This chapter is designed as an overview, highlighting potentially severe parasitic infections, based on disease of concern risk analysis. Further and more in depth information can be found in Cheeseborough (2005, in press) and at several websites (refer to the reference section). Life cycle diagrams in this chapter were sourced from http://www.biosci.ohio-state.edu/~parasite

General Guidelines
- All these diseases are zoonoses and must be managed as such
- Routinely check stools every 3 months. See section 3.17 for the formol-ether concentration technique for gastrointestinal parasites.
- Recheck 7-10 days after treatment to monitor effectiveness.
- Keep microscopy equipment, including slides, away from humidity.

A. GASTROINTESTINAL NEMATODES

1. HOOKworms – ANCYLOSTOMA SPP, NECATOR SPP.
   - Heavy infection can cause anaemia – especially in juveniles
   - Causes bloody diarrhoea
   - Primarily a human pathogen
   - Direct life cycle – no intermediate host - Refer to Life Cycle diagram (figure 4) for more details
   - 1 week is required for infective larvae to develop from eggs in the environment
   - Strict Hygiene and vector control are vital to prevent spread
   - Diagnosis based on finding eggs in the stool (figure 1 and 2) BUT larvae can hatch quickly in high ambient temperatures and should be differentiated from strongyloides L1 larvae. Hookworm L1 larvae have a long buccal cavity (figure 3).
   - Ivermectin, mebendazole, albendazole or possibly levamisole at standard doses can be used for treatment. Treatment for concurrent anaemia may also be required.

Hookworm eggs are standard ‘strongyle’ eggs – oval, thin shelled, relatively large (65 x 40μm). When identifying eggs, you cannot distinguish different hookworm species from each other, or from other strongyle infections, such as Oesophagostomum spp. Treatment however is the same.
Figure 1. Hookworm spp egg

Figure 2. Larvated hookworm egg

Figure 3. Buccal cavity (green line), L1 larva - hookworm
2. ASCARIDS

- Potentially can induce sudden death due to blockage – individual worms can grow up to 18 inches long (figure 5).
- Infection is frequently asymptomatic
- Eggs adhere to many surfaces and become mixed in soil and dust
- Primarily a human pathogen
- Direct life cycle (figure 6)
- Strict hygiene
- Vermin control
- Piperazine, ivermectin, mebendazole, pyrantel, fenbendazole, albendazole are all effective wormers.
- Egg 50-70 x 30-50um. yellow brown colour, “bumpy” coat (may be decorticated)
Figure 6. Ascarid life cycle

Figure 7. Ascarid eggs. Left: infertile (90um) Right: fertile (60-75um)

Figure 8. Decorticated, fertile (note double wall)
3. TRICHURIS (WHIPWORM)

- Heavy infections result in colitis (+/- secondary bacteria and/or protozoa) (figure 9)
- Direct life cycle (figure 10)
- Strict hygiene. The eggs are highly susceptible to desiccation, so keep areas as dry as possible
- Pest control is important to minimise spread
- Anthelmintics: mebendazole, albendazole, ivermectin, flubendazole, pyrantel/oxantel combo.
- Egg: must differentiate from Capillaria (has non-protruding polar plugs, size 45x21um) (figure 12)

![Figure 9. Colitis induced by a heavy Trichuris infection.](image)

![Figure 10. Trichuris life cycle](image)
Figure 11. Adult Trichuris (whipworm)

Figure 12. Trichuris Eggs, 50x25μm
4. ENTEROBIUS SPP (PINWORMS)

- Infection is often asymptomatic but anal pruritis also common
- Fatal cases reported in chimpanzees
- Direct life cycle (figure 16).
- Ova can spread in air/dust
- Strict hygiene essential
- Human to animal spread easy and common
- Effective anthelmintics: Mebendazole, Albendazole, pyrantel

Figure 13 Pinworm Male

Figure 14 Pinworm Female 8-13mm

Figure 15. Pinworm Egg, oval, larvated flattened on one side. 55x30µm
5. STRONGYLOIDES SPP

- Clinical signs highly variable. Often asymptomatic - especially in adults. Mucoid or Haemorrhagic diarrhoea. In chronic infections - progressive weight loss and weakness (no diarrhoea)
- Death is usually a result of pneumonia and peritonitis, due to a sudden and massive increase in L1 larval migration, due to concurrent immunosuppression.
- S.stercoralis in humans and apes
- S.fulleborni in chimps, baboons, guenons
- Various species in monkeys
- Complex life cycle - Parasitic and free living (figure 24)
- Strict hygiene – maintain enclosures as dry as possible to prevent free living stages.
- Anthelmintic possibilities: Ivermectin, thiabendazole, mebendazole, levamisole, pyrantel. A single dose of ivermectin or albendazole, or multiple doses of thiabendazole may give good results against the intestinal stages, but are ineffective against parental stages. Multiple treatments with ivermectin at a dose rate of 0.4mg/kg may be necessary to control this worm.
- Diagnosis: L1 larva with short buccal cavity, figure 17, (larvated egg if *S.fuelleborni*), culture for characteristic L3 larvae (unsheathed, size around 600μm)

![Figure 18. *S.fuelleborni* egg 50x35μm](image)

![Figure 19. *Strongyloides* spp L3 larva, notched posterior end](image)
Figure 20 and 21. Free-living Strongyloides spp. adult male, note curved posterior end

Figure 22 and 23. Adult female Strongyloides fuelleborni, note “waist” and vulval lips

Figure 24. Strongyloides spp. Life cycle

*Oesophagostomum bifurcum* is the most-common species infecting humans in Africa – especially in Togo and Ghana. This nematode has been the confirmed cause of death in drills and gorillas, and has been found in chimpanzees, in PASA sanctuaries. The majority of this section comes from the CDC website, as most of the photos from PASA show pathological changes, rather than the worms themselves. This worm is primarily a parasite of monkeys. Figure 25 shows the life cycle.

**Figure 25. Oesophagostomum life cycle.** Common livestock such as sheep, goats, and swine, as well as non-human primates, are the usual definitive hosts for *Oesophagostomum* spp., but other animals, including humans and cattle, may also serve as definitive hosts. Eggs are shed in the feces of the definitive host 1, and may be indistinguishable from the eggs of *Necator* and *Ancylostoma*. Eggs hatch into rhabditiform (L1) larvae in the environment 2, given appropriate temperature and level of humidity. In the environment, the larvae will undergo two molts and become infective filariform (L3) larvae 3. Worms can go from eggs to L3 larvae in a matter of a few days, given appropriate environmental conditions. Definitive hosts become infected after ingesting infective L3 larvae 4. After ingestion, L3 larvae burrow into the submucosa of the large or small intestine and induce cysts. Within these cysts, the larvae molt and become L4 larvae. These L4 larvae migrate back to the lumen of the large intestine, where they molt into adults 5. Eggs appear in the feces of the definitive host about a month after ingestion of infective L3 larvae.

Clinical signs include:

- Acute abdomen is the most-common manifestation in humans, mimicking an appendicitis.
- A low-grade fever and tenderness in the lower-right quadrant are the most-common symptoms; vomiting, anorexia, and diarrhea are less-common.
- Intestinal obstruction may also occur, mimicking a hernia.
- In rare instances, Oesophagostomum spp. will perforate the bowel wall, causing purulent peritonitis or migrate to the skin, producing cutaneous nodules. These nodules have been found in chimpanzees.

As a typical strongyle, like hookworm, diagnosis can be difficult based on worm egg examination alone, as they look the same. Eggs tend to be shed in greater numbers during cases of oesophagostomiasis than hookworm infection, however. Finding an intact worm during surgery or in a biopsy specimen can provide a definitive diagnosis.

The eggs of O. bifurcum measure 60-75 μm long by 35-40 μm wide (Figure 26). Eggs are often in a later stage of cleavage (increased cellular bifurcation within the egg), than hookworm species when shed in faeces.

![Figure 26 A and B. Eggs of Oesophagostomum sp. in an unstained wet mount of stool.](image)

Adults of Oesophagostomum spp. are bursate nematodes, related to and morphologically-similar to, the hookworms. Females measure 1.5-3.0 cm in length; males are smaller. In both sexes, the anterior end has a cephalic inflation or vesicle, a transverse cephalic groove, and an oral opening guarded by external and internal leaf crowns (corona radiata) (Fig 27 - 29). The posterior end of the female is short and pointed; the male possesses a symmetrical bursa and paired, equal spicules. Adults reside in the large intestine of the definitive host (Hookworm in the small intestine).
Figure 27 C and D. C: Adult of Oesophagostomum sp. D: Higher magnification of the anterior end of the specimen in Figure C. Note the presence of the cephalic vesicle (CV), cephalic groove (CG) and esophagus (ES).

Figure 28 E and F. E: Higher magnification of the anterior end of the specimen in Figures 27. Note the presence of the cephalic vesicle (CV) and corona radiata (CR). F: Posterior end of a female Oesophagostomum sp., showing the pointed tail.
Treatment is usually limited to the surgical removal of adult worms from tissue. Albendazole has been shown to be the most effective antihelminthic drug for the removal of worms from the lumen of the large intestine.
B. FILARIASIS

- Intermediate host – blood sucking insects
- In chimps, microfilariae remain in the dermis – *Mansonella streptocerca/ rodhaini* (in other species, because of the life cycle periodicity check blood for microfilaria at night).
- Diagnosis thick blood film (20µl drop) or membrane filtration (3.0µm pore)
- Usually asymptomatic – occasional skin disease
- Potential treatment - Diethylcarbamazine - dangerous

*Figure 31. Filariasis life cycle*

*Figure 32. W. bancrofti*

- 275-300 x 8-10um
- Sheathed, nuclei do NOT extend to tail tip
- Nocturnal periodicity
- In blood

*Figure 33. M. streptocerca*

- 180-240 x 5um
- Unsheathed, nuclei extend to tail tip which is often hooked
- In skin

*Parasites and Parasitological Resources*
C. SCHISTOSOMIASIS

- **Intestinal disease** caused by *S. mansoni, S. intercalatum, S. japonicum*—paired, mature adult flukes in venule of rectum and lower L.I.
- **Urinary disease** caused by *S. haematobium*—paired, mature adult flukes in veins surrounding bladder and may be found in veins of liver and rectum
- **Intermediate host:** snails
- **Transmission:** contact with water infected with cercariae. Potentially a big problem for staff in endemic areas.
- **Diagnosis:** faecal concentration technique usually required for intestinal schistosomes, membrane filtration (12.0μm pore) of urine.
- **Treatment** – Praziquantel is effective against all types. Oxamnique is cheaper, but only effective against *S. mansoni*. Metrifonate can be used for *S. haematobium*. Total cure is unfeasible in endemic areas due to the high rate of reinfection.

![The Life Cycle of Schistosoma spp.](image)

*Figure 34. Schistosoma life cycle*

![S. haematobium, terminal spine, 145 x 55μm](image)

*Figure 35. S. haematobium, terminal spine, 145 x 55μm*
Figure 36. *S. mansoni*, lateral spine, 150 x 60μm

Figure 37. Eggs containing miracidia (and 1 hatched in seminal fluid)
D. CESTODES

- Taenia spp. eggs are 47-77μm in diameter. They are relatively heavy, making them difficult to identify with normal flotation techniques.
- Strict hygiene with effective rodent and insect prevention and control.
- Adult tapeworms are not a big problem but due to their size, there is the possibility of intestinal blockage.
- Diagnosis of species by examination of gravid proglottid: size and number of uterine branches.
- Treatment: Praziquantel is the drug of choice.

Figure 38. Taenia spp. egg.

Proglottids

<table>
<thead>
<tr>
<th>Proglottid Type</th>
<th>Size</th>
<th>Eggs/Proglottid</th>
<th>Main Branches</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. solium</td>
<td>12x10 mm</td>
<td>8-10 passed/day</td>
<td>7-13 main branches</td>
</tr>
<tr>
<td>T. saginata</td>
<td>12x10 mm</td>
<td>5-8 passed/day</td>
<td>15-32 (20-23) main branches</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Proglottid Type</th>
<th>Size</th>
<th>Eggs/Proglottid</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. solium</td>
<td>12x10 mm</td>
<td>Up to 90,000</td>
</tr>
<tr>
<td>T. saginata</td>
<td>12x10 mm</td>
<td>80-100,000</td>
</tr>
</tbody>
</table>
**Figure 39.** Unstained *T. saginata* proglottid

**Figure 40.** *Taenia* spp. life cycle.
1. Entamoeba histolytica/ dispar (Amoebic dysentery)

- Opportunistic pathogen – often in the gut in low numbers.
- Only becomes pathogenic if it invades the gut mucosa, so infection is often asymptomatic.
- Clinically affected animals may have mild intermittent diarrhoea through to severe diarrhoea that may be dysenteric or catarrhal. They may be lethargic, show general weakness, dehydration, gradual weight loss, anorexia and vomiting.
- The infective cyst is resistant to drying and many disinfectants.
- Transmission is via food, water, insects and fomites, by ingestion (life cycle fig 49).
- In stool samples cysts of *E. histolytica* and *E. dispar* are identical (up to 4 nuclei around 12.0μm) those of *E.coli* are very similar but have more than 4 nuclei. Only *E. histolytica* can be dangerous – remember all are normal gut flora.
- As is usually a secondary invader, the immune status of the animal is important. So, if identified or implicated in diarrhoea, will very often be another cause for the illness. HOWEVER, if isolated, with clinical signs – will require treatment.
- Samples (mucoid/bloody, fluid) need to be less than 30 minutes old to identify possible *E.histolytica* trophozoites, as drying and low temperatures will kill motile trophozoites. Only *E.histolytica* trophozoites contain ingested RBC’s which is the diagnostic feature (figure 41, 46, 47).
- This is a human pathogen so check in contact staff
- Strict hygiene and vector control must be observed.
- Abscessation is possible, especially in the liver, if it invades beyond the gut.
- Signs to death – 7-130 days
- Metronidazole, Tinidazole, paramomycin and secnidazole are the drugs of choice.

*Figure 41. E.histolytica trophozoites (note ingested RBC’s)*
Figure 42 – 44. *Entamoeba histolytica/dispar* cysts, size range 10-15μm, contain up to 4 nuclei

Figure 45. Non pathogenic *Entamoeba coli* cyst, 10-30 μm, mature cyst has > 4 nuclei

Figure 46 – 47. *E.histolytica* trophozoites, 8-30μm, note ingested RBC’s. Amoebae show active, directional, amoeboid movement; single nucleus with central karyosome.
Figure 48. Non pathogenic Entamoeba coli trophozoite, 15-50µm, sluggish, rarely directional movement, nucleus has eccentric karyosome.

Figure 49. Entamoeba histolytica life cycle.
2. BALANTIDIUM COLI

- Ciliate found in the caecum of primates and pigs.

Figure 50. *B. coli* trophozoite, 50-200 x 40-70μm, large macronucleus and contractile vacuoles may be seen.

- Clinically signs include weight loss, anorexia, muscle weakness, lethargy, watery diarrhoea, dehydration, tenesmus, rectal prolapsed. Clinical signs are often self limiting.
- Primary pathogenicity not confirmed – (opportunistic pathogens) however, infection in gorillas can progress rapidly to death.
- Strict hygiene and vector control must be observed.
- Avoid sudden diet changes, which can change the gut flora, potentially leading to an overgrowth of *B. coli*.
- Treatment: Metronidazole, paramomycin, tinidazole, doxycycline.

Figure 51. *B. coli* cyst, trichrome, 50 x 70 μm source dpd.cdc.gov
3. GIARDIA

- Usually chronic intermittent diarrhoea and weight loss.
- Faeces are poorly formed, rather than watery, and rarely dysenteric. Vomiting is rarely involved. Bouts of diarrhoea (with abdominal pain) of several days duration are common, often with several weeks between bouts.
- Confirm infection with cysts/ trophozoites in faeces
- Cysts oval, 8-15 x 6μm, 4 nuclei, axoneme and remains of flagellae may be seen.
- Trophozoites 12-15 x 5-9μm, rapid tumbling movement (falling leaf), sucking disc on ventral surface, 4 pair flagellae, 2 axonemes & 2 nuceli
- Treatment: Although disease is often self limiting, treatment is recommended, especially in juveniles. Metronidazole resistance is increasing. Tinidazole is preferred, but compliance will be problematic due to the bad taste of both of these medications.
Figure 54 and 55. Giardia cysts, stained with trichrome (Left) and iodine (Right)

Figure 56. Giardia life cycle

THE LIFE CYCLE OF GIARDIA LAMBLIA
(the causative agent of giardiasis)

Cysts ingested with contaminated water or food

Cysts "excyst," and trophozoites colonize the small intestine

Cysts passed in feces

Reservoir host(s)
(Beavers are often implicated as potential reservoirs)

(Parasites and Parasitological Resources)
4. BLASTOCYSTIS HOMINIS

- Infect epithelial cells of digestive tract, multiply asexually.
- Size range 6-40 um (frequently 10um)
- Strong association with *D. fragilis*
5. DIENTAMOEBA FRAGILIS

- A flagellate, trophozoite only known stage, 5-15μm
- Need to examine stained preparations
- Uni or bi-nucleate- characteristic diffuse nucleus
- Associated with Irritable Bowel Syndrome?
- Associated with severe gastrointestinal disease in gorillas.

Figure 61. Proposed life cycle for Dientamoeba fragilis

Figure 62 – 64. Various stained preparations of D. Fragilis. Figure 58 is from a gorilla (Limbe). Fields stain.