

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
<i>Blastocystis hominis</i>	<i>B. hominis</i> has recently been reclassified as a protozoan, of which there are thought to be four separate serologic groups. ¹	This organism is transmitted via the fecal-oral route or from contaminated food or water. ² Prevention can be enhanced by improving personal hygiene and sanitary conditions. ³	The role of <i>B. hominis</i> in terms of colonization and disease is still considered controversial. When this organism is present in the absence of any other parasites, enteric organisms or viruses, it may be considered the etiological agent of disease. ⁴	Symptoms can include: diarrhea, cramps, nausea, fever, vomiting and abdominal pain. ⁵ <i>B. hominis</i> has been associated with irritable bowel syndrome, infective arthritis and intestinal obstruction. ⁶	Currently, Metronidazole (Flagyl) is considered the most effective drug (750 mg tid x 10 days). ⁷ Iodoquinol (Yodoxin) is also an effective medication (650 mg tid x 20 days). ⁸ Recommended therapy can also eliminate <i>G. lamblia</i> , <i>E. histolytica</i> and <i>D. fragilis</i> , all of which may be concomitant undetected pathogens and part of patient symptomatology. ^{9,10}



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<i>Cryptosporidium spp</i>	<i>Cryptosporidium spp</i> are coccidian parasites that belong to the <i>Cryptosporidiidae</i> family. ¹¹	Infection is thought to occur by environmentally resistant oocysts, zoonotic transmission, nosocomial transmission and direct person-to-person contact. ¹² Contamination of public water supply has been associated with outbreaks. Raw foods such as unpasteurized milk and raw meat can also harbor the organism. ¹³	<i>Cryptosporidium</i> is an important agent of diarrhea in both the immunocompetent and immunocompromised host. ¹⁴ The organism inhabits the intestinal mucosa causing diarrhea. ¹⁵ Infection in the immunocompromised host may cause life threatening disease and can disseminate from the intestinal tract. ¹⁶ <i>Cryptosporidium</i> is considered an important opportunistic pathogen in patients with AIDS, and detection is associated with a poor prognosis. ¹⁷	Acute infections can mimic Crohn's disease with villus atrophy, enlarged crypts, and infiltration of the lamina propria by inflammatory cells. ¹⁸ Clinical symptoms in the immunocompetent host include nausea, low-grade fever, abdominal cramps, anorexia and up to 5-10 watery bowel movements a day, which may be followed by constipation. ¹⁹ Immunocompetent hosts can also be asymptomatic. ²⁰ <i>Cryptosporidium</i> in the immunocompromised host may be ongoing and severe. The length and severity depend on the ability to reverse the immunosuppression. Extra-intestinal infections can occur with respiratory symptoms, cholecystitis, hepatitis and pancreatitis. ²¹ Chronic cryptosporidiosis in infants is associated with failure to thrive. ²²	Cryptosporidiosis is generally self-limiting in immunocompetent patients, lasting approximately 2 weeks. ²³ Currently, there is no totally effective therapy for cryptosporidiosis. Refer to the Medical Letter and/or Sanford Guide for therapeutic protocols in the immunocompromised host. ²⁴



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<i>Dientamoeba fragilis</i>	<i>D. fragilis</i> has recently been reclassified as an ameboflagellate (previously ameba) and is closely related to <i>Histomonas</i> and <i>Trichomonas</i> species. ²⁵	Because this organism does not have a cyst stage, there is uncertainty of the mode of transmission. ²⁶ Fecal oral transmission thus far has not been documented. ²⁷ Higher incidences have been reported for mental institutions, missionaries and Native Americans of Arizona. <i>D. fragilis</i> is also common in pediatric populations and patients under the age of 20. ²⁸	<i>D. fragilis</i> is known to cause non-invasive diarrheal illness in humans. 90% of children are symptomatic, whereas only 15-20% of adults are. ²⁹	The most common symptoms associated with <i>D. fragilis</i> are intermittent diarrhea, fatigue, abdominal pain, fatigue, nausea, anorexia, malaise and unexplained eosinophilia. ³⁰ Diarrhea is predominately seen during the first 1-2 weeks of infection and abdominal pain may persist for 1-2 months. ³¹	Iodoquinol (650 mg tid x 20 days) and Tetracycline (500 mg qid x 10 days) have been used to treat <i>D. fragilis</i> . ³² Other therapies include Doxycycline (100 mg bid x 10 days) and Paromomycin (500 mg tid x 7 days). ³³
<i>Entamoeba coli</i>	This organism is a protozoan belonging to the amebae family. ³⁴	<i>Entamoeba coli</i> has a worldwide distribution, the prevalence is generally greater in warmer climates. ³⁵ The cyst which is the infectious form is ingested from contaminated food and water. Direct transmission can also occur via the fecal-oral-route. ³⁶	While <i>Entamoeba coli</i> is the most common ameba isolated in humans, it is considered non-pathogenic. ³⁷	<i>Entamoeba coli</i> is not associated with intestinal symptoms.	The Medical Letter and Sanford Guide provide no therapeutic recommendations for <i>Entamoeba coli</i> . Treatment is not recommended for non-pathogenic amebae. Improving sanitary conditions and personal hygiene help to prevent infection. ³⁸



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<i>Entamoeba dispar</i>	<i>E. dispar</i> is a protozoan that belongs to the amebae family. ³⁹	Transmission is from the ingestion of infective cysts in contaminated food or water. Person-to-person contact is also a source of transmission. ⁴⁰	<i>Entameoba dispar</i> is considered to be non-pathogenic in humans. ⁴¹	This particular species of <i>Entameoba</i> is not known to produce intestinal symptoms, nor is it invasive in humans. ⁴²	The Medical Letter and Sanford Guide provide no therapeutic recommendations for <i>Entamoeba dispar</i> . Treatment is generally not recommended for non-pathogenic amebae, however this recommendation is based upon being able to accurately differentiate <i>E. dispar</i> from pathogenic <i>E. histolytica</i> . ⁴³
<i>Entamoeba hartmanni</i>	This organism belongs to the amebae family. ⁴⁴	Transmission is related to the ingestion of cysts from contaminated food or water. ⁴⁵	<i>Entamoeba hartmanni</i> is not considered a pathogen in humans. While early research identified this organism as a potential pathogen, subsequent studies were unable to adequately confirm. ⁴⁶	<i>Entamoeba hartmanni</i> is not routinely associated with clinical symptoms. ⁴⁷	Treatment for <i>E. hartmani</i> is usually not recommended, accordingly the Medical Letter and the Sanford guide have no therapeutic recommendations. ⁴⁸



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<i>Entamoeba histolytica</i>	<i>E. histolytica</i> belongs to the ameba family of protozoa. ⁴⁹	This organism has been recovered worldwide, though is more prevalent in the tropics and subtropics. In unsanitary conditions, infection rates are equivalent to tropical regions despite colder climates. ⁵⁰ Humans are the reservoir for <i>E. histolytica</i> and can transmit the parasite to other humans, primates, cats, dogs and possibly pigs. ⁵¹	<i>E. histolytica</i> is pathogenic for humans, causing invasive intestinal and extraintestinal amebiasis. ⁵² In 2-8% of infected individuals, invasion of the intestinal mucosa occurs with dissemination to other organs (most frequently the liver). ⁵³ The organism is capable of inducing both humoral and cellular immune responses. ⁵⁴	While a large number of people worldwide are infected with <i>E. histolytica</i> , only a few manifest clinical symptoms. ⁵⁵ Asymptomatic patients may excrete cysts for only a short period of time and are essentially unaffected and never experience symptoms. ⁵⁶ Some patients may experience symptoms that mimic ulcerative colitis. Others still may have a gradual onset of symptoms including diarrhea, colicky abdominal pain, and tenesmus. The incubation time for those symptomatic can vary from 1-4 weeks. With the onset of dysentery, diarrhea can occur with up to 10 movements a day that are characterized by blood-tinged mucus. ⁵⁷	<i>E. histolytica</i> should be treated even if patients are asymptomatic. Diloxanide furoate (500 mg tid x 10 days) and Iodoquinol (650 mg tid x 20 days) are used for cysts in the gut lumen, but are ineffective for extraintestinal infections. ⁵⁸ Metronidazole (500-750 mg x 10 days, then Paromomycin 500 mg tid x 10 days) are the drugs of choice for mild to moderate disease when tissue invasion occurs. ⁵⁹



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<i>Giardia lamblia</i>	<p><i>Giardia lamblia</i> is the most commonly diagnosed flagellate in the intestinal tract.⁶⁰</p> <p><i>Giardia intestinalis</i> and <i>Giardia duodenalis</i> are also used as names for this organism.⁶¹</p>	<p>Infection occurs via fecal-oral transmission or from food and water contaminated with the cysts.⁶²</p> <p><i>Giardia lamblia</i> has a worldwide distribution, though is more common in warmer climates than cooler ones. Isolation of the organism is more prevalent in children or those living in close quarters with poor sanitary conditions.⁶³</p>	<p><i>Giardia lamblia</i> is considered a pathogen in humans.⁶⁴</p>	<p>Most people infected with <i>G. lamblia</i> are asymptomatic. For those symptomatic, there can be an acute and a chronic phase of infection.⁶⁵</p> <p>After an incubation period of 2-20 days, symptoms of watery diarrhea, nausea, low grade fever and chills can occur lasting only a few days.⁶⁶ Acute infection can mimic food poisoning, bacillary dysentery, viral enteritis, acute intestinal amebiasis or travelers diarrhea. One point of differentiation is the lack of blood, mucus and cellular exudates in the stool with <i>G. lamblia</i>.⁶⁷</p> <p>In the chronic phase, symptoms can include recurrent foul smelling diarrhea, abdominal distention, belching and heartburn.⁶⁸</p> <p>Chronic Giardiasis may lead to dehydration, malabsorption and impaired pancreatic function.⁶⁹</p>	<p>The drug of choice is Metronidazole (250 mg tid x 5 days) and is recommended also for immunocompetent hosts with self limiting infections. Treatment helps prevent transmission of the organism and reduce the duration of symptoms.⁷⁰</p> <p>Paromomycin (25-35 mg/kg/day in three doses x 7 days) is the alternative for treating <i>G. lamblia</i> during pregnancy.⁷¹</p> <p>Other therapeutic alternatives include Tinidazole (2g once), and Furazolidone (100 mg qid x 7-10 days).⁷²</p>



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Helminths					
<i>Ascaris lumbricoides</i>	<i>Ascaris lumbricoides</i> is the largest and most prevalent of all the human intestinal nematodes. ⁷³	This organism is more prevalent in warm, moist climates, though it can survive in temperate regions. ⁷⁴ Infection is acquired through the ingestion of embryonated eggs in contaminated soil. ⁷⁵	This organism is a clearly defined pathogen in humans with infection rates as high as 45% in Central and South America. ⁷⁶ The pathogenesis of <i>A. lumbricoides</i> is attributed to (i) the immune response of the host (ii) the effects of larval migration (iii) the effects of adult worms (iv) nutritional deficiencies resultant from the adult worms. ⁷⁷	Symptoms relate to the migration of the worm after hatching in the stomach, penetrating the intestinal wall and migrating through the liver to the lungs. When in the intestine, patients are usually asymptomatic, unless the worm burden is high. Migration can result in intestinal blockage, entry into the bile or pancreatic duct, or liver or peritoneal cavity. Repeated infections or those with a large volume of eggs can result in pneumonitis (Loeffler's syndrome) during the larval migration phase through the lungs. Symptoms include cough, dyspnea, wheezing or coarse rales, fever and transient eosinophilia. ⁷⁸ Infection can be terminated by the spontaneous passage of the adult worms from the anus, mouth or nares. ⁷⁹	Mebendazole (100 bid x 3 days) is considered the most effective drug and is suitable for both children and adults. Pyrantel pamoate (11 mg/kg once (maximum 1 gram), repeat after two weeks) and Albendazole (400 mg once) are alternatives, however these drugs are still considered investigational. ⁸⁰



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<i>Enterobius vermicularis</i> (Pinworm)	<i>E. vermicularis</i> is a nematode, and is the most prevalent parasitic infection in the world. ⁸¹	Infection is more common in the cooler, temperate regions, and thought to be related to reduced bathing and changing of underclothes. ⁸² Infection is more prevalent in children and occurs more commonly in females. ⁸³	<i>E. vermicularis</i> is considered a pathogenic organism. ⁸⁴	Those infected may be asymptomatic or experience pruritus from the migration of the worms from the anus to the perianal skin where the eggs are deposited. ⁸⁵ Other symptoms found in infected children include insomnia, nervousness, irritability, nightmares and convulsions. ⁸⁶	Treatment is with Pyrantel pamoate (11 mg/kg once (maximum once), repeat after two weeks) or Mebendazole (100 mg once, repeat after two weeks), or Albendazole (400 mg once, repeat after two weeks). Therapy should always be based upon evidence of infection and symptomology. ⁸⁷



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<i>Strongyloides stercoralis</i>	<i>S. stercoralis</i> is classified as a nematode. ⁸⁸	The organism is more prevalent in tropics and subtropics, though can survive colder climates. ⁸⁹ The first stage larvae are contaminated in the soil, and infection occurs from skin penetration where the organism then travels to the intestine via the blood, lungs, trachea and upper Gastro-intestinal tract. ⁹⁰	<i>S. stercoralis</i> is considered a pathogen in humans. ⁹¹	Individuals can be asymptomatic, or exhibit symptoms in three key areas relative to the life cycle of the parasite and a heavy infective dose. ⁹² Cutaneous penetration may result in pruritis and erythema when the larvae are in high numbers. With larval migration through the lungs, infected hosts may develop a cough, shortness of breath, wheezing, fever, and pneumonia. When there is intestinal infestation, symptoms can mimic peptic ulcer and there may be damage to the intestinal mucosa with villous atrophy and crypt hyperplasia. Radiographic findings may be akin to Crohn's disease of the proximal small intestine. ⁹³ Reactive arthritis has also been associated with a heavy <i>Strongyloides</i> infection. ⁹⁴	Treatment options for <i>Strongyloides</i> include Ivermectin (200 ug/kg/day x 1-2 days) or Thiabendazole (25mg/kg.day bid (maximum of 3g/day)). ⁹⁵

* Treatment protocols sourced from the Medical Letter (03) or the Sanford Guide (03).

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² Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 31.

³ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 31.



Parasite Organism Chart

- ⁴ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 30.
- ⁵ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 30.
- ⁶ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 30.
- ⁷ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 31.
- ⁸ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 31.
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- ¹⁰ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 30.
- ¹¹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 60.
- ¹² Ortega YR. *Cryptosporidium, cyclospora, and isospora*. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1411.
- ¹³ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 74,75.
- ¹⁴ Ortega YR. *Cryptosporidium, cyclospora, and isospora*. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1411.
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- ¹⁶ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 76.
- ¹⁷ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 60,61.
- ¹⁸ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 63.
- ¹⁹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 63.
- ²⁰ Garcia LS, Bruckner DA. Taxonomy and classification of human parasites. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1331.



Parasite Organism Chart

- ²¹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 65.
- ²² Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 63.
- ²³ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 61,71.
- ²⁴ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 60.
- ²⁵ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 49.
- ²⁶ Leber AL, Movak SM In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1401.
- ²⁷ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Santa Monica; 2001; pg 51.
- ²⁸ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Santa Monica; 2001; pg 49.
- ²⁹ Leber AL, Movak SM In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1401.
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Parasite Organism Chart

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- ³⁸ Leber AL, Movak SM. Intestinal and urogenital amebae, flagellates, and ciliates. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Yolken RH, eds. *Manual of Clinical Microbiology*. 7th ed. Washington DC: ASM Press; 1999; 1399.
- ³⁹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 6.
- ⁴⁰ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 21.
- ⁴¹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 7.
- ⁴² Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 7.
- ⁴³ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 21.
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Parasite Organism Chart

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- ⁵⁹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 19.
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- ⁶¹ Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 36.
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Parasite Organism Chart

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