

Class Nematoda - The Roundworms

- A. Introduction - nematodes comprise the group of organisms containing the largest number of helminth parasites of humans. They are unsegmented, bilaterally symmetrical, and exhibit great variation in their life cycles. Generally, they are long-lived (1 - 30+ years).
1. Includes both free-living and parasitic forms. Some can be both free-living and parasitic at times, i.e. *Strongyloides stercoralis*.
 2. Vary greatly in size from a few millimeters to over a meter.
 3. The male worm frequently has a curved or coiled posterior end with copulatory spicules and in some species a bursa is present.
 4. The adult anterior may have oral hooks, teeth or plates in the buccal cavity for purpose of attachment.
 5. Body is fairly complex.
 6. The outer body surface is called a cuticle, there are muscle layers underneath.
 7. Internal organs include a complex nerve cord, a well-developed digestive system and complete reproductive organs.
 - a. The male has testes, vas deferens, seminal vesicle and an ejaculatory duct.
 - b. The female has ovaries, oviduct, seminal receptacle, uterus and vagina.
 8. Reproductive capacity (fecundity) is proportional to complexity of life cycle.
 9. Humans are definitive hosts for many roundworms of medical importance.
 10. The adult female produces fertilized eggs, or larvae which may be infective to new host in three ways:
 - a. Eggs are immediately infective after ingestion by humans.
 - b. Eggs or larvae require a period of development in the environment to become infective.
 - c. Eggs or larvae are transmitted to a new host by an insect.
 11. Developing larvae go through a series of 4 molts with the third stage, the filariform larva, being most often the infective stage.
 12. Infection with roundworms can be by ingestion of infective eggs or larvae or by larval penetration of skin or by transmission of larvae through insect bite.
 13. About one-half of the nematodes parasitic for man are intestinal, the others are found in various tissues.
 14. Pathogenicity of intestinal nematodes may be due to larval migration through body tissues, piercing of intestinal wall, bloodsucking activities of worms or allergic reactions to secretions, worms or larvae.

B. Overview

1. Terms

- a. Filariform larvae - the infective stage; long, thread-like; often “designed” for penetration.
- b. Rhabditiform larvae - characterized by the presence of a muscular esophagus and bulbular pharynx. The first “molt “ worms after leaving the egg are termed “rhabditiform”.

2. Life cycle stages

- a. Egg - characteristic of the Genus. Size & shape are relatively consistent.
- b. Larvae - undergo several molts (third stage generally the infective one)
- c. Adult - varies in size from Genus to Genus; range from less than 1 mm to over one meter.

3. General Life Cycle of Intestinal nematodes

- a. Humans ingest infective eggs. Hookworm and *Strongyloides stercoralis* are exceptions, in these filariform larvae penetrate the skin to gain entry.
- b. Larvae hatch in intestine
- c. Male and female adults develop in the intestine. With *Ascaris lumbricoides*, Hookworms, and *Strongyloides stercoralis* larvae penetrate the intestinal mucosa and initiate a heart – lung cycle enroute to the intestinal tract to mature to adults.
- d. Fertilized eggs are produced
- e. Diagnostic stage - eggs or larvae in feces.
- f. Larvae develop within the egg in warm, moist soil (except for *Strongyloides stercoralis*, whose eggs hatch in the intestine, with larvae passing in the feces)

C. Intestinal nematodes

1. *Enterobius vermicularis* - The Pinworm



Anterior end of adult worm



Pinworm eggs



Pinworm egg, enlarged

a. Distribution

- (1) Most common helminth infection in the U.S.A. Transmission is

direct, person-to-person; egg is infective immediately or within hours of being shed by the female.

- (2) Common worldwide but more prevalent in temperate climates.
- (3) Higher prevalence in Caucasians than in Negroes.
- (4) It is a group infection especially common among children. Very often associated with low sanitation and hygiene.
- (5) Humans are the only known host. Dogs and cats are not infected.

b. Life cycle

- (1) Eggs are ingested, hatch in intestine, larvae mature, adults live in the colon.
- (2) Gravid females migrate to the perianal area at night to lay eggs. Each female produces up to 15,000 eggs.
- (3) Eggs develop to the infective stage within 4-6 hours.
- (4) Eggs are resistant to drying, and can survive for extended periods in cool, moist environment. Viable eggs can be found on bed linens, towels, furniture, windowsills, door jams and in dust. Cleaning eggs from the environment and treating all persons in the household is important in order to break life cycle.
- (5) Eggs are found only rarely in fecal samples because release is most often external to the intestines. On occasion, a worm will die, releasing her eggs in the bowel. This is rare, however.

c. Morphology

- (1) Adults - female: creamy white, ~ 8-13 mm long, with sharply pointed tails, and wing-like flaps (cervical alae) at the head end; male: small (2-5 mm) with strongly curved posterior.
- (2) Eggs - 50 to 60 x 20 to 32 microns, broadly oval, and flattened on one side. Somewhat compressed laterally; normally are embryonated (contain a larva).

d. Diagnosis

- (1) Recovery and identification of eggs or adults from the perianal region utilizing the cellophane tape preparation.
- (2) Specimens must be collected the first thing in the morning upon waking, especially before bathing or bowel movements.
- (3) Eggs are rarely found in fecal samples because release is usually external to the intestines.

e. Major pathology and symptoms

- (1) One third of all cases are asymptomatic
- (2) Infections rarely cause serious lesions.
- (3) Other symptoms may be associated with the migration of the female out of the anus to lay her eggs and include: severe perianal itching due to hypersensitivity reaction (eggs can get on hands and re-infect), mild nausea or vomiting, loss of sleep, irritability, slight irritation of the intestinal mucosa, and vulval irritation in girls from migrating worms (after egg laying, worm may migrate into the vagina instead of the anus).

2. *Trichuris trichiura* - The Whipworm



T. trichiura female



T. trichiura male



T. trichiura egg

a. Life cycle

- (1) Infective, fully embryonated eggs are ingested, larvae hatch in small intestine, penetrate and develop in the intestinal villi, return to lumen and migrate to the area of the cecum.
- (2) Larvae mature and live in the colon. Worms embed their anterior portion (as much as two-thirds of the worm) into the mucosa.
- (3) Eggs are released into the stool.
- (4) Eggs must undergo development in the soil for a period of time (approximately 10 days to 3 weeks) before they become infective.
- (5) The worm's life span is estimated to be 4 - 8 years.

b. Morphology

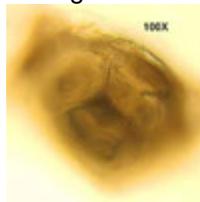
- (1) Adults - females: 35 to 50 mm long, anterior two-thirds is long and threadlike, then it expands into a broader posterior; males: 30 to 45 mm long, shape similar to female but exhibiting a very strong (360 or more degree) curvature of tail.
- (2) Eggs - 50 to 55 x 22 to 25 microns, barrel shaped, with clear polar plugs at each end.

- c. Diagnosis - recovery and identification of eggs in the feces.
- d. Major pathology and symptoms
 - (1) Slight infections - are usually asymptomatic, no treatment is necessary.
 - (2) Heavy infections - surface of colon is matted with worms which causes:
 - (a) bloody or mucoid diarrhea
 - (b) weight loss and weakness; infections with 200 or more worms in children may cause a chronic dysentery, profound anemia and growth retardation.
 - (c) abdominal pain and tenderness
 - (d) increased peristalsis and rectal prolapse, especially in children.
- e. Distribution
 - (1) In the U.S.A., it is prevalent in the warm, humid climate of the southeastern states and in immigrants from tropical areas.
 - (2) It is the third most common intestinal helminth infection.
 - (3) Higher prevalence in warm countries and areas of poor sanitation, especially in countries which utilize "night soil" for fertilizer.
 - (4) Common among children and in the institutionalized mentally retarded.
- f. Notes
 - (1) Commonly, double infections occur with *Ascaris lumbricoides* because of the similar mode of infection.
 - (2) Drug treatment may cause production of distorted eggs.

2. *Ascaris lumbricoides* - The Large Intestinal Roundworm



Ascaris lumbricoides adult



Head-on view of lips



Ascaris lumbricoides egg

- a. Life cycle - complex, involves a heart-lung cycle.

- (1) Humans ingest embryonated eggs containing infective larvae.
- (2) Larvae hatch from the eggs in the small intestine, penetrate the intestine wall, enter the bloodstream, migrate to the liver, travel to the lung via the blood stream.
- (3) Larvae break out of lung capillaries into alveoli, travel to the bronchioles, and are coughed up to the pharynx. They are swallowed and return to the intestine. Two molts to 4th stage larvae take place in alveoli.
- (4) Larvae mature to adults in the small intestine.
- (5) The large, muscular worms do not attach to the intestinal wall, but maintain their position by constant movement. Worms have a life span of approximately 1 year.
- (6) Undeveloped eggs are passed in the feces. These eggs embryonate in the soil and are infective after approximately two weeks to one month. The egg shell is very thick, making it quite resistant to environmental changes. Eggs have been noted to embryonate in 10% formalin.
- (7) Eggs remain infective for up to 5 years if protected from direct sunlight and desiccation.

b. Morphology

- (1) Adults - males measure 15 to 30 cm long, with strongly curved tails; females measure 20 to 35 cm long, and are a bit "fatter", with straight tails.
- (2) Eggs - one female produces approximately 200,000 per day. The thick-shelled egg has an outer shell membrane which is heavily mamillated (cortication). This layer is sometimes rubbed off in passage down the fecal stream. Infertile eggs often appear longer, and thinner shelled (sometimes confused for fluke eggs).

c. Diagnosis - recovery and identification of eggs and/or adults in the feces.

d. Major pathology and symptoms

- (1) Pneumonia - associated with migration of larvae in the lungs.
- (2) Obstruction of the intestines, appendix, or common bile duct.
- (3) Vomiting and abdominal pain.
- (4) May cause malnutrition in children with heavy infections or poor diet.
- (5) Some infections are asymptomatic

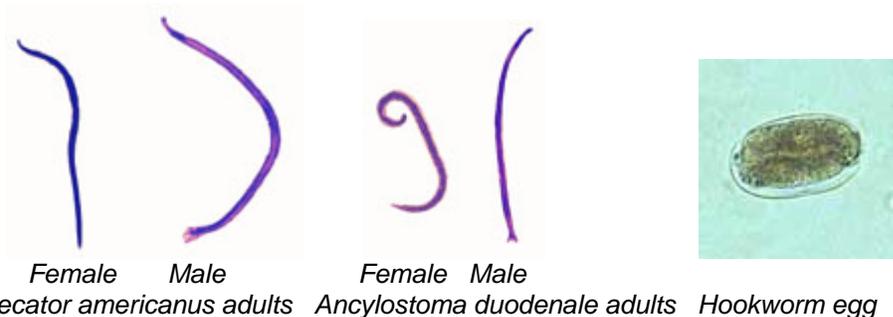
e. Distribution

- (1) Same as for *T. trichiura*.
- (2) Eggs thrive best in clay soil with dense shade, heavy rain and warm climate.

f. Notes

- (1) *Ascaris* is the largest intestinal nematode.
- (2) The second most common helminth infection in the U.S.
- (3) Mixed infections with *T. trichiura* are common. If both are present it is best to treat the *Ascaris* infection first, since it is the more likely of the two to migrate. Any non-specific therapy, or (especially) administration of anesthesia can cause worms to migrate, including penetrating the intestinal wall; forcing through the pyloric and cardiac valves of the stomach, thus entering the esophagus, or crawling into the common bile duct. All are potentially dangerous.
- (4) Adult worms may rarely be recovered from the anus, mouth, throat or nose.

4. *Necator americanus* - The New World hookworm
Ancylostoma duodenale - The Old World hookworm



Female

Male

Female Male

Necator americanus adults

Ancylostoma duodenale adults

Hookworm egg



Hookworm filariform larva



Hookworm rhabditiform larva

a. Life cycle

- (1) Under optimal conditions, eggs in fecally contaminated soil develop and hatch within 24 to 48 hours, becoming rhabditiform larvae. Growth and development continue to take place in the soil as the larvae feed on bacteria and organic material and undergo a first molt.

- (2) After about 7 days the worms stop feeding and molt a second time, transforming from the rhabditiform larvae to infective filariform larvae.
- (3) Infective larvae do not feed and can survive for approximately 2 weeks without a host. They usually live in the upper layers of the soil, and when the soil is cool and moist, they climb to the highest point covered by a film of moisture. They extend their bodies into the air and remain waving about in this position until driven down by drying of their surroundings or by heat, or until they come in contact with the skin of a suitable host.
- (4) Infections are acquired when the third stage filariform larvae penetrates the skin of a human.
- (5) Larvae enter the lymphatic system or bloodstream, and travel to the lungs. After maturing in the lungs, the larvae migrate up the trachea to be swallowed and reach the small intestine, where they mature.
- (6) Immature adults attach to the intestinal mucosa by means of their stout mouth parts and suck blood and tissue juices of the host.
- (7) About five weeks after infection, the worms have undergone a final molt to become sexually mature adults. Fertilization occurs, and the females begin to release eggs. Worm life span is about 1 year.

b. Morphology

- (1) Rhabditiform larvae - long buccal cavity, indistinct genital primordium. Filariform larvae have sharp pointed tails.
- (2) Adults - males: 7 to 11 mm long with a copulatory bursa; females: 8 to 15 mm long.
- (3) Eggs - 55 to 70 x 35 to 40 microns; very thin shell; usually seen in the 8 - 32 stage of cleavage.

b. Diagnosis

- (1) Recovery and identification of eggs (rarely larvae) in the feces.
- (2) Cannot differentiate Hookworm species by egg appearance.
- (3) To determine if a significant infection is present make a saline direct smear, count the number of eggs on the entire smear, if there are less than 5 eggs per smear it is indicative of a light infection (usually no anemia), 20 or more eggs per smear is clinically significant, and 100 or more per smear is indicative of a very heavy infection.

c. Major pathology and symptoms

- (1) After repeated infections severe allergic itching at site of penetration may occur (cutaneous larva migrans).
- (2) During migration through the lungs, patients may experience a sore throat and / or bloody sputum.
- (3) Heavy intestinal infections may result in enteritis, anemia, weakness, and loss of strength due to the anemia.
- (4) Chronic intestinal infections may experience slight anemia, weakness, weight loss and non-specific gastro-intestinal symptoms.
- (5) A combination of nutritional and disease factors are commonly seen in endemic areas. Children may exhibit stunted growth and intellectual development.

d. Distribution

- (1) *N. americanus* - North America and Africa
- (2) *A. duodenale* - Europe and south America

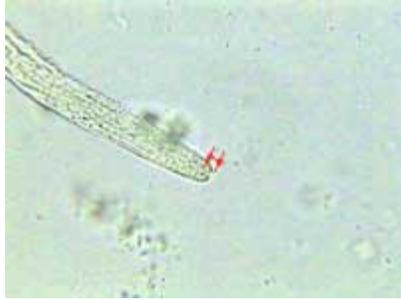
e. Notes

- (1) Moist, warm regions of the world where the skin frequently contacts the soil is optimal for heavy infections, especially in areas of poor sanitation.
- (2) Delayed fecal examination can result in eggs hatching. The technologist must then differentiate the larvae from those of *Strongyloides stercoralis*.
- (3) In heavy infections, blood loss can be up to 100 milliliters/day. In these instances, one must provide dietary and iron support with drug treatment.

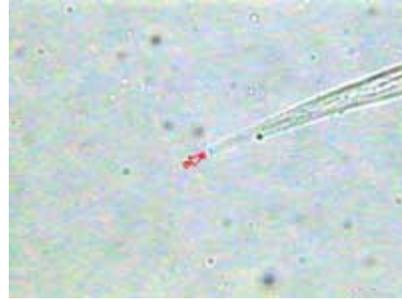
5. *Strongyloides stercoralis* - The Threadworm



Strongyloides stercoralis rhabditiform larva *Strongyloides stercoralis* filariform larva



Buccal cavity of rhabditiform larva



Notch in tail of filariform larva

a. Life cycle (very complex)

- (1) Infective third stage filariform larvae penetrate skin, and enter the lymphatic system or bloodstream.
- (2) Larvae migrate to the lungs, break out of lung capillaries into alveoli.
- (3) After maturation, larvae travel up to the pharynx, are swallowed, and return to the intestine.
- (4) Larvae mature to adults and attach to the mucosa of the small intestine.
- (5) Parthogenetic Females only - no parasitic males.
 - (a) Females are believed to be capable of unisexual reproduction, no fertilization required.
 - (b) Produce viable eggs.
- (6) Eggs hatch in mucosa – larvae have 3 pathways of existence
 - (a) First stage rhabditiform larvae (non-infectious) are passed in feces, live in the soil, mature into a free-living adult males and females, which produce eggs. Within 2-3 days rhabditiform larvae hatch from these eggs. Within 24 hours these develop into other free-living adults or molt to become infective filariform larvae. They can live up to two weeks in soil. Filariform larvae must penetrate skin. This free-living cycle can extend for long periods.
 - (b) Rhabditiform larvae live in soil and develop into infective filariform larvae which penetrate the skin.
 - (c) First stage larvae develop into infective stage larvae in the intestine (autoinfection) and causes hyperinfection. This is a problem, especially, in the immunosuppressed.

b. Morphology

- (1) Rhabditiform larvae - short buccal cavity; large, prominent genital primordium.

- (2) Filariform larvae - tail has a notch in it, in contrast with the filariform larva of hookworms.
- (3) Must be able to differentiate these from hookworm larvae.
- (4) Eggs hatch in the intestine and are not usually passed in stool specimens. Eggs may be found in duodenal drainage fluid and will appear hookworm-like. These eggs are invariably embryonated, however, in contrast to those of the hookworms.

b. Diagnosis

- (1) Recovery and identification of larvae in the feces.
- (2) Recovery and identification of eggs in duodenal drainage.

c. Major pathology and symptoms

- (1) Skin – allergic reactions; raised, itchy, red blotches at the site of larval penetration.
- (2) Lungs - pneumonia.
- (3) Intestinal - abdominal pain, diarrhea/constipation, vomiting, weight loss, variable anemia, increased eosinophil count.
- (4) Light infections are usually asymptomatic.
- (5) Heavy infection - bowel becomes edematous and congested.
- (6) Death occurs in immunosuppressed patients due to heavy autoinfection and larval migration throughout the body with bacterial infections secondary to larval spread and intestinal leakage.

c. Distribution - warm areas worldwide, similar to hookworm.

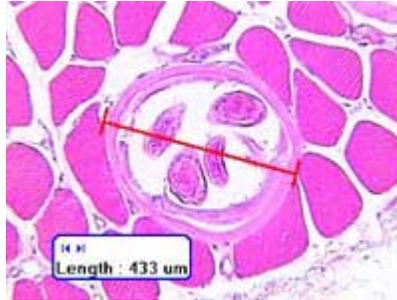
d. Notes

- (1) Parasitic female is parthenogenetic; no parasitic males exist.
- (2) While hookworm infection dies out over a period of some years after the patient has moved from an endemic area, strongyloidiasis may persist for years, regardless of change in location, due to autoinfection (internal infection).
- (3) Difficult to treat.
 - (a) Thiabendazole – the drug of choice, but it has side effects.
 - (b) Mebendazole - is better tolerated, but less effective.
 - (c) Albendazole - is tolerated fairly well, not FDA approved.

(d) Ivermectin has been found effective.

(4) In cases with severe diarrhea, embryonated eggs may be present in stool specimens. These must be differentiated from hookworm eggs. *Strongyloides* eggs contain well-developed larvae. Hookworm eggs do not have well developed larvae until passed from the body and for one to two weeks in the soil.

C. Blood and Tissue-Dwelling Nematodes



Trichinella spiralis larva encysted in muscle

1. *Trichinella spiralis* - trichinosis

a. Overview - due to meat inspection programs, this is not very prevalent in U.S.A. any longer. When seen, it is usually due to home butchering and meat preparation (smoking meat doesn't kill larvae unless a temperature of 150 degrees centigrade is reached). Outbreaks are most commonly associated with pork, but any meat-eating mammal has a potential for involvement.

(1) Subspecies

- (a) *Trichinella spiralis spiralis* - seen in temperate regions, acquired from domestic pigs, source of majority of infections in U.S.A.
- (b) *Trichinella spiralis nativa* seen in arctic regions, acquired by eating undercooked bear and walrus meat.
- (c) *Trichinella spiralis nelsoni* is acquired from wild pigs in southern Europe and Africa.

b. *Trichinella spiralis* is a parasite of carnivorous mammals. It is especially common in rats and swine fed uncooked garbage and slaughterhouse scraps.

c. Human infections occur most often as a result of consumption of raw or undercooked pork.

d. Life cycle

(1) Infective stage larvae are ingested in meat products (encysted larvae are in the muscle of infected and undercooked pork, deer, walrus, bear).

- (2) Tissue is digested, larvae are freed in the intestine. They mature into adult males and females.
 - (3) Female in the deep mucosa releases larvae. These disseminate throughout the body via the bloodstream. (No eggs)
 - (4) Larvae encyst in striated muscle.
- e. Morphology - females are 3.5 mm long; males measure 1.5 mm long; larvae measure 100 microns long.
- f. Diagnosis
- (1) Identification of encysted larvae in muscle biopsy. Muscles richest in blood supply are preferred for biopsy - larvae spread via bloodstream.
 - (2) Serology becomes positive 3 to 4 weeks after infection.
 - (3) A history of eating undercooked pork, deer, walrus or bear is indicative whenever appropriate symptoms appear.
- g. Major pathology and symptoms
- (1) Fever, muscle pain, bilateral periorbital edema, and increased eosinophil count warrant a presumptive diagnosis.
 - (2) Intestinal phase – patients experience small intestinal edema and inflammation, nausea, vomiting, abdominal pain, diarrhea, headache and fever.
 - (3) Migrational phase - high fever (104 degrees), blurred vision, edema, cough and pleural pains lasting one month with heavy infection. Death can occur during this phase in 4th - 8th week following infection.
 - (4) Muscle phase – acute, local inflammation with edema and pain of the musculature.
- h. Distribution
- (1) Cosmopolitan in distribution; worldwide among meat-eating populations, highest incidence is now reported from China, still common in Spain, France, Italy, and Yugoslavia.
 - (2) Prevalence in U.S.A. is about 4% based on autopsy studies.
 - (3) Only about 100 cases are recognized and reported per year in the U.S.A.

2. *Dracunculus medinensis* – The Guinea Worm



Blister containing *Dracunculus medinensis*



Adult *Dracunculus* emerging from broken blister

a. Overview

- (1) An important parasite in the Middle East (Saudi Arabia, Iran, Yemen), in central India and Pakistan.
- (2) It is found in Africa in the Sudan and scattered through central equatorial regions, and on its west coast.
- (3) It is believed to no longer occur in the Western Hemisphere, except in reservoir hosts, but once was found in the West Indies, Guyana, Suriname, French Guiana, and Brazil. A morphologically similar worm, *Dracunculus insignis*, is found in raccoons along the Brazos River through the Bryan-College Station area. There have been no reports of human involvement.
- (4) Sometimes classified with the filarial worms, but *Dracunculus* is not a true filaria, as the larvae have a well-developed digestive tract and are never found in the blood or tissues of the host. They are discharged directly into water.

b. Life cycle

- (1) Infective stage exists in a water flea (copepod – the intermediate host).
- (2) Humans become infected by drinking water containing the infected copepod.
- (3) Larvae penetrate the digestive tract to enter the deep connective tissues where they mature in about 1 year.
- (4) Females migrate to the subcutaneous tissue (usually the skin of the extremities), once they become gravid. A papule is produced at the site. This becomes vesicular and ulcerates, exposing the worm.
- (5) Females release larvae which leave the human through ruptured blisters on the skin. Cool water stimulates release of the larvae.
- (6) The larvae enter the water and are ingested by copepods.

c. Morphology

- (1) Males measure 40mm in length; females measure 800mm in length. This is the largest adult nematode parasite of humans. Females have been reported to measure a meter or more in length, although averaging somewhat less.

d. Diagnosis

- (1) Visual observation of skin blister. The worm's serpentine presence beneath skin can be seen.
- (2) Induced release of larvae from the skin ulcer when cold water is applied.

e. Major pathology and symptoms

- (1) Mild allergic symptoms such as urticaria during the migration phase.
- (2) A papule (near the head of the worm) develops into a blister with localized erythema and tenderness.
- (3) Generalized symptoms may include nausea, vomiting, diarrhea, and possibly asthma attacks. These usually disappear after the blister has ulcerated allowing drainage of fluids and initial discharge of larvae.
- (4) Additional complications include secondary bacterial infections, severe disabilities lasting an average of 6 weeks, and permanent damage to joints.

f. Distribution - Middle East and Africa

3. The Filariae

a. Overview

- b. The Filariae are long thread-like nematodes. Eight species inhabit portions of the human subcutaneous tissues and lymphatic system.
- c. Adults of all species are parasites of vertebrate hosts.
- d. Female worms produce eggs. The eggs modify, becoming elongated and worm-like in appearance and adapting to life within the vascular system.
- e. These modified eggs, referred to as microfilariae, are capable of living a long time in the vertebrate host, but cannot develop further until ingested by an intermediate host and vector, an insect.
- f. The microfilariae transform into infective larvae in the insect and are deposited in the skin of the next suitable host when the insect next takes a blood meal.

g. General life cycle

- (1) Human infection is acquired when infective larvae enter the skin at the arthropod's feeding site.
- (2) Larval migration and development takes place in the tissue.
- (3) Adults are in various tissues (species determined) and mature and produce microfilariae.

h. *Wuchereria bancrofti* – a blood & lymphatic dweller. The infection often results in elephantiasis. Commonly called “Bancroft's Filariasis.”



Wuchereria bancrofti microfilaria in blood smear

i. *Brugia malayi* – also a blood & lymphatic dweller. The infection can cause elephantiasis, but is not as disfiguring or as common as with *Wuchereria bancrofti*. The disease is often referred to as “Malayan filariasis.”



Brugia malayi microfilariae in blood smear

j. *Onchocerca volvulus* – the “blinding filarial.” Infections involve the dermis and subcutaneous tissues, where adults gather within nodules.



Onchocerca volvulus microfilariae in skin snip

- k. *Loa loa* – the “eyeworm.” Infections involve the dermis and subcutaneous tissues (Calabar swelling).



Loa loa microfilariae in blood smear

l. Vectors:

- (1) *Wuchereria bancrofti* – Culex, Aedes, & Anopheles mosquitoes.
- (2) *Brugia malayi* – Mansonia, Anopheles & Aedes mosquitoes
- (3) *Loa loa* – Crysops, a large mango fly with biting mouthparts
- (4) *Onchocerca volvulus* – Simulium flies (blackfly, or buffalo gnat)

m. Morphology & Diagnosis

- (1) *W. bancrofti*, *B. malayi*, and *Loa loa* are diagnosed by detection and identification of microfilaria in stained blood smears.
- (2) *Wuchereria bancrofti* - demonstrates a marked circadian migration, best seen at night after 10 P.M. Microfilariae are sheathed, and the nuclear column does not extend to tip of tail.
- (3) *Brugia malayi* - same as for *Wuchereria*. Microfilariae are sheathed, nuclear column extends to tip of tail with two nuclei near end of tail, one in a swelling just short of tail's end, the other in the end of the tail.
- (4) *Loa loa* - diagnosis is usually made by clinical symptoms, but if laboratory confirmation is required, blood should be drawn between 11 am & 1 pm. Microfilariae are sheathed, nuclear column extends to tip of tail.
- (5) *Onchocerca volvulus* - microfilariae are found in skin scrapings around nodules. Microfilariae not sheathed – found only in skin, not in the blood stream.

n. Major pathology and symptoms

- (1) *W. bancrofti* - Swelling due to allergic reaction occurring around the worms produces obstruction. Each individual reacts differently. Very few develop elephantiasis, but in some this is extensive.
- (2) *B. malayi* - same as above. More often asymptomatic.
- (3) *O. volvulus* – characterized by fibrotic nodules encapsulating adults, especially on the trunk in Africa and characteristically on the head in central America. Progressive allergic skin rash. Blindness occurs due to the presence of microfilariae in all ocular structures. This parasite is a major cause of blindness in Africa. Control is difficult because Simulium flies breed in running water.

- (4) *Loa loa* – Infections cause a localized subcutaneous edema, particularly around the eye, because of larval migration and death in capillaries. Living adults cause no inflammation; dying adults induce granulomatous reactions.