab. Many antihelmintics are available to treat patients with these infections. Control depends largely on proper disposal of human feces and on personal hygiene.

The enteric nematodes discussed in this chapter are *A lumbricoides*; the hookworms *N americanus* and *A duodenale*; *S stercoralis*; *T trichiura*; and *E vermicularis*.

## ***Ascaris Lumbricoides***

### **Clinical Manifestations**

Adult *A lumbricoides* infections involving only a few worms are usually asymptomatic, but as the worm load increases, symptoms of abdominal discomfort, nausea, vomiting, weight loss, fever, and diarrhea develop. Allergic manifestations in hypersensitized persons lead to pneumonitis, cough, low-grade fever, and eosinophilia. Large numbers of worms may form a bolus and cause intestinal obstruction. Stimulation causes adult worms to become erratic and invade the appendix and biliary and pancreatic ducts. Worms may enter and block small orifices. Migrating adults have been vomited and passed from the nose and mouth, anus, umbilicus, and lacrimal glands. They can perforate the intestines and enter the peritoneal cavity, the respiratory tract, urethra, and vagina, and even the placenta and fetus. Excessive worm loads, especially among the malnourished, can lead to nutritional impairment because the worms interfere with the absorption of proteins, fats, and carbohydrates.

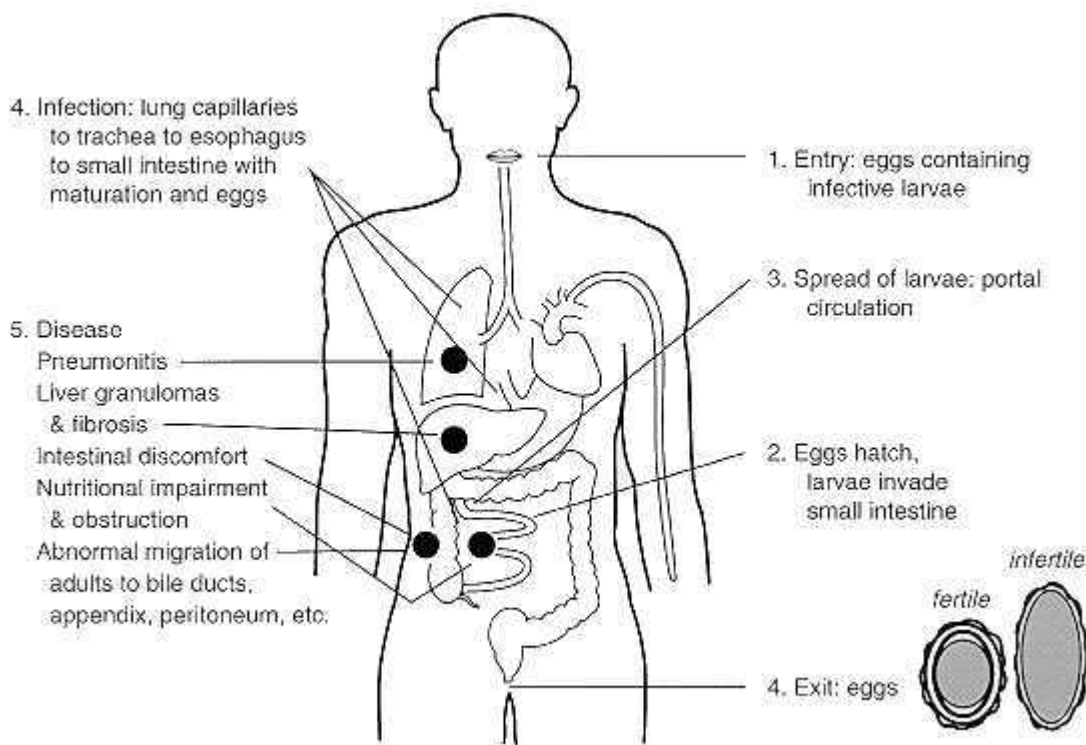
### **Structure**

*Ascaris lumbricoides* is the largest and most common intestinal nematode of humans. Females are approximately 30 cm long; sexually mature males are smaller. The diameter varies from 2 to 6 mm. Mated females produce fertile eggs that are oval to subspherical, 45 to 75  $\mu\text{m}$  by 35 to 50  $\mu\text{m}$ , and are covered by a thick shell with a light brown, mammillated, albuminous outer coat. Unmated females (for example, in a single-sex infection), produce unfertilized eggs that are thin-shelled, ellipsoidal, and measure 78 to 105  $\mu\text{m}$  by 38 to 55  $\mu\text{m}$ . The mammillated coat of unfertilized eggs is

irregular and the contents are granular and disorganized. Some eggs are passed without the outer mammillated coat (decorticated eggs) and can be confused with eggs from hookworms or other worms.

### Multiplication and Life Cycle

*Ascaris lumbricoides* is found in the small intestine, particularly the jejunum. Females produce as many as 240,000 eggs per day and as many as 65 million in a lifetime. The eggs are unsegmented and are passed in the feces. In moist, warm, shady soil, the eggs embryonate, and an infective larva develops within the egg in about 3 weeks. After ingestion by a human, the eggs pass to the duodenum where they hatch; the released larvae penetrate the intestinal mucosa, enter the lymphatics and portal system, and are carried to the liver, heart, and lungs. This migratory phase requires a few days. The larvae then break out of the capillaries into the alveoli, pass up the respiratory tree, and are swallowed. They reach the intestines and continue their development, and 8 to 12 weeks after infection, become sexually mature adults. The adults live for about a year and are subsequently passed in the feces (Fig. 90-1).



**FIGURE 90-1** Life cycle of *Ascaris lumbricoides*.

### Pathogenesis

The initial pathology is associated with migrating larvae; the severity depends upon the number of invading organisms, the sensitivity of the host, and the host's nutritional status. Persons repeatedly infected become sensitized, and migrating larvae may cause tissue reactions in the liver and lungs, with eosinophilic infiltration and granuloma formation. The reactions lead to pneumonitis and a condition known as Loeffler's

syndrome. Adult worms may cause blockage of the intestines, and migrating adults may provoke severe pathology when they wander into other organs. Acute pancreatitis and biliary stones may occur. The rare fatalities usually result from intestinal obstruction or biliary ascariasis. Furthermore, the pathogenicity of the worms may vary in different regions of the world.

### **Host Defenses**

The fact that children are more often infected with *A lumbricoides* than adults suggests that resistance develops with age. The mechanisms underlying this resistance are not known. IgE antibodies are present in infected persons, and some persons can develop allergic manifestations such as urticaria, asthma, fever, conjunctivitis, and eosinophilia. Some parasitologists become sensitized and subsequently develop severe reactions when exposed to *A lumbricoides* antigens.

### **Epidemiology**

*Ascaris lumbricoides* is distributed widely in tropical and subtropical areas, especially in the developing countries of South America, Africa, and Asia. More than one billion infections are estimated to exist at any given time. In rural areas of Asia, it is not unusual to find 85 percent of the population passing *Ascaris* eggs. Prevalence rates are much lower in the United States. Some people appear to be predisposed to infection with intestinal helminths, including *A lumbricoides*. Some individuals are found to be constantly infected and usually have a higher intensity of infections than others.

### **Diagnosis**

Symptomatic ascariasis is rarely diagnosed on clinical grounds alone because the pneumonitis, eosinophilia, and intestinal symptoms are similar to those caused by other infectious agents. Infections before the appearance of eggs in the feces, infections with only male worms, and extraintestinal infections are difficult to diagnose. Radiologic computed tomography (CT) and sonographic examination may reveal adult worms in the intestine and bile ducts, but definitive diagnosis requires finding characteristic eggs in feces. Eggs are usually so numerous in any infection involving female worms that simple microscopic examination of a fecal smear is all that is necessary. Concentration techniques involving flotation or sedimentation of eggs also may be used. Techniques are available to estimate the intensity of an infection on the basis of the number of eggs in a measured stool sample.

### **Control**

The most effective method to control ascariasis, as well as other soil-transmitted helminthiasis, is sanitary disposal of feces. In some areas, this requires changing centuries-old habits and educating the population. Mass treatment programs have been initiated in many parts of the world and, in some Asian countries, efforts are being made to deworm all school children. In a pilot program in the Philippines aimed at eradication of the soil-transmitted helminths by periodic mass treatment of a barrio population, the prevalence of ascariasis decreased from 78 percent to less than 1 percent over 3 years. Mebendazole, the drug used, is effective against numerous intestinal nematode infections and causes few side effects. Levamisole is also useful, as are pyrantel



pamoate, piperazine citrate, thiabendazole and albendazole. Care must be taken in treating mixed helminthic infections involving *A lumbricoides*, because an ineffective ascaricide may stimulate the parasite to migrate to another location. Persons in whom asymptomatic ascariasis is detected incidentally should be treated to prevent the possibility of a future abnormal migration of these large worms into extraintestinal sites.

## Hookworms

### Clinical Manifestations

"Ground itch" (itching at the site of invasion by the larvae), pneumonitis, cough, dyspnea, and, occasionally, hemoptysis are early symptoms of hookworm infection. Symptoms associated with the intestinal phase of infection include anorexia or a huge appetite with pica (desire to eat unusual substances, such as dirt), fever, diarrhea, abdominal discomfort, weight loss, nausea and vomiting, spleen and liver enlargement, and edema. Children may suffer from mental, physical, and sexual retardation. Eosinophilia is usually marked. Hemoglobin levels as low as 2 percent are common in some endemic areas. Even where hookworm infection is widespread, not all infections lead to hookworm disease.

### Structure

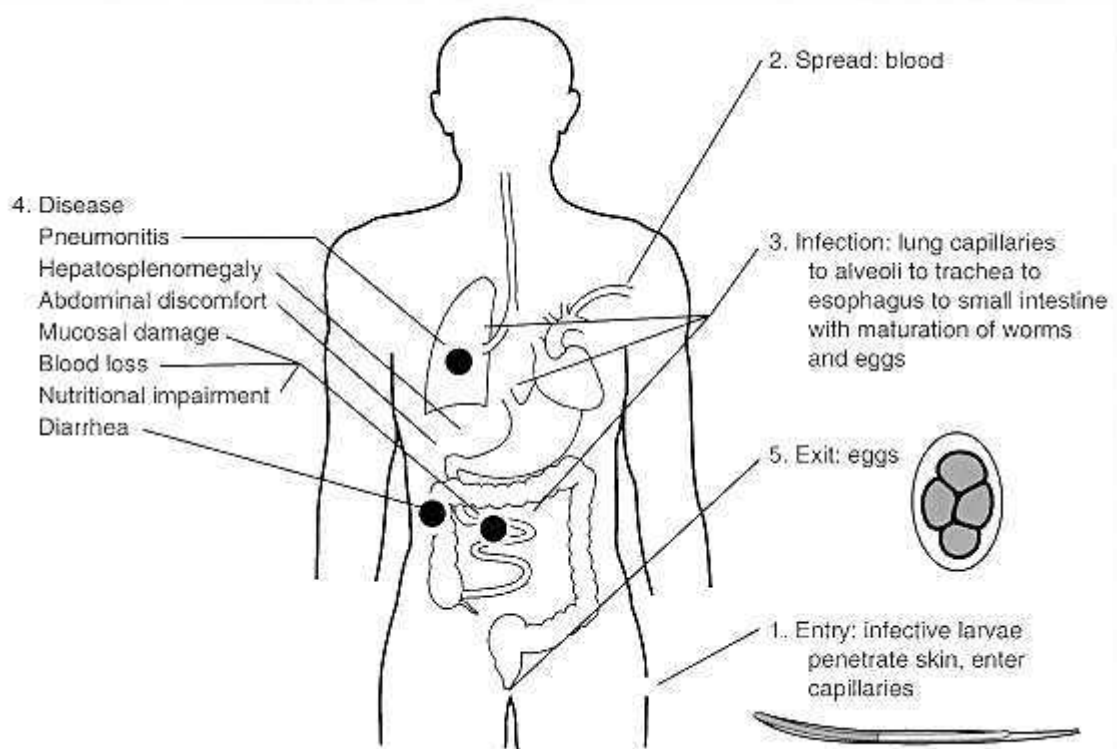
Two major hookworm species infect humans: the Old World hookworm *Ancylostoma duodenale* and the New World hookworm *Necator americanus*. The worms are cylindrical and grayish white. Females are approximately 1 cm long; males are smaller. The major differentiating characteristics between the two species involve the buccal capsule and the male bursa. The bell-shaped bursa, used for attachment to the female during copulation, is membranous and symmetrical and has finger-like rays that are arranged differently in each species. The most prominent difference in the buccal capsules is that *N americanus* has two ventral semilunar cutting plates, whereas *A duodenale* has four ventral teeth. Female *A duodenale* hookworms produce 10,000 to 20,000 eggs per day, compared to 5,000 to 10,000 for *N americanus*. The eggs of both species are ovoid, thin-shelled, and transparent. Eggs in fresh stools contain embryos in the four- or eight-cell stage. The eggs from these species are indistinguishable from each other and measure 55 to 79  $\mu$ m by 35 to 47  $\mu$ m. The first-stage (rhabditiform) larva develops within the egg and has a thick-walled, long, narrow buccal cavity. The muscular esophagus is flask-shaped and occupies the anterior one-third of the body. Slender third-stage (filariform) larvae are 500 to 700  $\mu$ m long. The mouth is closed, and the elongate esophagus occupies one-third of the body.

The tail is sharply pointed. The rhabditiform larvae of the two species cannot be differentiated, but the filariform larvae of *N americanus* have dark, prominent buccal spears and a striated cuticle seen more clearly at the posterior end; these characteristics are not seen in *A duodenale*.

### Multiplication and Life Cycles

Adult hookworms generally attach to the jejunal mucosa, and the females deposit eggs that pass in the feces. In the proper soil, under ideal conditions, the eggs hatch in 1 to 2 days. The rhabditiform larvae that emerge feed on bacteria and organic debris, molt

twice, and develop into slender, infective filariform larvae in 5 to 8 days. These larvae do not feed; if they are unable to penetrate a host, they die in a few weeks. Once in the skin, they enter the venules and are carried to the heart and lungs where they grow and eventually break out into the alveoli and pass up the respiratory tree. After they are swallowed, they attach to the intestinal mucosa and become sexually mature in 5 to 6 weeks (Fig. 90-2). Although infections are known to persist for as long as 14 years, most terminate in 2 to 6 years. Infection by ingestion of larvae may also occur.



**FIGURE 90-2 Life cycle of hookworms.**

### Pathogenesis

Hookworm larvae usually gain access to the body by penetrating the skin. A local reaction called ground itch may occur at the invasion site (usually the feet or hands), particularly in sensitized individuals. Secondary bacterial infection may also occur at these sites. Large numbers of larvae migrating through the lungs at the same time may cause pneumonitis. In the small intestine, worms attach to the mucosa by the buccal capsule. As the worms feed on the mucosa they cause a considerable amount of blood loss. The worm ingests mucosal tissue with blood; much of the blood is then excreted into the lumen of the host's intestine. Blood also is lost by seepage around the attachment site. When the worm changes attachment sites, the wound oozes blood for several days. One *A duodenale* is estimated to be responsible for the loss of 0.15 to 0.26 ml blood per day, and one *N americanus* for the loss of 0.03 ml per day. An anti-coagulant secreted in the buccal capsule of these worms also contributes to blood loss. Excessive blood loss can lead to iron deficiency anemia. Infections involving a few worms usually are asymptomatic, but in heavy infections, hookworm disease with hypoproteinemia, hepatosplenomegaly, and hypochromic microcytic anemia results,

especially if the hookworm infection is accompanied by other infections and by malnutrition. Infections with *A duodenale* are considered more pathogenic than *N americanus* infections.

### **Host Defenses**

Immunity to hookworm develops in dogs, but no strong evidence suggests that it occurs in humans. Ground itch is thought to be an allergic reaction, but the response is minimal unless accompanied by bacterial infection.

Seasonal fluctuation in hookworm egg production in *A duodenale* has been reported and is thought to be due to host resistance, but may also reflect arrested development of the parasite. In some persons, a self-cure or a spontaneous reduction of worms occurs and may be attributed to immediate hypersensitivity reactions in the intestinal wall. IgE antibody (high in persons with hookworm), mast cells, and worm allergens are thought to provide the necessary ingredients for the reaction.

### **Epidemiology**

Close to one billion infections are estimated to exist at any time. Surveys conducted in Asia often find prevalences as high as 70 percent. Infections are found equally in males and females, with the lowest prevalence rates in children.

The incidence of hookworm infection has decreased dramatically in the southern United States, where it was once highly endemic.

Both *N americanus* and *A duodenale* are endemic in warm, moist tropical areas where people defecate in the soil. Places where individuals defecate collectively, such as homes and schools without outhouses and underground mines such as coal mines, are frequent sites of infection. *Necator americanus* is not confined to the Americas, being the most common species in Asia, Central and South Africa, and Central and South America. *Ancylostoma duodenale* is found in these areas to a lesser degree, but is more prominent in India, China, the Soviet Union, and North Africa. Surveys using Harada-Mori cultures in the Philippines showed 30 percent of the stools positive for *N americanus*, 5 percent for *A duodenale*, and less than 1 percent positive for both. Some individuals have a predisposition to hookworm infection..

Temperatures between 25 C and 35 C and a shady, sandy, or loamy soil with vegetation favor larval development. A population that does not wear shoes also facilitates spread of the parasite.

### **Diagnosis**

Hookworm infection is difficult to differentiate clinically from other parasitic infections and certain other diseases. Diagnosis is made by demonstrating eggs in stool specimens. The two species cannot be distinguished on the basis of their eggs. Direct microscopic examination of the stools may suffice in heavy infections, but a concentration method should be used in most cases (e.g., zinc sulfate flotation or formalin-ethylacetate concentration). An estimate of the intensity of infection can be made on the basis of the number of eggs in a measured fecal sample. Specimens should be examined promptly

since rhabditiform larvae may develop and hatch from the egg within a few hours. When larvae are present in the feces, they must be differentiated from those of other nematode species.

## **Control**

Hookworm can be controlled in a population by sanitary disposal of feces, treatment of infected persons, wearing of shoes, health education, and improved nutrition. In the Philippines, the prevalence rate was decreased from 33 percent to less than 1 percent in 3 years by a control program of mass treatment with mebendazole.

Mebendazole is an effective treatment. Also effective are albendazole, thiabendazole, pyrantel pamoate, and levamisole. Anthelmintic treatment should be supplemented with an improved diet, including administration of iron.

## ***Strongyloides Stercoralis***

### **Clinical Manifestations**

Most infections with *S stercoralis* are asymptomatic except for the ground itch that may occur when infective larvae from the soil penetrate the skin in large numbers.

Pneumonitis can result from larval invasion in the lung. Intestinal invasion may lead to epigastric pain and mucous diarrhea. Eosinophilia is common. Dissemination of strongyloidiasis into extraintestinal organs sometimes occurs in persons receiving immunosuppressive drugs. The infection can be perpetuated by an autoinfection cycle, which can lead to massive infection, especially in the immunocompromised host. Linear skin lesions on the lower abdomen and buttocks may also develop in patients with autoinfection due to penetration of the perianal skin by infective larvae. This condition is called *larva currens* (see discussion of the similar conditions called larva migrans in Ch. 91).

### **Structure**

*Strongyloides stercoralis* is unique in that adults may be either parasitic or free-living. Parasitic adults are exclusively female (approximately 2 mm by 40 to 50  $\mu\text{m}$ ); there are no parasitic males. Very thin-shelled, embryonated eggs (55 to 60  $\mu\text{m}$  by 28 to 32  $\mu\text{m}$ ) release rhabditiform larvae, which develop into more slender infective-stage filariform larvae.

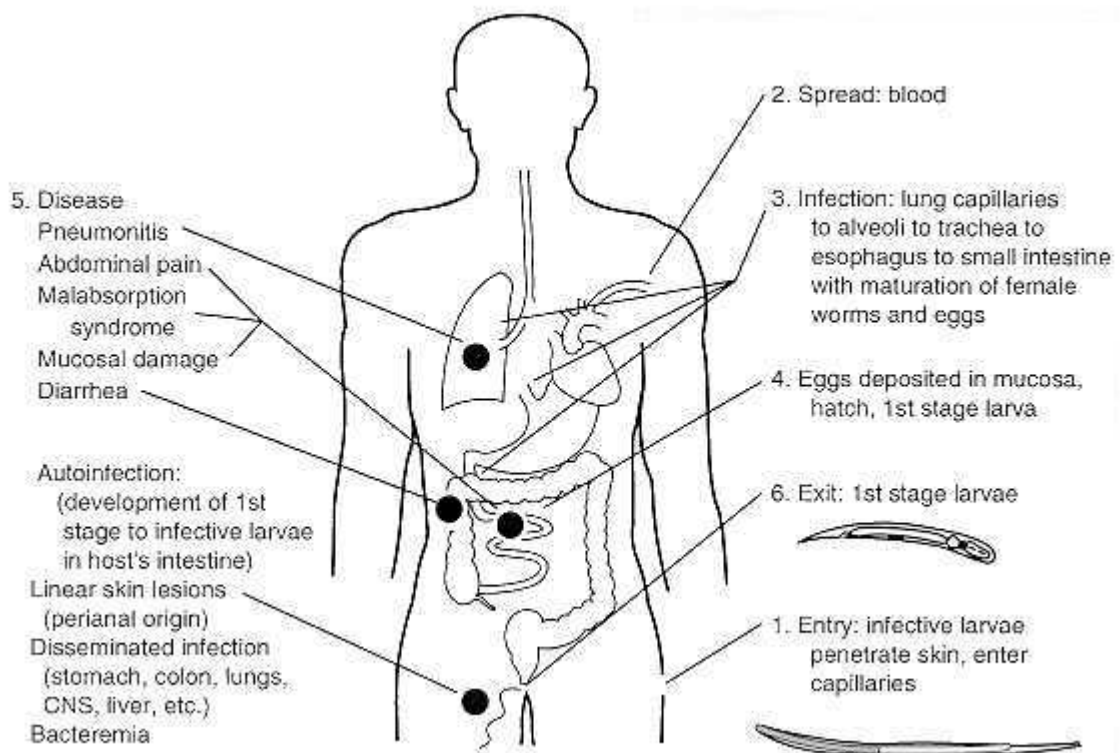
Free-living adult males (650 to 950  $\mu\text{m}$  long) and females (0.8 to 1.6  $\mu\text{m}$  long) live in the soil and reproduce sexually.

### **Multiplication and Life Cycle**

Parasitic females are found in the epithelium of the duodenum or upper jejunum and reproduce by parthenogenesis. Embryonated eggs are laid in the mucosa. The eggs mature rapidly and hatch in the mucosa. First-stage rhabditiform larvae pass in the feces and develop in the soil into infective stage filariform larvae. These penetrate the skin of humans, enter cutaneous blood vessels, migrate throughout the body, especially to the lungs, and finally mature in the small intestines. Females begin to lay eggs about 1

month after infecting the host. In the indirect life cycle, larvae passed in the feces develop in the soil into free-living adult males and females which mate. The eggs that are laid hatch and give rise to a generation of infective larvae, which can penetrate the skin and develop as in the direct life cycle.

In autoinfection, first-stage larvae transform into infective larvae while they are in the intestine or on the skin of the perianal region; these larvae penetrate the wall of the intestine or the perianal skin. Some eventually develop into adults in the small intestine after a migration. Thus, autoinfection can replace or increase the patient's intestinal worm burden and accounts for the persistence of this infection in patients who no longer live in endemic areas (Fig. 90-3).



**FIGURE 90-3** Life cycle of *Strongyloides stercoralis*

### Pathogenesis

Light infections elicit only a mild inflammatory response, whereas in heavy infections, damage to the intestines may be severe, with edema, inflammation, ulceration, increased secretion of mucus and sloughing of the mucosa, as well as functional changes of the gut. A malabsorption syndrome has been reported. In disseminated strongyloidiasis the parasite may be found in any part of the body. In pulmonary infections there may be pneumonia and hemorrhage. Meningitis is also reported. Hyper-disseminated infections may be fatal.

### Host Defenses

Immunity in strongyloidiasis is not well understood but autoinfection generally occurs in persons with suppressed cell-mediated immunity. Most susceptible are patients who have lymphocytic leukemia, malignancy, malnutrition, leprosy, or systemic lupus erythematosus and who are receiving immunosuppressive therapy. Serum IgE levels usually are elevated in persons with this parasite. In severe strongyloidiasis, some patients may have significantly decreased IgG levels and low levels of IgA and IgM. It has also been suggested that human lymphotropic virus type 1 (HTLV-1) has a association with strongyloidiasis and that the mechanism may involve the suppression of the IgE response. Eosinophil counts also may be depressed in patients with massive infections. These findings indicate that eosinophils and antibodies may be important in the defense against *S stercoralis* larvae.

## **Epidemiology**

*Strongyloides stercoralis* may coexist with hookworms; both require similar soil and climatic conditions for development. Warm, moist soils that foster reproduction by the free-living stages may become heavily contaminated with *S stercoralis*. Because of autoinfection, persons who have contracted this infection in endemic areas may remain infected for years after leaving such areas.

Strongyloidiasis is most common in tropical and subtropical areas, but is much less prevalent than hookworm infection. Recent surveys in the Philippines and Indonesia rarely found the parasite, even with the use of filter paper cultures. Other parts of Southeast Asia, however, have a higher prevalence of infection, and parts of Africa report prevalence as high as 21 percent. In the United States, infections are more common in the South and in institutionalized populations.

Dogs are sometimes infected with *S stercoralis*. Although dogs are considered a source of human infections, the primary source continues to be humans.

Cases have been reported in which the infection was transferred to a new host along with a kidney transplant. These patients were immunosuppressed.

## **Diagnosis**

Eosinophilia, epigastric pain, and mucous diarrhea suggest *S stercoralis* infection, but definitive diagnosis requires finding larvae in the stool or, on rare occasions, in sputum or urine. Eggs are not found except in cases of severe dysentery. Direct smear or concentration methods of stool examination usually suffice, but the sample can be cultured in cases where infection is suspected but unconfirmed. Baermanization of charcoal fecal cultures is recommended. (A Baermann apparatus is a funnel with a rubber tube with a pinch-clamp attached to the spout. A sieve is placed in the funnel, gauze is added, and a culture placed on the gauze. Warm water is added to the funnel just above the culture. Larvae will migrate into the water and fall to the bottom. After a few hours, the pinch-clamp is opened and the larvae are flushed out into a flask and examined microscopically.) A newly described culture method using agar plates has been reported to be successful in detecting the parasite. Larval stages of *S stercoralis* must be distinguished from hookworm larvae. The rhabditiform larvae resemble those of hookworms but can be distinguished by the shorter buccal capsule and larger genital

primordium. The filariform larvae also resemble those of hookworms, but the tail is notched and the esophagus is about one-half the length of the body.

Duodenal intubation and examination of aspirates, or a string test (Enterotest) and examination of intestinal mucus, is recommended in suspected cases, even when serial stool examinations are negative. A number of reliable serology tests are also available to aid in the diagnosis of strongyloidiasis.

### **Control**

Like other soil-transmitted nematode infections, strongyloidiasis can be controlled by improving sanitary conditions and by proper disposal of feces. Patients with this infection should be treated even if they are asymptomatic to preclude possible onset of autoinfection. Immunosuppressants are contraindicated in these patients. Strongyloidiasis must be ruled out in persons to be given immuno-suppressants, especially those with eosinophilia.

Thiabendazole, the most effective therapeutic agent, can cause side effects of vertigo, nausea and vomiting. Prolonged or repeated treatment may be required in patients receiving immunosuppressive drugs. Ivermectin and albendazole have recently been reported to also be effective.

### ***Trichuris Trichura***

#### **Clinical Manifestations**

The parasite *T trichiura* lives primarily in the cecum and appendix but can also be found in large numbers in the colon and rectum. Light infections are asymptomatic but heavy infections may cause diarrhea, at times containing mucus and blood. Anemia may develop, along with weight loss, abdominal pain, nausea, vomiting, tenesmus, and rectal prolapse. Nutritional changes can cause stunted growth and clubbing of fingers. Eosinophilia may also develop in response to worms embedded in the mucosa.

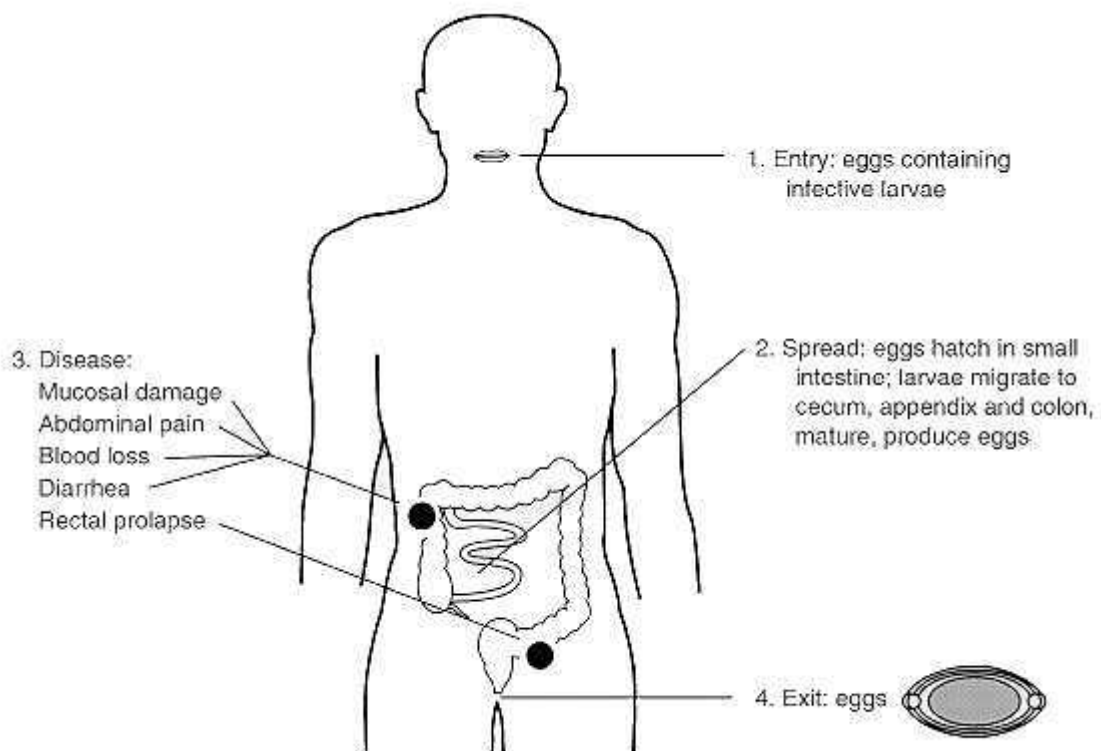
#### **Structure**

*Trichuris trichiura* is known as whipworm because the long, narrow anterior end and the shorter, more robust posterior end give the worm the look of a whip. The pinkish-white worms are threaded through the mucosa and attach by their anterior end. Females (approximately 45 mm long) are larger than males; they are bluntly rounded posteriorly, whereas the males have a coiled posterior. The characteristic eggs are brown and barrel-shaped with prominent bipolar blister-like protuberances; they measure 22 µm by 52 µm.

#### **Multiplication and Life Cycle**

Females produce 2,000 to 10,000 single-celled eggs per day. These pass in the feces and embryonate in the soil. Under favorable conditions, they become infective in about 3 weeks. After being ingested, embryonated infective eggs hatch in the small intestine. The infective larvae penetrate the villi and continue to develop. Young worms move to

the cecum, penetrate the mucosa, and complete development. Females begin to lay eggs about 3 months after infecting the host (Fig. 90-4).



**FIGURE 90-4** Life cycle of *Trichuris trichiura*.

### Pathogenesis

Petechial hemorrhage, edema, inflammation, and mucosal bleeding develop, and heavy infections can cause rectal prolapse. Small amounts of blood (0.005 ml per worm) are lost each day by seepage at the attachment site. Colitis/proctitis, anemia, clubbing of fingers, and growth retardation are also reported to be associated with heavy *T trichiura* infections.

### Host Defenses

Little information is available on resistance and immune responses to *T trichiura* infection. In some endemic areas, the equal occurrence of infection in all age groups suggests that resistance does not develop with age.

### Epidemiology

Whipworm infections are prevalent in tropical and subtropical countries with moist, shaded, warm soil. Up to 800 million infections are estimated to exist. In Asia, some surveys indicate that *T trichiura* is more common than *A lumbricoides*.



No reservoir hosts for *T trichiura* are known to exist. Most infections are acquired by eating infective eggs in contaminated soil, foods, or drink. A single infection may last for several years.

### **Diagnosis**

The symptoms of trichuriasis are nonspecific, but the infection is readily diagnosed by identifying eggs in the feces. In heavy infections, the stools are frequently mucoid and contain Charcot-Leyden crystals. Concentration methods are required for diagnosis in light infections. In heavy infections, the parasite can be seen in the rectal mucosa by sigmoidoscopy.

### **Control**

Sanitary disposal of feces is the best control measure. Mass treatment of populations with mebendazole has shown promise in the Philippines, where administration of the drug periodically over 3 years has reduced the prevalence rate from 88 percent to 2 percent. Mebendazole is presently the drug of choice for treating trichuriasis. Oxantel is also known to be effective.

## ***Enterobius Vermicularis***

### **Clinical Manifestations**

Enterobiasis, or pinworm infection, usually causes little disease. The most common symptom is pruritus ani, which disturbs sleep and which, in children, may be responsible for loss of appetite. abdominal pain, irritability, and pallor may also be signs of enterobiasis. The parasite has been suspected as a cause of appendicitis, and gravid female worms have been known to migrate up the vagina and fallopian tubes and into the peritoneal cavity, where they become encapsulated with granulomatous tissue. Recurrent urinary tract infections have been attributed to ectopic pinworm infections.

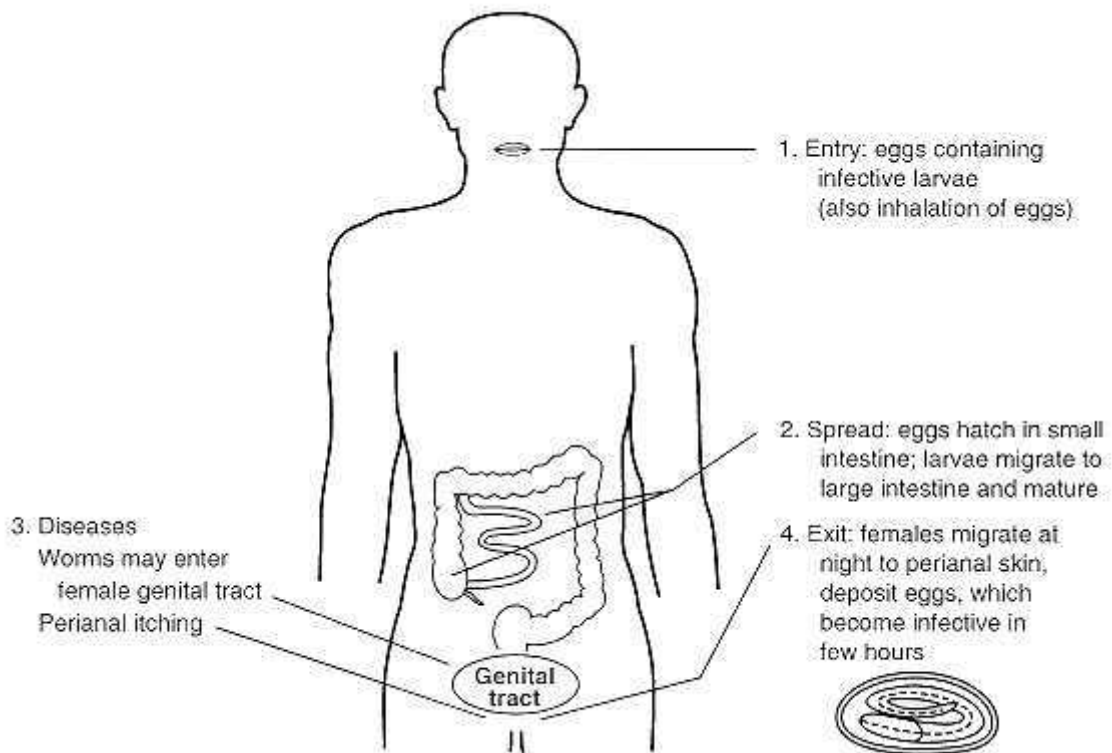
### **Structure**

The whitish, spindle-shaped worms have characteristic cephalic swellings (alae) and a large muscular esophagus with a large posterior bulb. Females are approximately 1 cm long and males are half that size. The curved posterior end of male worms has a single copulatory spicule. The males are rarely seen because they die shortly after copulation and are expelled. The eggs are thin-shelled, ovoid, flattened on one side, and measure 50 to 60  $\mu\text{m}$  by 20 to 30  $\mu\text{m}$ .

### **Multiplication and Life Cycle**

The parasites mature in the large intestine. When gravid, female worms migrate out of the anus at night when the anal sphincter is relaxed and lay eggs that adhere to the perianal skin. The female essentially ruptures, releasing as many as 10,000 eggs. The eggs embryonate and become infective within a few hours after being deposited onto the skin. Infection is transmitted hand-to-mouth. The ingested eggs hatch in the small intestine, each releasing an infective stage larva. The parasite moves to the cecum and

matures into an adult 2 to 4 weeks after infecting the host (Fig. 90-5). Infections are self-limited; reinfection can occur.



**FIGURE 90-5** Life cycle of *Enterobius vermicularis*.

### Pathogenesis

Intestinal lesions are reported, but the worms usually cause little intestinal pathology. The parasite has been found in diseased appendices but is not necessarily the cause of the pathology. Pinworms can make their way to extraintestinal locations and cause complications. For example, the parasites may carry bacteria into other organs, resulting in abscess formation.

### Host Defenses

Little is known about immune responses to pinworm infection. Infections are more common in children than in adults, suggesting that acquired immunity or some other type of age-related resistance develops. IgE immunoglobulin serum levels in patients are reported to be within normal limits.

### Epidemiology

It is safe to say that everyone, at one time or another, has pinworms. It is a cosmopolitan parasite found most often in families and in institutionalized children. The parasite is transmitted hand-to-mouth after scratching the perianal region, by handling contaminated bedding and night clothing, or by inhaling eggs in airborne dust. Eggs will

not embryonate at temperatures below 23 C, but embryonated eggs remain viable for several weeks under moist and cool conditions.

Prevalence rates for *E vermicularis* are highest in temperate regions. It is estimated that more than 200 million persons are infected. In the United States, pinworm is considered the most common helminthic infection. No animal reservoir exists for *E vermicularis*, although dogs and cats have been incriminated erroneously.

### **Diagnosis**

Children suffering sleepless nights because of perianal itching often have pinworms. Eggs are rarely found in the feces, and the diagnosis is made by finding eggs on perianal swabs made of Scotch tape. The tape is pressed first onto the perianal region and then onto a microscope slide, and is examined microscopically. Perianal specimens are best obtained in the morning before bathing or defecation. Three specimens should be taken on consecutive days before pinworm infection is ruled out.

### **Control**

When an infection is recognized, efforts should be made to improve personal hygiene. Fingernails should be cut short, the perianal region washed in the morning, and bedding and sleeping garments washed daily. Other members of a patient's family should be checked; the entire family may need treatment to eliminate infection. Although several anthelmintics are effective in treating enterobiasis, the drugs presently recommended are pyriminyl pamoate, pyrantel pamoate, and mebendazole. It is advisable to re-treat the patient one month later.

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