# Parasitic Organisms

## NEMATODES – ROUNDWORMS

| Organism | Description | Epidemiology/Transmission | Pathogenicity | Symptoms |
|---|---|---|---|---|
| *Ancylostoma -Necator*<br><br>*Ancylostoma duodenale*<br><br>*Necator americanus* | Hookworms<br><br>Soil-transmitted nematodes<br><br>(P) | Found in tropical and subtropical climates, as well as in areas where sanitation and hygiene are poor.[1]<br><br>Infection occurs when individuals come into contact with soil containing fecal matter of infected hosts.[2] | *Necator* can only be transmitted through penetration of the skin, whereas *Ancylostoma* can be transmitted through the skin and orally.<br><br>*Necator* attaches to the intestinal mucosa and feeds on host mucosa and blood.[2]<br><br>*Ancylostoma* eggs pass from the host's stool to soil. Larvae can penetrate the skin, enter the lymphatics, and migrate to heart and lungs.[3] | Some are asymptomatic, though a heavy burden is associated with anemia, fever, diarrhea, nausea, vomiting, rash, and abdominal pain.[2]<br><br>During the invasion stages, local skin irritation, elevated ridges due to tunneling, and rash lesions are seen.[3]<br><br>*Ancylostoma* and *Necator* are associated with iron deficiency anemia.[1,2] |
| *Ascaris lumbricoides* | Soil-transmitted nematode<br><br>Most common human worm infection<br><br>(P) | Common in Sub-Saharan Africa, South America, Asia, and the Western Pacific. In non-endemic areas, infection occurs in immigrants and travelers.<br><br>It is associated with poor personal hygiene, crowding, poor sanitation, and places where human feces are used as fertilizer.<br><br>Transmission is via the fecal-oral route.[4] | *Ascaris* eggs attach to the small intestinal mucosa. Larvae migrate via the portal circulation into the pulmonary circuit, to the alveoli, causing a pneumonitis-like illness. They are coughed up and enter back into the GI tract, causing obstructive symptoms.[5] | Most patients are asymptomatic or have only mild abdominal discomfort, nausea, dyspepsia, or loss of appetite.<br><br>Complications include obstruction, appendicitis, right upper quadrant pain, and biliary colic.[4]<br><br>Intestinal ascariasis can mimic intestinal obstruction, bowel infarction, intussusception, and volvulus. Hepatic and pancreatic ascariasis can mimic biliary colic, acute acalculous cholecystitis, hepatic abscess, acute pancreatitis, and ascending cholangitis. Appendicular ascariasis can mimic appendicular colic, appendicitis, appendicular gangrene. Gastric ascariasis can mimic pyloric obstruction.[6] |
| *Capillaria philippinensis* | Fish-borne nematode<br><br>(P) | Although rare in the US, it is more common in Asia (Thailand and the Philippines)[4]<br><br>Infection occurs from eating raw or undercooked fish containing larvae. | Ingested larvae reside in the human small intestine, where the female deposits eggs, which then develop, causing autoinfection and hyperinfection.[4] | Diarrhea, anorexia, malaise, and vomiting.[4]<br><br>Capillariasis can mimic IBD and other causes of protein losing enteropathy.[6] |
| *Enterobius vermicularis* | Pinworm<br><br>The most common worm infection in children ages 5-10 in the US<br><br>(P) | Compared to other intestinal parasites, the transmission of pinworm is limited because their eggs are unable to survive in the environment. The main routes of infection are autoinfection from eggs or larvae deposited on the anus, contamination from bed sheets, clothing, door handles, and inhalation of eggs from | Eggs are deposited around the anus by the worm. Autoinfection occurs due to scratching the perineal area, then thumb-sucking or nail-biting. Pinworms reside in the intestine but can migrate to distant organs.[4] | Some infections are asymptomatic.<br><br>Symptoms may include itching and irritation. Occasional migration of the worm to distant organs can cause dysuria, vaginal discharge, enuresis, and peritoneal granulomas.[4]<br><br>Enterobiasis can mimic hemorrhoids and IBD.[6] |

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|---|---|---|---|---|
| | | hands, bed mattresses, or dust. As a result, infections tend to be limited to families and individuals in close proximity, like nurseries and boarding schools.[7]<br><br>Spread by overcrowding and poor hygiene. | | |
| *Strongyloides stercoralis* | Soil-transmitted nematode<br><br>(P) | Endemic to the tropics and temperate subtropics where poor sanitation facilitates fecal contamination. Also found in poorer areas of the US: Appalachian mountain communities, Kentucky, and rural Tennessee.[1]<br><br>Transmission is from contaminated soil.[8] | Infection occurs from skin penetration where the organism then travels systemically (blood, lung, GI tract).[8]<br><br>*Strongyloides stercoralis* is unique among nematodes infections in humans because larvae passing in the feces can give rise to a free-living generation of worms. The potential for autoinfection exists if larvae attain infectivity while in the host.[4] | Most patients have subclinical or asymptomatic infections. They are commonly chronic and longstanding due to the autoinfective lifecycle.[4]<br><br>Irritation, edema, and urticaria at the site of skin penetration.[8]<br><br>Diarrhea, constipation, abdominal pain, anorexia.<br><br>Dry cough, tracheal irritation, recurrent asthma.[8]<br><br>Strongyloidiasis can mimic IBD and eosinophilic enterocolitis.[6] |
| *Trichuris trichiura* | Whipworm<br><br>Soil-transmitted nematode<br><br>The third most common roundworm in humans[4]<br><br>(P) | Found in areas where human feces is used as fertilizer. Found in the tropics and places with poor sanitation.<br><br>Transmitted via the fecal-oral route.[4] | A human host consumes eggs, sometimes in food. Once the eggs are ingested, the larvae hatch in the small intestine. From there they migrate to the large intestine, where the anterior ends lodge within the mucosa. This leads to cell destruction and activation of the host immune system, recruiting eosinophils, lymphocytes, and plasma cells. This causes the typical symptoms of rectal bleeding and abdominal pain.[9] | Mild infections are usually asymptomatic. Heavy worm burden causes painful defecation with mucus, water, and blood (Trichuris dysentery syndrome). Rectal prolapse is also seen.[9]<br><br>Children develop iron deficiency anemia, growth retardations, and impaired cognitive development.[4]<br><br>Trichuriasis can mimic IBD, bacillary dysentery and acute intestinal amebiasis.[6] |

## CESTODES – TAPEWORMS

| Organism | Description | Epidemiology/Transmission | Pathogenicity | Symptoms |
|---|---|---|---|---|
| *Dipylidium caninum* | Dog (or cat) tapeworm<br><br>(P) | Human infection is rare but can occur in those who kiss or are licked by their infected pets.[10] | Fleas ingest *D. caninum* eggs. Adult fleas are ingested by pets and establish in the small intestine where the eggs develop into the adult tapeworm. The tapeworm sheds proglottids, which are found in the stool. Humans are infected by accidental ingestion of infected dog or cat fleas.[10] | Most are asymptomatic. When present, symptoms include weight loss, colic, and vomiting.[11] |
| *Diphyllobothrium latum* | Fish tapeworm<br><br>(P) | *D. latum* occurs in freshwater fish throughout much of the northern hemisphere; intermediate hosts include bears, pigs, cats, dogs, foxes, and wolves.<br><br>Humans become infected after eating raw or undercooked fish.[11] | After ingestion, in humans the adult helminth can live up to 20 years in the small intestine. It adheres to the mucosa and can eliminate millions of eggs each day. Diagnosis is made by the demonstration of eggs or proglottids in the stool.[12] | Mostly asymptomatic, but signs and symptoms can include nausea, vomiting, diarrhea, abdominal pain, and weight loss.[11]<br><br>Can cause megaloblastic anemia.[6] |
| *Hymenolepis diminuta* | Rat tapeworm<br><br>(P) | Human infection with *H. diminuta* is rare with only a few hundred cases reported, mainly in children.<br>*H. diminuta* is prevalent worldwide in temperate to tropical conditions with poor sanitation.[13]<br><br>*H. diminuta* infection requires an intermediate host (usually rodents, but also insects). Humans become infected by ingesting food contaminated with larvae, or by direct hand contact.[14] | Once ingested, *H. diminuta* grows to adult form and sheds eggs through the stool. It attaches to the mucosal surface of the intestine and grows to approximately 20-50 cm. in length. | Infection is usually asymptomatic, though may cause abdominal pain, diarrhea, and irritability.[14] |
| *Hymenolepis nana* | Dwarf tapeworm<br><br>(P) | *H. nana* is one of the most common parasitic tapeworm infections worldwide, found mainly in children.<br><br>It does not require an intermediary host and can be transmitted human to human, though rodents can also carry *H. nana*.[15]<br><br>It has fecal-oral transmission from food and water in areas of poor sanitation.[16] | *H. nana* eggs are immediately infective when passed through the stool and cannot last more than 10 days in the environment.<br><br>Once ingested, larvae penetrate intestinal villi and develop into adults that measure 15-40 cm. in length. Eggs pass into stool or can reside within the intestinal villi and cause continual autoinfection.[17] | Symptoms include abdominal pain, diarrhea, anorexia, weight loss, malnutrition, and anemia.[18] |

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| *Taenia* spp.<br><br>*Taenia saginata*<br><br>*Taenia solium* | Tapeworm<br><br>(P) | This tapeworm is found in people who have traveled outside of the US where the infection is endemic, or in Latin American immigrants. Locally acquired infections are rare but have been diagnosed in Los Angeles, New York, Chicago, and Oregon.<br><br>Infection occurs upon ingestion of raw or undercooked meat.[4] | Ingested parasite cysts reach the intestine and develop into adult tapeworms, releasing motile segments and/or eggs in the stool.[19]<br><br>One adult tapeworm can expel a minimum of 100,000 eggs per day. The enclosed larvae penetrate the intestinal wall and are transported via the bloodstream to various tissues where they undergo multiple development stages to become cysticerci.[20] | The adult tapeworm stage is relatively innocuous and does not have human pathogenic effects. However, some species' intermediate stages can develop in human brains causing neurocysticercosis, a major cause of neurologic disease in developing countries.<br><br>Cysticercosis can develop in other organs causing intramuscular, ocular, subcutaneous, and spinal cystericoses.[19]<br><br>Taeniasis can mimic IBS.[6] |

## TREMATODES – FLUKES

| Organism | Description | Epidemiology/Transmission | Pathogenicity | Symptoms |
|---|---|---|---|---|
| *Clonorchis* -<br>*Opisthorchis* spp. | Liver flukes<br><br>(P) | *Clonorchis* and *Opisthorchis* infections have been reported in many parts of east Asia, Thailand, Laos, Cambodia, and Japan.[21]<br><br>These flukes live in freshwater snails and fish. Humans are infected by eating raw or partially cooked, infected fish. | Adult flukes attach to bile ducts where they feed for as long as 10-30 years, resulting in chronic inflammation, epithelial hyperplasia, fibrosis, and granuloma.[22]<br><br>Both are classified as Group 1 carcinogens. Infection by these parasites is frequently asymptomatic and rarely diagnosed during early exposure. Persistent infection is associated with parasite-associated cancer. The mechanism of this transformation is yet to be fully defined.[23] | Early infection is often asymptomatic. Chronic infection is associated with cholangitis, obstructive jaundice, biliary fibrosis, cholecystitis, and cholangiocarcinoma.[24] |
| *Fasciola* spp.-<br>*Fasciolopsis buski* ova | Plant-borne intestinal fluke<br><br>*F. buski* is known as the giant intestinal fluke, and is one of the largest flukes to infect humans.[25]<br><br>*F. hepatica* is a liver fluke.<br><br>(P) | Largely confined to Asian countries, including China.[25]<br><br>Humans are infected by ingesting eggs adhering to the surface of edible water plants.[25] | After ingestion, gastric juices aid the release of the worm. The worm migrates to the small intestine, and produces eggs, which are passed in feces.[26] | Light infections are often asymptomatic. Moderate to heavy infection causes abdominal pain, diarrhea, nausea, vomiting, and fever. Extensive intestinal inflammation, erosions, ulceration, abscess, and hemorrhage are possible.[26] |
| *Heterophyes* -<br>*Metagonimus* ova | Fish-borne intestinal fluke<br><br>(P) | These are mostly seen in Far East and Asian countries.[27]<br><br>These flukes are exclusively fish-borne and are contracted by humans by ingesting raw or improperly cooked freshwater or brackish fish. | Mechanical irritation is caused by movement of the worms causing mucosal villous atrophy. Chemical excretory/secretory proteins acts as active antigens and toxins, provoking a systemic immune response.[27] | Mucosal changes lead to nutrient malabsorption, intestinal permeability, and watery diarrhea.[27] Abdominal pain, weight loss, and anorexia are also seen.[27] |

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| *Paragonimus* spp. | Lung fluke<br><br>(P) | There are roughly 9 species that cause clinical disease in humans, but only *P. kellicotti* is endemic to North America, where it is found in streams and rivers in the Mississippi River Basin, including the central United States west to the Rocky Mountains.[11]<br><br>Infection is caused by ingestion of raw or undercooked crabs or crayfish.[28] | After ingestion of infected crabs or crayfish, the fluke resides in the small intestine and migrates through the intestinal wall into the peritoneal space and eventually into the pleural space.[11] | Patients are often asymptomatic after ingestion and during the initial migration phase. Some patients may develop abdominal pain and diarrhea. After migration to the pleural space, inflammatory pulmonary symptoms begin.[11]<br><br>Typical features of pulmonary paragonimiasis include cough, hemoptysis, chest pain, and dyspnea.[28] |
| *Schistosoma* spp.<br><br>*Schistosoma mansoni*<br><br>*Schistosoma japonicum*<br><br>*Schistosoma haematobium*<br><br>*Schistosoma mekongi* | Blood fluke<br><br>S. *mansoni*, S. *japonicum,* and *S. mekongi* cause intestinal disease[29]<br><br>S. *haematobium* causes urinary disease<br><br>(P) | This organism is prevalent in the tropics and subtropics where poor sanitation is common.[30]<br><br>*S. mekongi* is primarily limited to the Mekong River Basin stretching from Laos to Cambodia.[29]<br><br>Humans contract the infection via water sources.[30] | Humans acquire the infection by direct contact with water sources containing infectious larvae. The larvae penetrate skin and enter the circulation via the capillaries and lymphatics.[30] | Schistosome egg deposition and fluke burden can occur in any ectopic site, giving rise to site-specific symptoms and disease. These include dermatitis, abdominal pain, diarrhea, ascites, GI bleeding, and urinary obstructive symptoms.[30]<br><br>Intestinal schistosomiasis can mimic diverticulitis and IBD. Hepatic schistosomiasis can mimic alcoholic liver disease and liver cirrhosis.[6] |

# Parasitic Organisms

## PROTOZOA

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|---|---|---|---|---|
| *Balantidium coli* | Ciliate protozoan<br><br>(PP) | *Balantidium* is reported worldwide, but is more prevalent in temperate and tropical regions.<br><br>Human infections are related to poor sanitation and drinking water contaminated with human and animal (swine) feces.[4] | Trophozoites inhabit the intestine, feeding on bacteria and other intestinal contents. In most cases, infections are asymptomatic and the infected host shows no clinical signs, suggesting that this ciliate is an opportunistic parasite that could take advantage of the host's weakened status caused by other infections or diseases. In such cases, the parasite could invade the intestinal wall, causing the disease known as balantidiasis or balantidial dysentery.[31] | Often asymptomatic, but in the acute form symptoms may include mucus and blood in feces. In severe cases, hemorrhages and perforation could occur. Chronic infection may present with unspecific abdominal disorders (diarrhea, abdominal pain), cramping rectal pain, nausea, and vomiting.[31]<br><br>Balantidiasis can mimic traveler's diarrhea, invasive amebiasis, bacterial dysentery and IBD.[6] |
| *Blastocystis* spp. | Although there are 17 different Blastocystis subtypes, subtypes 1-9 are the only subtypes found in humans.<br><br>Subtypes 1-4 make up 90%.<br><br>Subtype 3 is most common.<br><br>Subtypes have geographic distribution.<br><br>(PP) | *Blastocystis* is one of the most common parasites, affecting 1.5-30% of those in industrialized countries and 30-76% in developing countries.[32,33]<br><br>*Blastocystis* transmission is via the fecal-oral route by ingesting contaminated food or water, exposure to daycare environments, and exposure to domestic and wild animals.[34,35]<br><br>Various subtypes have different zoonotic transmissions. | *Blastocystis* resides in the ileum and cecum, adheres to mucus' outer layer, and uses certain bacteria as a nutritional source. (30) Its pathogenicity is controversial.[32]<br><br>It is associated with higher microbial diversity, and may be regarded as a commensal organism.[33]<br><br>Literature-based conclusions about disease associations and subtype pathogenicity is conflicting, but evolving. | *Blastocystis* is often asymptomatic. When present, symptoms include nausea, anorexia, abdominal pain, flatulence, acute/chronic diarrhea, constipation, anal itching, fatigue, joint pain, and urticaria.<br><br>It is associated with irritable bowel syndrome (IBS), and is three times higher in patients with IBS-D.[36]<br><br>Blastocystosis can mimic acute viral enteritis, and traveler's diarrhea.[6] |
| *Chilomastix mesnili* | Non-pathogenic parasite<br><br>(NP) | *C. mesnili* is found in about 3.5% of the US population.[37]<br><br>Transmission is fecal-oral via the ingestion of mature cysts from contaminated water or food. | *C. mesnili* lives in the cecum and colon, but is noninvasive and nonpathogenic.[37] | Although *C. mesnili* is nonpathogenic, and causes no symptoms, it often occurs with other parasitic infections.[37] |
| *Cryptosporidium* spp. | Coccidian parasite<br><br>(P) | *Cryptosporidium* is endemic to North, Central, and South America, Africa, and Australia.<br><br>Infection is spread via the fecal-oral route and indirectly through contaminated water. *Cryptosporidium* is a common cause of food and water-borne outbreaks.[4] | The parasite adheres to intestinal epithelial cells. The intestinal epithelium releases cytokines to incite an immune response and causes cell apoptosis and villous atrophy. *Cryptosporidium* has developed ways to slow this protective mechanism; therefore, host immune competency determines pathogenicity.[38] | In immunocompetent patients, infection is self-limiting with 2 weeks of watery diarrhea. Other symptoms include fever, nausea, vomiting, and abdominal pain. Symptoms can be cyclical.[39,40]<br><br>It can be life threatening in immunocompromised patients.[4,41]<br><br>Enteric cryptosporidiosis can mimic malabsorption syndrome, Giardiasis, and viral diarrhea. Biliary cryptosporidiosis can mimic acute cholangitis.[6] |

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|---|---|---|---|---|
| *Cyclospora cayetanensis* | Food- and waterborne coccidian parasite<br><br>(P) | *Cyclospora* is endemic to Nepal, Haiti, Peru, and Guatemala, but it has been found as a cause of traveler's diarrhea worldwide. There have been many US outbreaks related to imported fruits and vegetables.[4,42] In the US, increased cases are reported during the spring and summer months.<br><br>Transmission is fecal-oral via ingestion of contaminated water or food. | Individuals with *Cyclospora* infection excrete unsporulated oocysts in their feces. These oocysts require 7 to 15 days to sporulate and become infectious to a susceptible host. When food or water contaminated with infectious oocysts is ingested by a susceptible host, the oocysts excyst and sporozoites are released and infect epithelial cells of the duodenum and jejunum.[43] | Cyclosporiasis is marked by profuse, non-bloody, watery diarrhea, anorexia, fatigue, weight loss, nausea, flatulence, abdominal cramping, myalgias, vomiting, and low grade fever.<br><br>Symptoms start approximately 7 days after ingestion. If left untreated, it can last weeks to months, with remitting and relapsing symptoms.[4]<br><br>Cyclosporiasis can mimic acute viral enteritis and traveler's diarrhea.[6] |
| *Cystoisospora* spp.<br><br>*Cystoisospora belli* | Coccidian parasite<br><br>Previously referred to as *Isospora*[44]<br><br>(P) | *Cystoisospora* is an uncommon human intestinal parasite. It has a worldwide distribution.<br><br>*Cystoisospora* is frequently found in tropical and subtropical regions.[45]<br><br>Transmission is through the fecal-oral route by the ingestion of contaminated water and food.[46] | *Cystoisospora* releases sporozoites that penetrate the small intestinal columnar epithelium.[47] | Some *Cystoisospora* infections are asymptomatic. When present, infections are usually mild and self-limiting, consisting of diarrhea and abdominal pain.[45]<br><br>It can cause severe chronic diarrhea in immunocompromised AIDS patients. It has also been reported to be a cause of traveler's diarrhea in the normal host and can mimic giardiasis or cryptosporidiosis.[48] |
| *Dientamoeba fragilis* | Flagellate protozoan parasite<br><br>(P) | *D. fragilis* has a worldwide distribution, and is transmitted via the fecal-oral route.<br><br>Transmission via helminth eggs (Ascaris, Enterobius) has also been postulated, but is still being investigated. | The role of *D. fragilis* as a pathogen is controversial because the trophozoites are not invasive and patients are commonly asymptomatic.[4] | Although many patients are asymptomatic, *D. fragilis* has been associated with diarrhea, abdominal pain, nausea, weight loss, anorexia, and flatulence.[4]<br><br>Dientamebiasis can mimic eosinophilic colitis and IBD.[6] |
| *Entamoeba coli* | Non-pathogenic amoeba<br><br>(NP) | *E. coli* is cosmopolitan in distribution and has been postulated to occur in approximately 50% of the population.[49]<br><br>Transmission is fecal-oral via the ingestion of contaminated water or food.[49] | *E. coli* lives inside the large intestine but never enters the mucosa or sub-mucosal intestinal layers. | The presence of *E. coli* is not cause to seek treatment and is harmless. However, when a patient is infected with this benign amoeba, introduction of other pathogenic organisms is possible and may cause symptoms.[37] |
| *Entamoeba dispar* | Non-pathogenic amoeba<br><br>*E. dispar* is morphologically and genetically similar to the virulent *E. histolytica;* therefore, other laboratory methods are necessary to distinguish the two.[50]<br><br>(NP) | It is speculated that this species is responsible for most infections that were previously considered to be *E. histolytica*. *E. dispar* has a high worldwide prevalence.[50]<br><br>Transmission is fecal-oral via the ingestion of contaminated water or food.[51] | *E. dispar* is noninvasive and considered non-pathogenic.[50] | *E. dispar* infection is not associated with clinical symptoms. |

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| *Entamoeba hartmanni* | Non-pathogenic amoeba<br><br>*E. hartmanni* can be distinguished from the virulent *E. histolytica* by the much smaller cyst size.[52]<br><br>(NP) | *E. hartmanni* has a worldwide distribution, but is most common in developing countries with poor sanitation.[53]<br><br>Transmission is fecal-oral via the ingestion of mature cysts from contaminated water or food.[52] | *E. hartmanni* is non-pathogenic.[53] | *E. hartmanni* infection is not associated with clinical symptoms. |
| *Entamoeba histolytica* | The leading parasitic cause of mortality globally,[52] and the third most common parasitic infection in the US.[4]<br><br>Erythrophagocytosis has been used as a diagnostic indicator of invasive *E. histolytica* by microscopy.[54]<br><br>(P) | *E. histolytica* has a worldwide distribution, but is most common in developing countries with poor sanitation.[53]<br><br>Transmission is fecal-oral via ingestion of mature cysts from contaminated water or food, or contaminated individuals.[53] | Cysts are ingested and excystation occurs. Colonization usually happens in the large bowel, but the cecum is most common. It penetrates the endothelium causing ulceration. Host factors appear to promote a more invasive, systemic disease, leading to dysentery, liver abscess, pleuropulmonary involvement, and many other systemic complications.[53] | Although many cases are asymptomatic, it is likely that misdiagnosis is the cause (failure to identify *E. dispar/hartmanni*).<br><br>Symptoms of *E. histolytica* infection (amoebiasis) include hemorrhagic diarrhea, fatigue, nausea, fever, weight loss.[55]<br><br>Intestinal amebiasis can mimic infectious diarrhea, IBD, ischemic colitis, diverticulitis, AV malformation. Amoeboma can mimic colon carcinoma. Amebic strictures can mimic lymphogranuloma venereum (chlamydia) and malignancy. Hepatic amebiasis can mimic pyogenic liver abscess, necrotic hepatoma and echinococcal cyst.[6] |
| *Entamoeba polecki* | Non-pathogenic amoeba<br><br>*E. polecki* can be distinguished from other Entamoeba species by microscopy. It is the only uninucleated species.[56] *E. polecki* comprises four subtypes, all of which are found in humans.[56]<br><br>(NP) | Infection with *E. polecki* is rare, though its prevalence and distribution are often confused with those of the other Entamoeba species.[56,57]<br><br>E. polecki, much like all Entamoeba species, is transmitted through the fecal-oral route by the ingestion of contaminated food or water.[56] | *E. polecki* is non-pathogenic. | *E. polecki* infection is not associated with clinical symptoms. |
| *Endolimax nana* | Non-pathogenic protozoa<br><br>(NP) | *E. nana* has a global distribution.<br><br>*E. nana* is transmitted through the fecal-oral route through ingestion of contaminated food or water.[58] | *E. nana* inhabits the colon and has been found in the appendix. *E. nana* feeds on bacteria and is non-invasive.[58] | *Endolimax* is an indicator of fecal contamination, which often entails co-infection by other organisms capable of causing diarrhea. There are rare cases of associations with urticaria, polyarthritis, and diarrhea. However, there is too little evidence to support pathogenicity.[58] |

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|---|---|---|---|---|
| *Giardia* | Flagellate protozoa<br><br>(P) | *Giardia* has a worldwide distribution. In the US, it is more frequently reported in children aged 1-9.[59]<br><br>*Giardia* is transmitted through the fecal-oral route by the ingestion of contaminated water and food.[59] | *Giardia* (via secretory and excretory proteases) may alter the structure and composition of human intestinal microbiota biofilms. Bacteria from these dysbiotic microbiota in turn can cause epithelial and intestinal abnormalities after the enteropathogen has been cleared.[60] | *Giardia* is a leading cause of diarrhea worldwide. Some cases may be asymptomatic; when present symptoms include diarrhea, bloating, malabsorption, nausea, vomiting, and abdominal cramping. Infections are normally self-limiting, but chronic diarrhea may occur in children.[59]<br><br>It is emerging as a prominent precursor to post-infectious irritable bowel syndrome and a variety of chronic extra-intestinal disturbances, such as reactive arthritis and chronic fatigue.[60] Acute giardiasis can mimic acute viral enteritis, bacillary dysentery, acute intestinal amebiasis and IBD.[6] |
| *Iodamoeba butschlii* | Non-pathogenic amoeba<br><br>*Iodamoeba* gets its name from its appearance when stained with iodine.[37]<br><br>(NP) | *Iodamoeba* has a worldwide distribution. Humans have a low prevalence of *Iodamoeba butschili.*<br><br>Transmission is through the fecal-oral route by the ingestion of contaminated water and food.[37] | *Iodamoeba* is usually found in the large intestine and are non-invasive.[37] | *Iodamoeba* is not associated with symptoms. However, it is an indicator of fecal contamination, which often entails co-infection by other organisms capable of causing diarrhea.[37] |
| *Trichomonads-Pentatrichomonas*<br><br>*Pentatrichomonas hominis*<br><br>*Enteromonis hominis*<br><br>*Retortamonas intestinalis* | Flagellate parasite<br><br>*Trichomonas tenax* is usually found in oral/periodontal infections and cannot survive intestinal passage.<br><br>*Pentatrichomonas hominis* (also known as *Trichomonas hominis*) will not survive in the oral cavity or genitourinary tract. It is considered a non-pathogenic parasite.[37]<br><br>*Trichomonas vaginalis* is confined to the urogenital system. Among trichomonads, there is a habitat restriction: each can survive only in its site-specific location.[37]<br><br>(NP) | Trichomonads have worldwide distribution.<br><br>*E. hominis, P. hominis, and R. intestinalis* are transmitted via the fecal-oral route by the ingestion of contaminated water, food, and flies.[37] | *P. hominis, R. intestinalis, and E. hominis* are considered non-pathogenic commensals found in the cecum and colon.[37] | Trichomonads in the stool are not related to gastrointestinal illness.[37]<br><br>The presence of trichomonad trophozoites in the stool can be an indicator of fecal contamination, and therefore doesn't rule out other infections as a cause of symptoms.[37] |

## TREATMENT RESOURCES:

The decision to treat parasitic organisms should be based on the patient's clinical presentation.
The following resources provide valuable insight into the clinical management of parasitic infections:

- Center for Disease Control – monographs on individual parasites and treatment protocols: https://www.cdc.gov/parasites/
- Sanford Guide - infectious disease treatment guidelines: https://www.sanfordguide.com/
- CDC hotline for healthcare providers with questions regarding parasites:
  - Parasitic Diseases Hotline (M-F; 8am-4pm EST) 404-718-4745
  - Emergency, after-hours hotline 770-488-7100
- Mayo Clinic – conditions search engine: https://www.mayoclinic.org/
- PubMed – literature search engine: https://www.ncbi.nlm.nih.gov/pubmed/

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