Ascaris lumbricoides and Ascaris suum)  
(intestinal roundworms of humans and pigs)

Introduction:
*Ascaris lumbricoides* is one of the largest and most common parasites found in humans. The adult females of this species can measure up to 18 inches long (males are generally shorter), and it is estimated that 25% of the world's population is infected with this nematode. The adult worms live in the small Intestine and eggs are passed in the feces.

Habitat:
The adult worm lives in the small intestine of man

Morphology :
The adult worm is the largest round worm parasitizing the human intestinal tract. It is elongated, cylindrical, and tapers both anteriorly & posteriorly to relatively blunt conical ends. The head is provided with three fleshy lips the digestive & reproductive organs float inside the body cavity which contain an irritating allergic fluid. The irritant action is due to the presence of atoxin called a scarone or a scarase which is probably of the nature of primary albomenoses.

A single female can produce up to 200,000 eggs each day! About two weeks after passage in the feces the eggs contain an infective larval eggs

Eggs:
The fertilized egg of *Ascaris lumbricoides* at the time of oviposition is spherical or sub-spherical, measures 65-75µm by 35-50 µm & consists of the following observable structures

1. A coarsely granular, spherical ovum that usually does not completely fill the shell.
2. A thin innermost membrane that is highly impermeable.
3. A relatively thick, colorless middle layer that is smooth on both inner & outsides.
4. An outermost, coarsely mammilated

Female worms without males produce infertile eggs that are markedly subspherical (88 µm by 38-44 µm), internally they contain
a mass of disorganized granules that completely fill the shell

**Life cycle**, The humans are infected when they ingest such infective eggs. The eggs hatch in the small intestine, the juvenile penetrates the small intestine and enters the circulatory system, and eventually the juvenile worm enters the lungs. In the lungs the juvenile worm leaves the circulatory system and enters the air passages of the lungs. The juvenile worm then migrates up the air passages into the pharynx where it is swallowed, and once in the small intestine the juvenile grows into an adult worm. Why *Ascaris* undergoes such a migration through the body to only end up where it started is unknown. Such a migration is not unique to *Ascaris*, as its close relatives undergo a similar migration in the bodies of
Pathology and symptoms
Ascaris infections in humans can cause significant pathology. The migration of the larvae through the lungs causes the blood vessels of the lungs to hemorrhage, and there is an inflammatory response accompanied by edema. The resulting accumulation of fluids in the lungs results in "ascaris pneumonia," and this can be fatal. The large size of the adult worms also presents problems, especially if the worms physically block the gastrointestinal tract. Ascaris is notorious for its reputation to migrate within the small intestine, and when large worm begins to migrate there is not much that can stop it. Instances have been reported in which Ascaris have migrated into and blocked the bile or pancreatic duct or in which the worms have penetrated the small intestine resulting in acute (and fatal) peritonitis. Ascaris seems to be especially sensitive to anesthetics, and numerous cases have been documented where patients in surgical recovery rooms have had worms migrate from the small intestine, through the stomach, and out the patient's nose or mouth. Ascaris suum is found in pigs. Its life cycle is identical to that of A. lumbricoides. If a human ingests eggs of A. suum the larvae will migrate to the lungs and die. This can cause a particularly serious form of "ascaris pneumonia." Adult worms of this species do not develop in the human's intestine. (Some parasitologists believe that there is but one species of Ascaris that infects both pigs and humans, but any commentary on this issue is beyond the scope of this website.)

Diagnosis
Infections of Ascaris are diagnosed by finding characteristic eggs in the feces of the infected host.
Note the presence of three large lips, a characteristic of ascarids.
A scanning electron micrograph of the anterior end of *Ascaris* showing the three prominent "lips" of *Ascaris lumbricoides*, fertilized egg. Note that the egg is covered with a thick shell that appears lumpy (bumpy) or...
mammillated; approximate size = 65 μm in length. Another example

Another example of a fertilized *Ascaris lumbricoides* egg. (Original image from: *Atlas of Medical Parasitology.*)
An example of an unfertilized *A. lumbricoides* egg. (Original image from: *Atlas of Medical Parasitology.*)
Eggs of *Ascaris suum*. *A. suum* is a common parasite of pigs. The eggs are virtually indistinguishable from those of *A. lumbricoides*. (Original image from Oklahoma State University, College of Veterinary Medicine.)
females of this species can measure over 16 inches long. This specimen was passed by a young girl in Florida. (Original image from DPDx [Identification and Diagnosis of Parasites of Public Health Concern].)

Female and male *Ascaris lumbricoides*; the female measures approximately 16 inches (40 cm) in length.
A large mass of *Ascaris lumbricoides* that was passed from the intestinal tract. The ruler at the bottom of the image is 4 cm (about 1.5 inches) in length.

**Conclusion**

*the infective stage is larvated eggs*

*to cause pneumonia in the lung during circulation& the adult will be blocked*

*the small intestinal tract*

*Up stream movement this movement for \textit{A. lumbericoides} through mouth or nose*

*sometimes the infected man may die due to this irritation action after changing to anaphylactic or HSR*
Lecture 2  DR. Jabar Etaby

Introduction

Patients with hookworm infection often are asymptomatic; however, chronic hookworm infection is a common cause of moderate and severe hypochromic, microcytic anemia in people living in tropical developing countries, and heavy infection can cause hypoproteinemia with edema.

EPIDEMIOLOGY
Humans are the only reservoir. Hookworms are prominent in rural, tropical, and subtropical areas where soil contamination with human feces is common. Although the prevalence of both hookworm species is equal in many areas, *A duodenale* is the predominant species in the Mediterranean region, northern Asia, and
selected foci of South America. *N americanus* is predominant in the Western hemisphere, sub-Saharan Africa, Southeast Asia, and a number of Pacific islands.

**LIFE CYCLE**

Larvae and eggs survive in loose, sandy, moist, shady, well-aerated, warm soil (optimal temperature 23°C–33°C [73°F–91°F]). Hookworm eggs from stool hatch in soil in 1 to 2 days as rhabditiform larvae. These larvae develop into infective filariform larvae in soil within 5 to 7 days and can persist for weeks to months. Percutaneous infection occurs after exposure to infectious larvae. *A duodenale* transmission can occur by oral ingestion and possibly through human milk. Untreated infected patients can harbor worms for 5 years or longer. The time from exposure to development of noncutaneous symptoms is 4 to 12 weeks.

**Clinical signs**

Patients with hookworm infection often are asymptomatic; however, chronic hookworm infection is a common cause of moderate and severe hypochromic, microcytic anemia in people living in tropical developing countries, and heavy infection can cause hypoproteinemia with edema. Chronic hookworm infection in children may lead to physical growth delay, deficits in cognition, and developmental delay. After contact with contaminated soil, initial skin penetration of larvae, often involving the feet, can cause a stinging or burning sensation followed by pruritus and a papulovesicular rash that may persist for 1 to 2 weeks. Pneumonitis associated with migrating larvae is
uncommon and usually mild, except in heavy infections. Colicky abdominal pain, nausea, and/or diarrhea and marked eosinophilia can develop 4 to 6 weeks after exposure. Blood loss secondary to hookworm infection develops 10 to 12 weeks after initial infection and symptoms related to serious iron-deficiency anemia can develop in long-standing moderate or heavy hookworm infections. After oral ingestion of infectious *Ancylostoma duodenale* larvae, disease can manifest with pharyngeal itching, hoarseness, nausea, and vomiting shortly after ingestion.

**ETIOLOGY**

*Necator americanus* is the major cause of hookworm infection worldwide, although *A duodenale* also is an important hookworm in some regions. Mixed infections are common. Both are roundworms (nematodes) with similar life cycles.

*Ancylostoma* spp. and *Necator* spp. (hookworms)
There are many species of hookworms that infect mammals. The most important, at least from the human standpoint, are the human hookworms, *Ancylostoma duodenale* and *Necator americanus*, which infect an estimated 800,000,000 persons, and the dog and cat hookworms, *A. caninum* and *A. braziliense*, respectively. Hookworms average about 10 mm in length and live in the small intestine of the host. The males and females mate, and the female produces eggs that are passed in the feces. Depending on the species, female hookworms can produce 10,000-25,000 eggs per day. About two days after passage the hookworm egg hatches, and the juvenile worm (or larva) develops into an infective stage in about five days.

The next host is infected when an infective larva penetrates the host's skin. The juvenile worm migrates through the host's body and finally ends up in the host's small intestine where it
grows to sexual maturity. The presence of hookworms can be demonstrated by finding the characteristic eggs in the feces; the eggs can not, however, be differentiated to species. Juveniles (larvae) of the dog and cat hookworms can infect humans, but the juvenile worms will not mature into adult worms. Rather, the juveniles remain in the skin where they continue to migrate for weeks (or even months in some instances). This results in a condition known as "cutaneous" or "dermal larval migrans" or "creeping eruption." Hence the importance of not allowing dogs and cats to defecate indiscriminately. The following image provides an excellent example of how hookworms are attached to and embedded in the epithelium of the host's gastrointestinal tract.

**DIAGNOSTIC TESTS**

Microscopic demonstration of hookworm eggs in feces is diagnostic. Adult worms or larvae rarely are seen. Approximately 5 to 8 weeks are required after infection for eggs to appear in feces. A direct stool smear with saline solution or potassium iodide saturated with iodine is adequate for diagnosis of heavy hookworm infection; light infections require concentration techniques. Quantification techniques (eg, Kato-Katz, Beaver direct smear, or Stoll egg-counting techniques) to determine the clinical significance of infection and the response to treatment may be available from state or reference laboratories.

**CONTROL MEASURES**

Sanitary disposal of feces to prevent contamination of soil is necessary in areas with endemic infection. Treatment of all known infected people and screening of high-risk groups (ie, children and agricultural workers) in areas with endemic infection can help decrease environmental contamination. Wearing shoes may not be
fully protective, because cutaneous exposure to hookworm larvae over the entire body surface of children could result in infection. Despite relatively rapid reinfection, periodic deworming treatments targeting preschool-aged and school-aged children have been advocated to prevent morbidity associated with heavy intestinal helminth infections.

A histological section of a hookworm in the host's small intestine. Original image copyrighted and provided by Dr. A.W. Shostak, and used with permission.
**Pathology and Clinical features**

Ground itch may follow skin penetration by filariform larvae. Pneumonitis can result from larval migration through the lungs. Adult worms in the jejunal ingest blood. Occult gastrointestinal bleeding occurs. Iron deficiency anaemia and its sequela in heavy infections.

**Distribution**

900 million infected worldwide.

**Laboratory diagnosis**

Eosinophilia.

Ova may be recovered from faeces by concentration methods. Rhabditiform larvae may be seen in old faecal specimens and must be distinguished from *Strongyloides* by the appearance of the buccal cavity.
Strongyloides stercoralis is an intestinal nematode of humans that infects tens of millions of people worldwide. S. stercoralis is unique among intestinal nematodes in its ability to complete its life cycle within the host through an asexual autoinfective cycle, allowing the infection to persist in the host indefinitely. Under some conditions associated with immunocompromise, this autoinfective cycle can become amplified into a potentially fatal hyperinfection syndrome, characterized by increased numbers of infective filariform larvae in stool and sputum and clinical manifestations of the increased parasite burden and migration, such as gastrointestinal bleeding and respiratory distress. S. stercoralis hyperinfection is often accompanied by sepsis or meningitis with enteric organisms. Glucocorticoid treatment and human T-lymphotropic virus type 1 infection are the two conditions most specifically associated with triggering hyperinfection, but cases have been reported in association with hematologic malignancy, malnutrition, and AIDS. Anthelmintic agents such as ivermectin have been used successfully in treating the hyperinfection syndrome as well as for primary and secondary prevention of hyperinfection in patients whose exposure history and underlying condition put them at increased risk.
INTRODUCTION

*Strongyloides stercoralis* is an intestinal nematode of humans. It is estimated that tens of millions of persons are infected worldwide, although no precise estimate is available. Although most infected individuals are asymptomatic, *S. stercoralis* is capable of transforming into a fulminant fatal illness under certain conditions associated with a compromise of host immunity. Such conditions have commonly been summarized as “defects in cell-mediated immunity,” although the specific circumstances under which *S. stercoralis* hyperinfection develops are not always predictable. Given the increasing numbers of immunocompromised individuals throughout the world, a closer examination of the conditions under which *S. stercoralis* infection becomes dangerous is warranted. Better approaches to identifying, screening, and treating those at risk will likely decrease the morbidity and mortality associated with *S. stercoralis* infection.

**Etiology:** *Strongyloides stercoralis* is the Nematodes of small intestinal tract

**Life cycle of Strongyloides stercoralis** is an unusual "parasite" in that it has both free-living and parasitic life cycles. In the parasitic life-cycle, female worms are found in the superficial tissues of the human small intestine; there are apparently no parasitic males. The female worms produce larvae parthenogenically (without fertilization), and the larvae are passed in the host's feces. The presence of nematode larvae in a fecal sample is characteristic of strongyloidiasis. Once passed in the feces, some of the larvae develop into "free-living" larvae, while others develop into "parasitic" larvae. The "free-living" larvae will complete their development in the soil and mature into free-living males and females. The free-living males and females mate, produce more larvae, and (as above) some of these larvae will develop into "free-living" larvae, while other will develop into "parasitic larvae." As one might imagine, this
free-living life cycle constitutes an important reservoir for human infections. The "parasitic" larvae infect the human host by penetrating the skin (like the hookworms). The larvae migrate to the lungs, via the circulatory system, penetrate the alveoli into the small bronchioles, and they are "coughed up" and swallowed. Once they return to the small intestine, the larvae mature into parasitic females. *S. stercoralis* also infects humans via a mechanism called "autoinfection." Under some circumstances, such as chronic constipation, larvae produced by the parasitic females will remain in the intestinal tract long enough to develop into infective stages. Such larvae will penetrate the tissues of the intestinal tract and develop as if they had penetrated the skin. Autoinfection can also occur when larvae remain on and penetrate the perianal skin. Autoinfection often leads to very high worm burdens in humans (view diagram of the life cycle).

**EPIDEMIOLOGY**

Although *S. stercoralis* is often considered a disease of tropical and subtropical areas, endemic foci are also seen in temperate regions. Low socioeconomic status, alcoholism, white race, and male gender have been associated with higher prevalences of *S. stercoralis* stool positivity. Clusters of cases in institutionalized individuals with mental retardation others suggest that nosocomial transmission can occur. Occupations that increase contact with soil contaminated with human waste, which may include farming and coal mining depending on local practices, increase the risk of infection. Swimming in or drinking contaminated water has not been proven to be a significant source of transmission, perhaps because larvae do not thrive when immersed in water. Different prevalences among ethnic groups may simply reflect behavioral or socioeconomic factors, but some have suggested that different skin types may be more or less resistant to larval penetration.

**CLINICAL SYNDROMES**
As the clinical syndromes of *S. stercoralis* encompass a spectrum and terms are used variably, it is necessary to set forth some definitions before proceeding further.

**Acute Strongyloidiasis**

From experimental human infections, it is known that a local reaction at the site of larval entry can occur almost immediately and may last up to several weeks. Pulmonary symptoms such as a cough and tracheal irritation, mimicking bronchitis, occur as larvae migrate through the lungs several days later. Gastrointestinal symptoms (diarrhea, constipation, anorexia, abdominal pain) begin about 2 weeks after infection, with larvae detectable in the stool after 3 to 4 weeks. Experimental human infections on which this description is based were initiated with many hundreds of larvae and most likely overestimate the severity and perhaps the tempo of naturally acquired infections.

**Chronic Strongyloidiasis**

Chronic infection with *S. stercoralis* is most often asymptomatic. There are a number of signs and symptoms attributable to chronic strongyloidiasis that are unrelated to accelerated autoinfection. Chronic gastrointestinal manifestations, such as intermittent vomiting, diarrhea, constipation, and borborygmus, are common complaints. Pruritus ani and dermatologic manifestations such as urticaria and larva currens rashes are also common. Recurrent asthma and nephrotic syndrome have also been associated with chronic strongyloidiasis infection. Complications such as intestinal obstruction, ileus, hemodynamically significant gastrointestinal bleeding, and acute worsening of chronic intestinal manifestations have occurred in the context of an increased larval burden. Even in the absence of pulmonary symptoms, such presentations could be considered a manifestation of hyperinfection.

**Hyperinfection**

Hyperinfection describes the syndrome of accelerated autoinfection, generally — although not always the result of an alteration in immune status. Parasitologically, the distinction between autoinfection and hyperinfection is quantitative and not
strictly defined. Therefore, the diagnosis of hyperinfection syndrome implies the presence of signs and symptoms attributable to increased larval migration. Development or exacerbation of gastrointestinal and pulmonary symptoms is seen, and the detection of increased numbers of larvae in stool and/or sputum is the hallmark of hyperinfection. Larvae in nondisseminated hyperinfection are increased in numbers but confined to the organs normally involved in the pulmonary autoinfective cycle (i.e., gastrointestinal tract, peritoneum, lungs), although enteric bacteria, which can be carried by the filariform larvae or gain systemic access through intestinal ulcers, may affect any organ system.

**Disseminated Infection**

The term disseminated infection is often used to refer to migration of larvae to organs beyond the range of the pulmonary autoinfective cycle. This does not necessarily imply a greater severity of disease. Extrapulmonary migration of larvae has been shown to occur routinely during the course of experimental chronic *S. stercoralis* infections in dogs and has been reported to cause symptoms in humans without other manifestations of hyperinfection syndrome. Similarly, many cases of hyperinfection are fatal without larvae being detected outside the pulmonary autoinfective route.

As documenting disseminated infection may be more a matter of vigilance than a fundamental difference in disease mechanisms, the term hyperinfection will be used here to include cases with evidence of larval migration beyond the pulmonary autoinfective route.
CONCLUSIONS

Our understanding of *S. stercoralis* infections in normal and immunocompromised hosts continues to evolve. Relatively recently, it was thought that any defect in cellular immunity could tip the equilibrium of chronic strongyloidiasis toward hyperinfection. Although various immunocompromising conditions have been associated with hyperinfection,
Cutaneous (dermal) larval migrans

There are several examples of parasites that are normally found in pets but can be transmitted to humans. For example, a common tapeworm of dogs, *Dipylidium caninum*, can be transmitted to humans. Immature forms of the common roundworm of dogs, *Toxocara canis* can also be found in humans, causing a disease known as **visceral larval migrans**. Immature forms of both cat and dog hookworms can also infect humans, and this results in a disease called cutaneous or dermal larval migrans (CLM or DLM).

The eggs of dog and cat hookworms hatch after being passed in the host's feces, and the next host is infected when these larvae penetrate the host's skin. Unfortunately, these larvae can not tell the skin of one animal from another, so they will penetrate human skin if they come in contact with it. However, a human is an unnatural host, so the larvae do not enter the blood stream as they would in a dog or cat. Rather, they remain in the skin for extended periods of time (weeks or months in some instances) and finally die. As the larvae migrate through the skin and finally die, there is an inflammatory response, and the progress of the larvae through the skin can actually be followed since they leave a tortuous "track" of inflamed tissue just under the surface of the skin. Treatment of such infections requires surgical removal of the migrating larvae. Considering the location of larvae, just under the skin, in light infections this can be done under local anesthesia and is a relatively simple procedure. Infections involving large numbers of larvae can be very uncomfortable, and treatment (removal) might require general anesthesia and supportive treatment with anti-inflammatory drugs.

How do humans come in contact with the larvae of dog and cat hookworms? A common source of infection in developed countries is probably sandboxes. If you have a sandbox in your backyard, it is almost certain that cats in the neighborhood are using it as a large litter box. Moreover, the sand provides a nearly ideal environment for the hookworm eggs to develop and hatch and for the larvae to survive. Thus, keeping sandboxes covered to prevent cats from defecating in them is a worthwhile "ounce of prevention." Other places where cats might defecate are also possible sources of infection, including flower beds and vegetable gardens.

Dogs are much less fastidious about where they defecate, so it is more difficult to control dog feces as a possible source of infection. If you own a dog two measures that you should take are (1) keep your dog free of hookworms and (2) make sure that you clean up the dog's feces on a regular basis. Also, if you "walk" your dog in a park or playground, and in particular in my front yard, make sure that you pick up and dispose of any fecal material the dog might leave behind.
CLM of the foot.
(Original image from: Companion Animal Surgery.")
CLM of the foot.
CLM (Original image from and copyrighted by Dermatology Internet Service, Department of Dermatology, University of Erlangen.)
CLM of the foot.
(Original image from and copyrighted by Dermatologic Image Database, Department of Dermatology, University of Iowa
College of Medicine).
*Strongyloides stercoralis* in the wall of the small intestine; numerous adults are visible in this section, as is the abnormal appearance of the intestinal mucosa.
A higher power magnification of the above image; the adult worms are labeled (*), and a higher power magnification of the enclosed area is shown in the following image. An enlargement

An enlargement of the enclosed area in the above image
Strongyloides stercoralis adults in the small intestine. (From "Parasite of the Month.")
Strongyloides stercoralis larva as it would appear in a fecal sample. Note the rhabditiform esophagus. (From "Parasite of the Month"

Another example of the larva in which the rhabditiform esophagus shows up clearly. (Original image from "Atlas of Medical Parasitology.")
CONCLUSIONS

Our understanding of *S. stercoralis* infections in normal and immunocompromised hosts continues to evolve. Relatively recently, it was thought that any defect in cellular immunity could tip the equilibrium of chronic strongyloidiasis toward hyperinfection. Although various immunocompromising conditions have been associated with hyperinfection, steroids and HTLV-1 infection are the most consistent