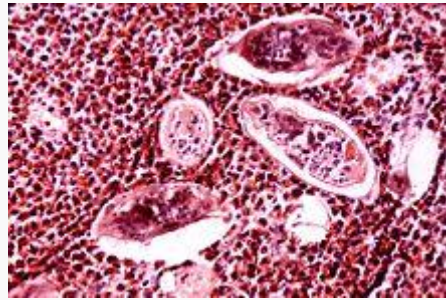


Schistosoma haematobium



Histopathology of bladder shows eggs of *Schistosoma haematobium* surrounded by intense infiltrates of eosinophils.

Scientific classification

Kingdom: Animalia

Phylum : Platyhelminthes

Class : Trematoda

Subclass : Digenea

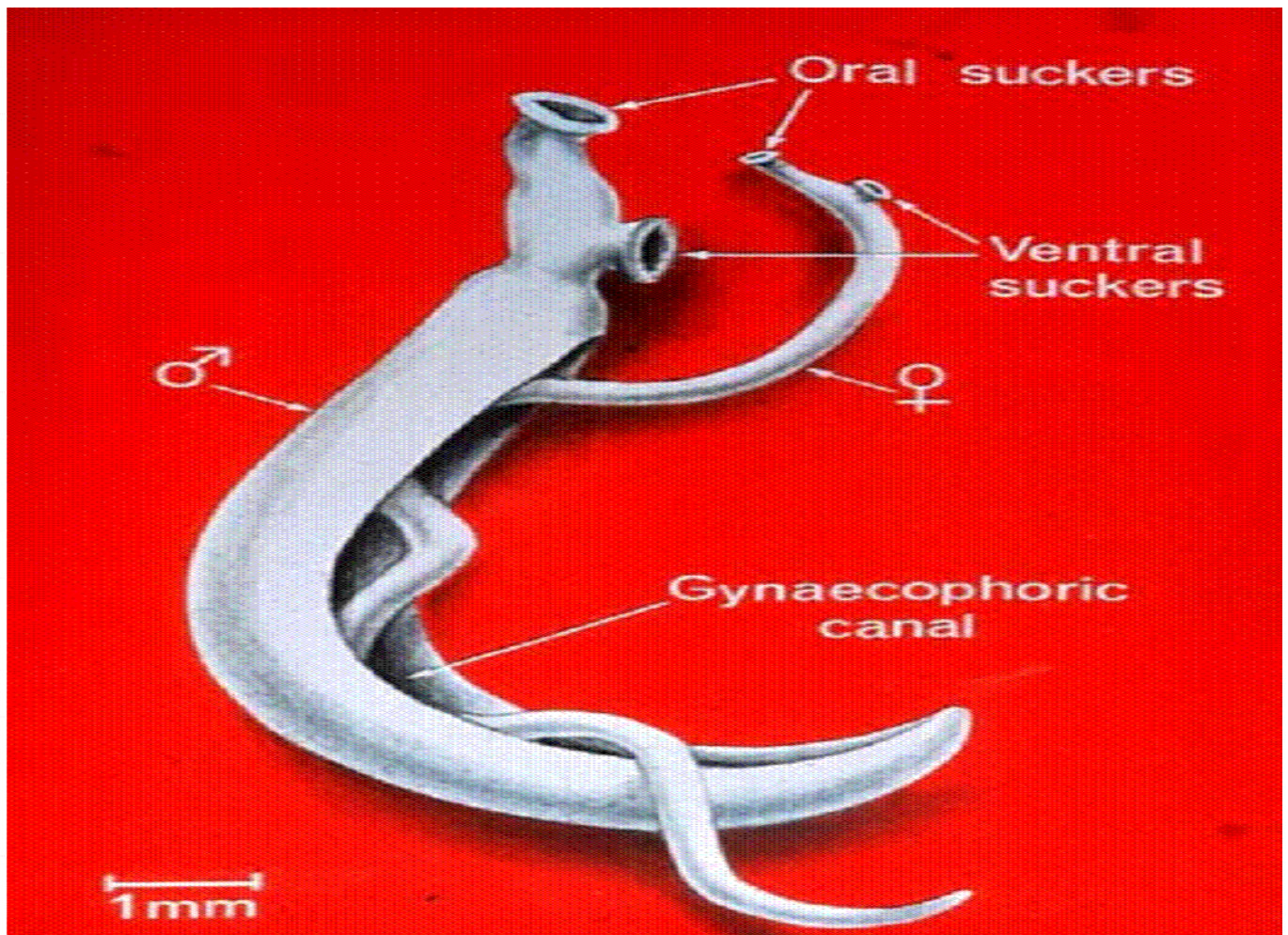
Order : Prosostomata

Family : Schistosomatoidea

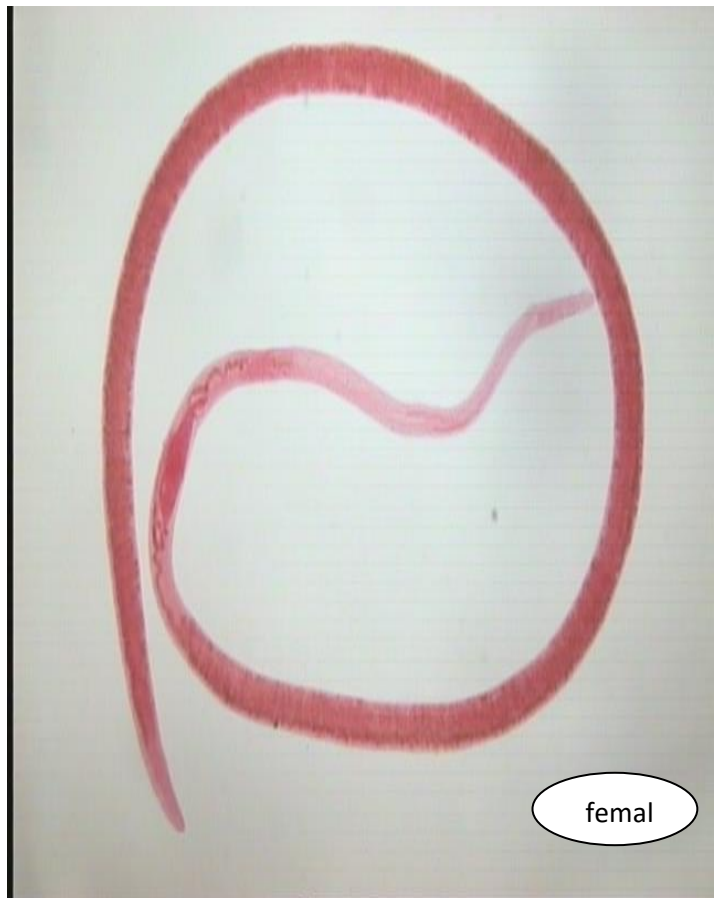
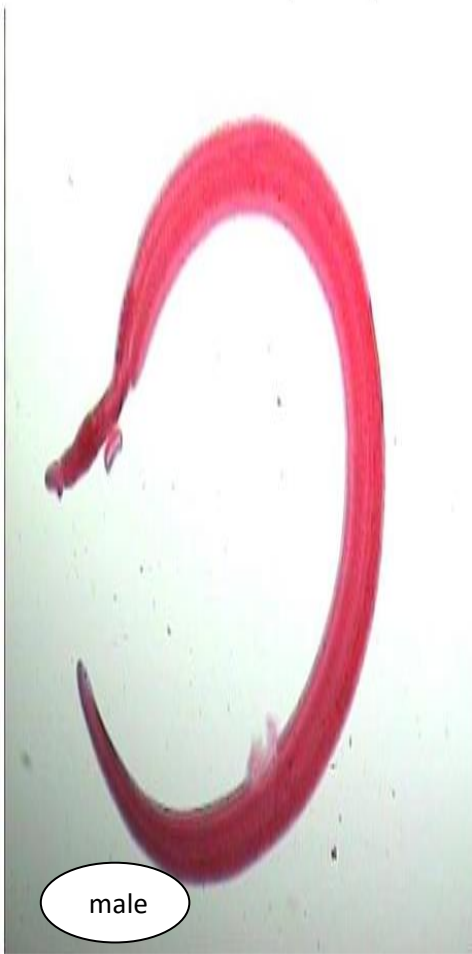
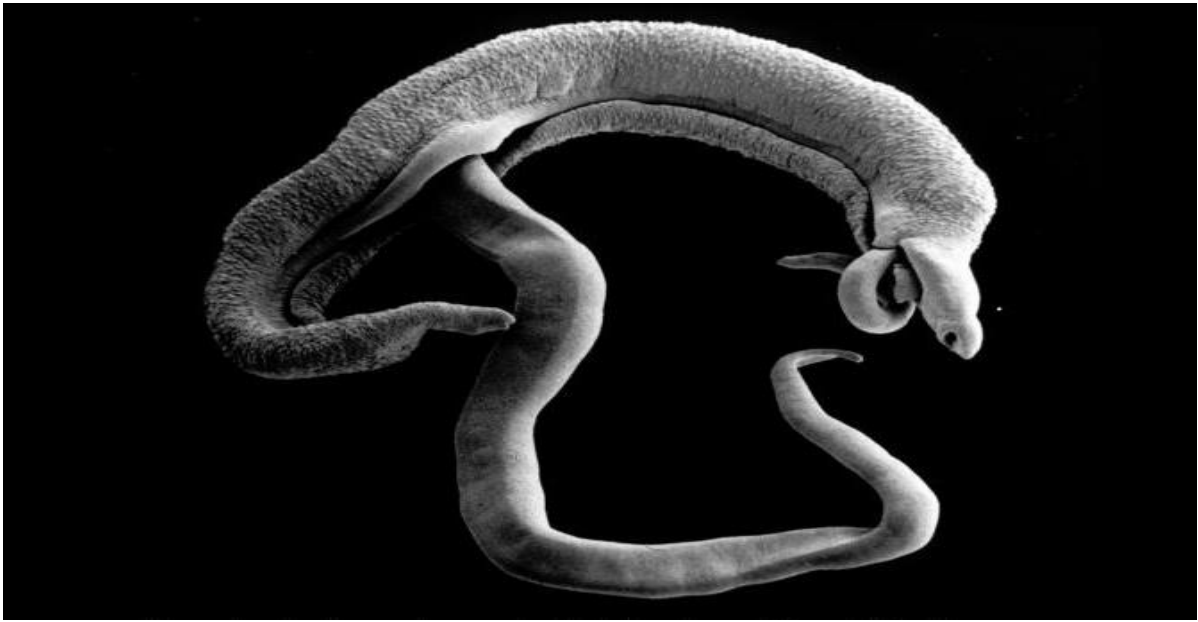
Genus : *Schistosoma*

Species : *Schistosoma haematobium*

Description



Adult males are 10 to 15 mm long. They have deep grooves called gynecophoral canals in which adult females typically lie. Males have many small nodules (tubercles) on their dorsal surfaces and many tiny spines on their suckers and inside their gynecophoral canals. Females are longer (16-22 mm), smoother, and more slender. Both sexes have two suckers, one anterior and one ventral, which are used to grip venule walls.





Epidemiology

Schistosoma haematobium is an important digenetic trematode, and is found in Africa and the Middle East. It is a major agent of schistosomiasis; more specifically, it is associated with urinary schistosomiasis.

Adults are found in the Venous plexuses around the urinary bladder and the released eggs traverse the wall of the urine bladder causing haematuria and fibrosis of the bladder. The bladder

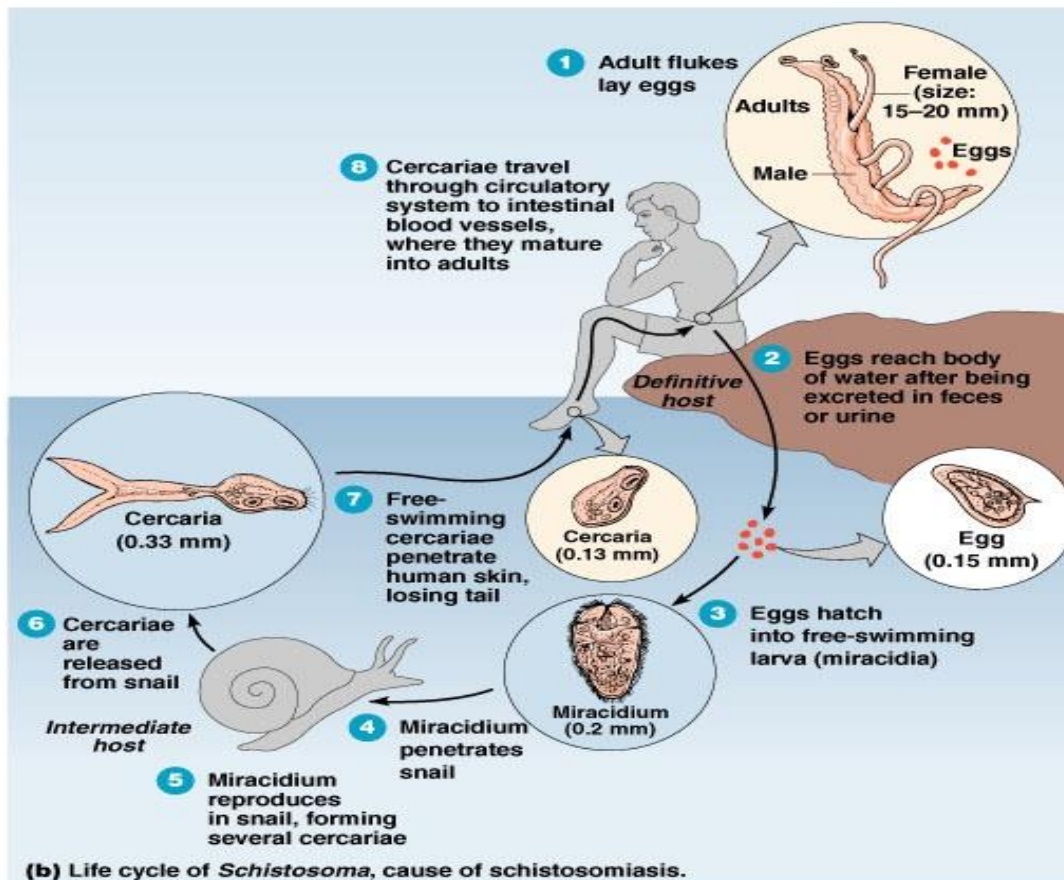
becomes calcified, and there is increased pressure on ureters and kidneys otherwise known as hydronephrosis. Inflammation of the genitals due to *S. haematobium* may contribute to the propagation of HIV. Studies have shown the relationship between *S. haematobium* infection and the development of squamous cell carcinoma of the bladder.



Life cycle

The free swimming infective larval cercariae burrow into human skin when it comes into contact with contaminated water. The cercariae enter the blood stream of the host where they travel to the liver to mature into adult flukes. In order to avoid detection by the immune system inside the host, the adults have the ability to coat themselves with host antigen after a period of about three weeks the young flukes migrate to the urinary bladder veins to copulate. The female fluke lays as many as 30 eggs per day which migrate to the lumen of the urinary bladder and ureters. The eggs are eliminated from the host into the water supply with micturition. In fresh water, the eggs hatch forming free swimming miracidia which penetrate into the intermediate snail host^[7] (*Bulinus* spp., e.g. *B. globosus*, *B. forskalii*, *B. nyassanus* and *B. truncatus*). Inside the snail, the miracidium sheds its epithelium and develops into a mother sporocyst. After two weeks the mother begins forming daughter sporocysts. Four weeks after the initial penetration of the miracidium into the snail furcocercous cercariae begin to be released. The cercariae cycle from the top of the water to the bottom for three days in the search of a human host. Within half an hour the cercariae enter the host epithelium.

دورة حياة دودة البلهارسيا



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Diagnosis

The majority of diagnoses are made by examination of the urine for eggs. In chronic infections, or if eggs are difficult to find, an intradermal injection of schistosome antigen to form a wheal is effective in determining infection. Alternate diagnosis can be made by complement fixation tests.



Prevention

The main cause of schistosomiasis is the dumping of human waste into water supplies. Hygienic disposal of waste would be sufficient to eliminate the disease.

Immunopathology

The immune system responds to eggs in liver causing hypersensitivity; an immune response is necessary to prevent damage to hepatocytes. The hosts' antibodies which bind to the tegument of the Schistosome don't bind for long since the tegument is shed every few hours. The schistosome can also take on host proteins. Schistomiasis can be divided into three phases:

(١) the migratory phase lasting from penetration to maturity,

(٢) the acute phase which occurs when the schistosomes begin producing eggs, and

(٣) the chronic phase which occurs mainly in endemic areas.

Pathology

The ova are initially deposited in the muscularis propria which leads to ulceration of the overlying tissue. Infections are characterized by pronounced acute inflammation, squamous metaplasia, blood and reactive epithelial changes. Granulomas and multinucleated giant cells may be seen.

Treatment

The drug of choice is praziquantel, a quinolone derivative.

Ascaris lumbricoides

Ascaris lumbricoides



An adult female *Ascaris* worm

Scientific classification

Kingdom: Animalia

Phylum : Nematoda

Class : Secernentea

Order : Ascaridida

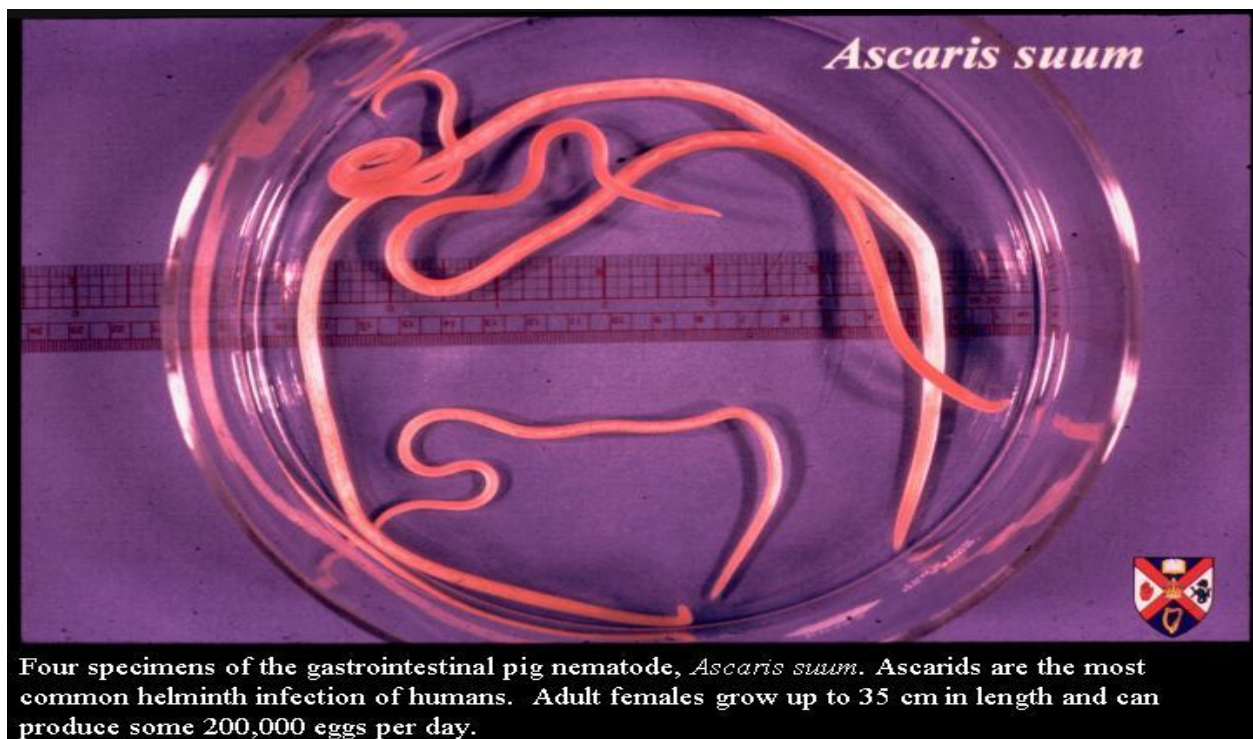
Family : Ascarididae

Genus : *Ascaris*

Species : *Ascaris lumbricoides*.

Description

Ascaris lumbricoides is the giant roundworm of humans, growing to a length of up to 30 cm. It is one of several species of *Ascaris*. An ascarid nematode of the phylum Nematoda, it is the largest and most common parasitic worm in humans. This organism is responsible for the disease ascariasis, a type of helminthiasis and one of the groups of neglected tropical diseases. An estimated one-sixth of the human population is infected by *A. lumbricoides* or another roundworm.



Epidemiology

Ascariasis is prevalent worldwide, especially in tropical and subtropical countries.

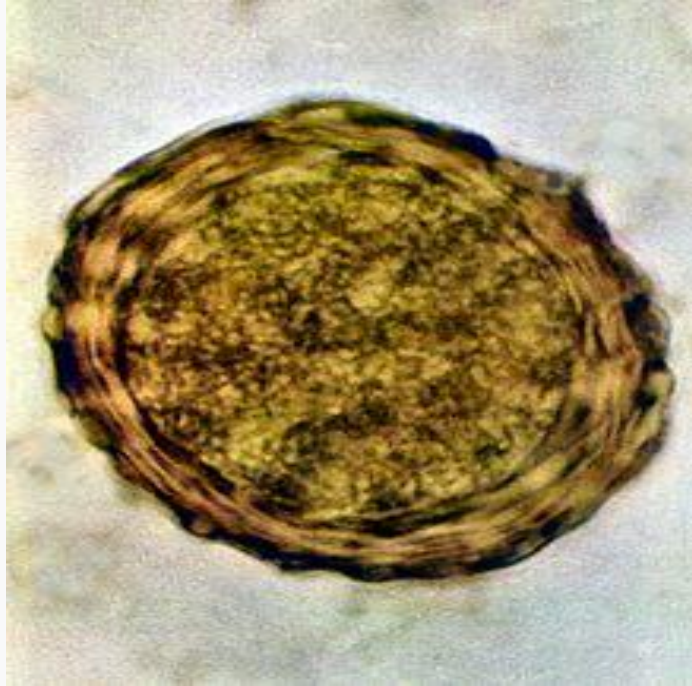
More than 2 billion people are affected by this infection.^[3] The United States has a reported prevalence of 0.8% of the total population as of 1987. *A. lumbricoides* eggs are extremely resistant to strong chemicals, desiccation, and low temperatures. The eggs can remain viable in the soil for several months or even years.^[6]

Eggs of *A. lumbricoides* have been identified in archeological coprolites in the Americas, Europe, Africa, the Middle East, and New Zealand, the oldest ones being more than 24,000 years old.

Morphology



Fertile egg as can be seen in a microscope



Fertile egg in human faeces (detail)



Infertile egg

A. lumbricoides is characterized by its great size. Males are 2–4 mm in diameter and 15–31 cm long. The male's posterior end is curved ventrally and has a bluntly pointed tail. Females are 3–6 mm wide and 20–49 cm long. The vulva is located in the anterior end and accounts for about one-third of its body

length. Uteri may contain up to 27 million eggs at a time, with 200,000 being laid per day. Fertilized eggs are oval to round in shape and are 40-70 µm long and 30-50 µm wide with a thick outer shell. Unfertilized eggs measure 11-14 µm long and 11 µm wide.

Lifecycle

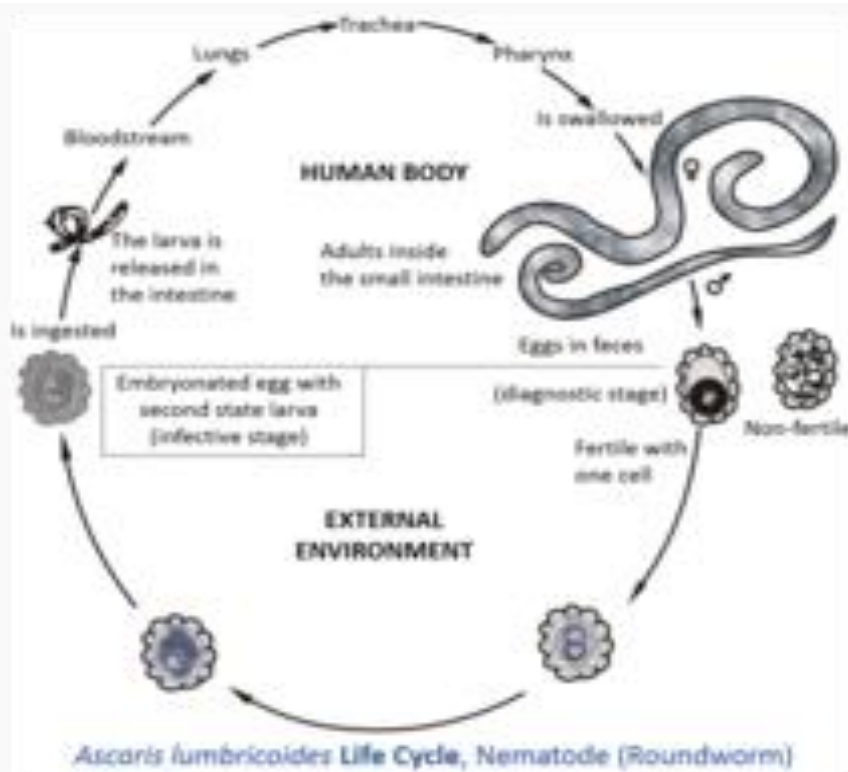
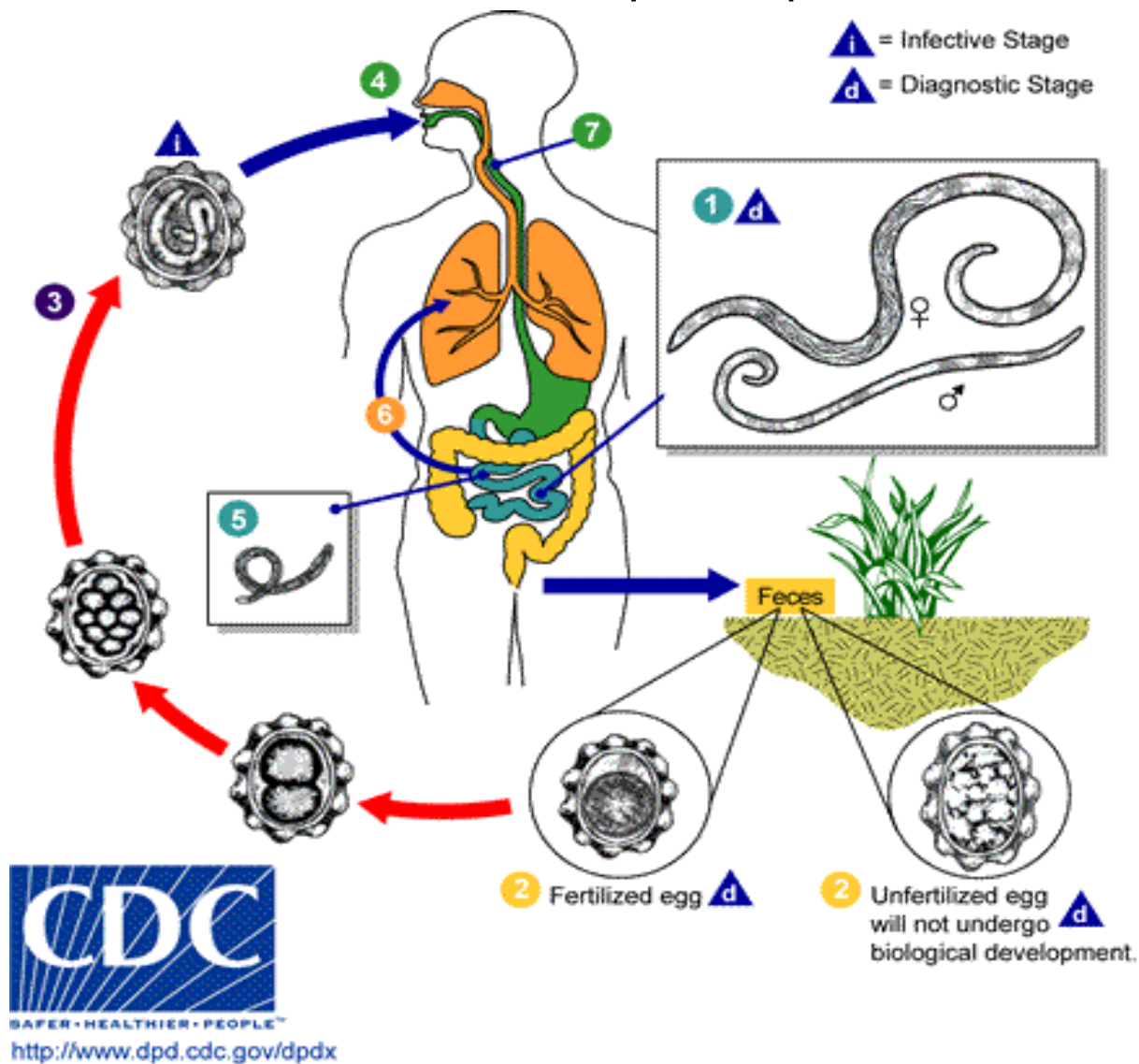


Image showing lifecycle inside and outside of the human body of one fairly well described helminth: *A. lumbricoides*

A. lumbricoides, a roundworm, infects humans when an ingested fertilised egg becomes a larval worm that penetrates the wall of the duodenum and enters the blood stream. From there, it is carried to the liver and heart, and enters pulmonary circulation to break free in the alveoli, where it grows and molts. In three weeks, the larva passes from the respiratory system to be coughed up, swallowed, and thus returned to the small intestine, where it matures to an adult male or female worm. Fertilization can now occur and the female produces as many as 200,000 eggs per day for a year. These fertilized eggs become infectious after two weeks in soil; they can persist in soil for 10 years or more.^[3]

The eggs have a lipid layer which makes them resistant to the effects of acids and alkalis, as well as other chemicals. This resilience helps to explain why

this nematode is such a ubiquitous parasite.



Infections

Infections with these parasites are more common where sanitation is poor, and raw human feces are used as fertilizer.

Symptoms

Often, no symptoms are seen with an *A. lumbricoides* infection. However, in the case of a particularly bad infection, symptoms may include bloody sputum, cough, fever, abdominal discomfort, intestinal ulcer, passing worms, etc.^{[^][9]} Ascariasis is also the most common cause of Löffler's syndrome worldwide. Accompanying symptoms include pulmonary infiltration, eosinophilia, and radiographic opacities.

Prevention

Preventing any fecal-borne disease requires educated hygienic habits/culture and effective fecal treatment systems. This is particularly important with *A. lumbricoides* because its eggs are one of the most difficult pathogens to kill (second only to prions), and the eggs commonly survive 1–3 years. *A. lumbricoides* lives in the intestine where it lays eggs. Infection occurs when the eggs, too small to be seen by the unaided eye, are eaten. The eggs may get onto vegetables when improperly processed human feces of infected people are used as fertilizer for food crops. Infection may occur when food is handled without removing or killing the eggs on the hands, clothes, hair, raw vegetables/fruit, or cooked food that is (re)infected by handlers, containers, etc. Bleach

does not readily kill *A. lumbricoides* eggs, but it will remove their sticky film, to allow the eggs to be rinsed away. *A. lumbricoides* eggs can be reduced by hot composting methods, but to completely kill them may require rubbing alcohol, iodine, specialized chemicals, cooking heat, or "unusually" hot composting (for example, over 60 °C (140 °F) for 24 hours.

Details of infection process

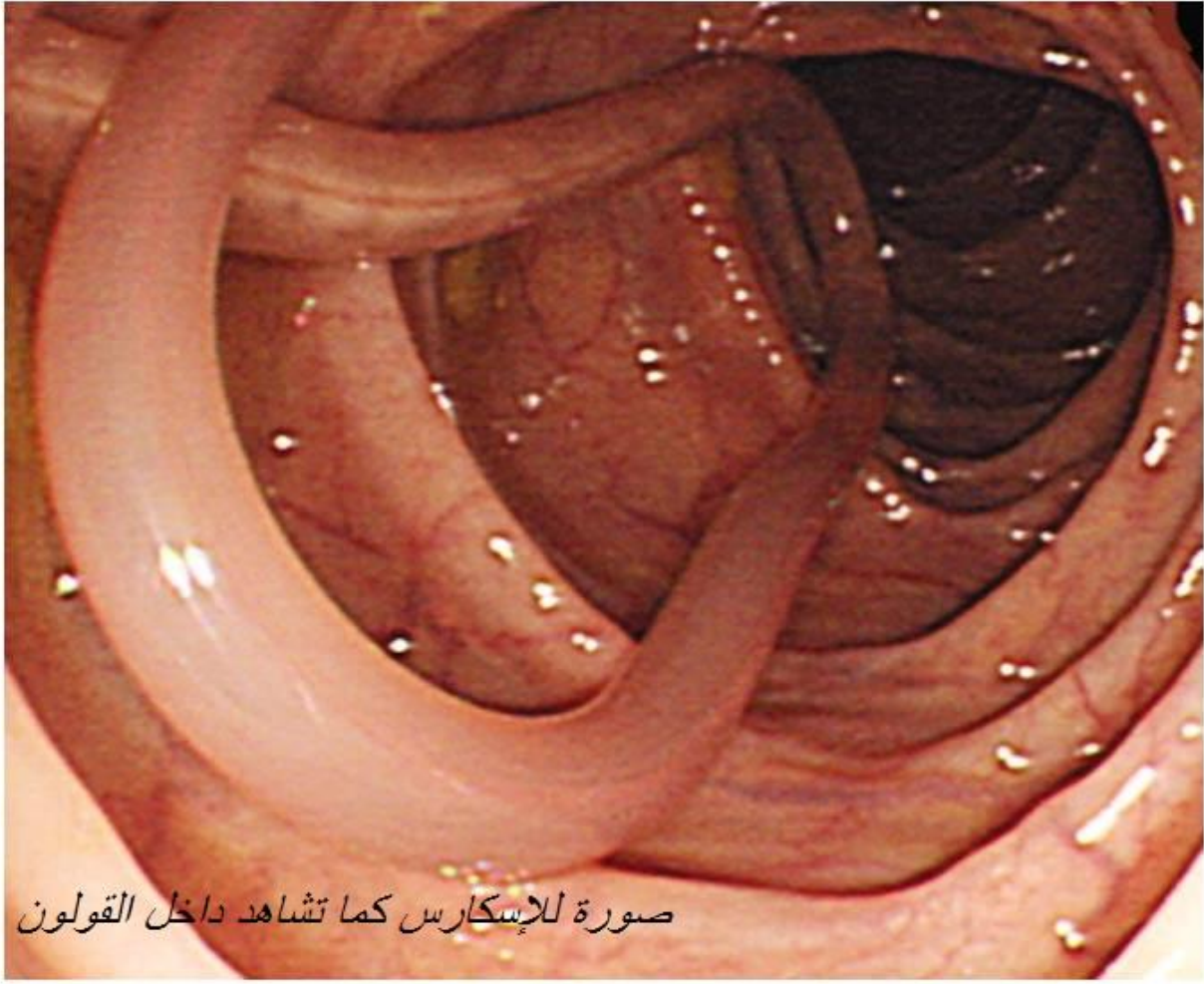
Infections happen when a human swallows water or food contaminated with unhatched eggs, which hatch into juveniles in the duodenum. They then penetrate the mucosa and submucosa and enter venules or lymphatics. Next, they pass through the right heart and into pulmonary circulation. They then break out of the capillaries and enter the air spaces. Acute tissue reaction occurs when several worms get lost during this migration and accumulate in other organs of the body. The juveniles migrate from the lung up the respiratory tract to the pharynx where they are swallowed. They begin producing eggs within 60–65 days of being swallowed. These are produced within the small intestine, where the juveniles mature. It might seem odd that the worms end up in the same place where they began. One hypothesis to account for this behavior is that the migration mimics an intermediate host, which would be required for juveniles of an ancestral form to develop to the third stage. Another possibility is that

tissue migration enables faster growth and larger size, which increases reproductive capacity.^[11]

Diagnosis and treatment

Most diagnoses are made by identifying the appearance of the worm or eggs in feces. Due to the large quantity of eggs laid, physicians can diagnose using only one or two fecal smears.

Infections can be treated with drugs called ascaricides. The treatment of choice is mebendazole. The drug functions by binding to tubulin in the worms' intestinal cells and body-wall muscles. Nitazoxanide and ivermectin can also be used.



صورة للإسكارس كما تشاهد داخل القولون



جزء من الأمعاء تم إستانصاله جراحيا بسبب موت أنسجته بعد إنسداده ،
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