Protozoan diseases essay sample

Malaria is a mosquito-borne infectious disease of humans and other animals caused by protists (a type of microorganism) of the genus Plasmodium. It begins with a bite from an infected female mosquito (Anopheles Mosquito), which introduces the protists via its saliva into the circulatory system, and ultimately to the liver where they mature and reproduce. The disease causes symptoms that typically include fever and headache, which in severe cases can progress to coma or death. Malaria is widespread in tropical and subtropical regions in a broad band around the equator, including much of Sub-Saharan Africa, Asia, and the Americas. Five species of Plasmodium can infect and be transmitted by humans. The vast majority of deaths are caused by P. falciparum while P. vivax, P. ovale, and P. malariae cause a generally milder form of malaria that is rarely fatal.

The zoonotic (an infectious disease that is transmitted between species (sometimes by a vector) from animals to humans or from humans to animals) species P. knowlesi, prevalent in Southeast Asia, causes malaria in macaques (Old World monkeys) but can also cause severe infections in humans. Malaria is prevalent in tropical and subtropical regions because rainfall, warm temperatures, and stagnant waters provide habitats ideal for mosquito larvae. Disease transmission can be reduced by preventing mosquito bites by distribution of mosquito nets and insect repellents, or with mosquito-control measures such as spraying insecticides and draining standing water. Diagnosis

Malaria is typically diagnosed by the microscopic examination of blood using blood films, or with antigen-based rapid diagnostic tests. Modern techniques that use the polymerase chain reaction to detect parasite DNA have also

been developed, but these are not widely used in malaria-endemic areas due to their cost and complexity. The World Health Organization has estimated that in 2010, there were 216 million documented cases of malaria. That year, between 655, 000 and 1. 2 million people died from the disease (roughly 2000–3000 per day), many of whom were children in Africa. The actual number of deaths is not known with certainty, as precise statistics are unavailable in many rural areas, and many cases are undocumented. Malaria is commonly associated with poverty and may also be a major hindrance to economic development. Treatment

Despite a need, no effective vaccine currently exists, although efforts to develop one are ongoing. Several medications are available to prevent malaria in travellers to malaria-endemic countries (prophylaxis). A variety of antimalarial medications are available. Severe malaria is treated with intravenous or intramuscular quinine or, since the mid-2000s, the artemisinin derivative artesunate, which is superior to quinine in both children and adults and is given in combination with a second anti-malarial such as mefloquine. Resistance has developed to several antimalarial drugs; for example, chloroquine-resistant P. falciparum has spread to most malarial areas, and emerging resistance to artemisinin has become a problem in some parts of Southeast Asia. Signs and symptoms

The signs and symptoms of malaria typically begin 8–25 days following infection; however, symptoms may occur later in those who have taken antimalarial medications as prevention. Initial manifestations of the disease —common to all malaria species—are similar to flu-like symptoms, and can resemble other conditions such as septicemia (potentially deadly medical

condition characterized by a whole-body inflammatory state), gastroenteritis, and viral diseases. The presentation may include headache, fever, shivering, arthralgia (joint pain), vomiting, hemolytic anemia, jaundice, hemoglobinuria, retinal damage, and convulsions. Approximately 30% of people however will no longer have a fever upon presenting to a health care facility. Owing to the non-specific nature of disease presentation, diagnosis of malaria in non-endemic countries requires a high degree of suspicion, which might be elicited by any of the following: recent travel history, splenomegaly (enlarged spleen), fever without localizing signs, thrombocytopenia (relative decrease of platelets in blood), and hyperbilirubinemia combined with a normal peripheral blood leukocyte count.

The classic symptom of malaria is paroxysm—a cyclical occurrence of sudden coldness followed by rigor (chill) and then fever and sweating, occurring every two days (tertian fever) in P. vivax and P. ovale infections, and every three days (quartan fever) for P. malariae. P. falciparum infection can cause recurrent fever every 36–48 hours or a less pronounced and almost continuous fever. Severe malaria is usually caused by P. falciparum (often referred to as falciparum malaria). Symptoms of falciparium malaria arise 9–30 days after infection. Splenomegaly, severe headache, hepatomegaly (enlarged liver), hypoglycemia, and hemoglobinuria with renal failure may occur. Renal failure is a feature of blackwater fever, where hemoglobin from lysed red blood cells leaks into the urine. Cerebral malaria is a form of severe malaria that involves encephalopathy specifically related to P. falciparum infection. It is associated with retinal whitening, which may be a useful clinical sign in distinguishing malaria from other causes of fever.

Individuals with cerebral malaria frequently exhibit neurological symptoms, including abnormal posturing, nystagmus, conjugate gaze palsy (failure of the eyes to turn together in the same direction), opisthotonus, seizures, or coma. There are a number of serious complications of malaria. Among these is the development of respiratory distress, which occurs in up to 25% of adults and 40% of children with severe P. falciparum malaria. Possible causes include respiratory compensation of metabolic acidosis, noncardiogenic pulmonary oedema, concomitant pneumonia, and severe anaemia. Acute respiratory distress syndrome (ARDS) may develop in 5–25% in adults and up to 29% of pregnant women but it is rare in young children. Coinfection of HIV with malaria increases mortality. Malaria in pregnant women is an important cause of stillbirths (fetus has died in the uterus), infant mortality and low birth weight, particularly in P. falciparum infection, but also with P. vivax Life cycle

In the life cycle of Plasmodium, a female Anopheles mosquito (the definitive host) transmits a motile infective form (called the sporozoite) to a vertebrate host such as a human (the secondary host), thus acting as a transmission vector. A sporozoite travels through the blood vessels to liver cells (hepatocytes), where it reproduces asexually (tissue schizogony), producing thousands of merozoites. These infect new red blood cells and initiate a series of asexual multiplication cycles (blood schizogony) that produce 8 to 24 new infective merozoites, at which point the cells burst and the infective cycle begins anew. In a process called gametocytogenesis, other merozoites develop into immature gametes, or gametocytes. When a fertilized mosquito bites an infected person, gametocytes are taken up with the blood and

mature in the mosquito gut. The male and female gametocytes fuse and form zygotes (ookinetes), which develop into new sporozoites. The sporozoites migrate to the insect’s salivary glands, ready to infect a new vertebrate host.

The sporozoites are injected into the skin, alongside saliva, when the mosquito takes a subsequent blood meal. This type of transmission is occasionally referred to as anterior station transfer. Only female mosquitoes feed on blood; male mosquitoes feed on plant nectar, and thus do not transmit the disease. The females of the Anopheles genus of mosquito prefer to feed at night. They usually start searching for a meal at dusk, and will continue throughout the night until taking a meal. Malaria parasites can also be transmitted by blood transfusions, although this is rare Malaria is typically diagnosed by the microscopic examination of blood using blood films or using antigen-based rapid diagnostic tests (RDT).

Microscopy is the most commonly used method to detect the malaria parasite—about 165 million blood smears were performed in 2010. Despite its widespread usage, diagnosis by microscopy suffers from two main drawbacks: many settings (especially rural) are not equipped to perform the test, and the accuracy of the results depends on both the skill of the person reading the smear and the levels of the parasite in the blood. The sensitivity of blood films ranges from 75–90% in optimum conditions, to as low as 50%. Commercially available RDTs are often more accurate than blood smears at predicting the presence of malaria parasites, but they are widely variable in diagnostic sensitivity and specificity depending on manufacturer, and are unable to tell how many parasites are present

Leishmaniasis

Leishmaniasis is a disease caused by protozoan parasites that belong to the genus Leishmania and is transmitted by the bite of certain species of sand fly (subfamily Phlebotominae). Although the majority of the literature mentions only one genus transmitting Leishmania to humans (Lutzomyia) in America, a 2003 study by Galati suggested a new classification for American sand flies, elevating several subgenera to the genus level. Elsewhere in the world, the genus Phlebotomus is considered the vector of leishmaniasis. Most forms of the disease are transmissible only from animals (zoonosis), but some can be spread between humans.

Human infection is caused by about 21 of 30 species that infect mammals: the different species are morphologically indistinguishable, but they can be differentiated by isoenzyme analysis, DNA sequence analysis, or monoclonal antibodies. The symptoms of leishmaniasis are skin sores which erupt weeks to months after the person affected is bitten by sand flies. Other consequences, which can manifest anywhere from a few months to years after infection, include fever, damage to the spleen and liver, and anemia. In clinical medicine, leishmaniasis is considered one of the classic causes of a markedly enlarged spleen; the organ, which is not normally felt during examination of the abdomen, may become larger even than the liver in severe cases. Leishmaniasis may be divided into the following types:

Visceral leishmaniasis is the most serious form, and is potentially fatal if untreated. Cutaneous leishmaniasis is the most common form, which causes a sore at the bite site, which heals in a few months to a year, leaving an unpleasant-looking scar. This form can progress to any of the other three

forms. Diffuse cutaneous leishmaniasis produces widespread skin lesions which resemble leprosy, and is particularly difficult to treat. Mucocutaneous leishmaniasis commences with skin ulcers which spread, causing tissue damage, to, particularly, the nose and mouth. Leishmaniasis is transmitted by the bite of female phlebotomine sandflies. The sandflies inject the infective stage, metacyclic promastigotes, during blood meals (1). Metacyclic promastigotes that reach the puncture wound are phagocytized by macrophages (2) and transform into amastigotes (3). Amastigotes multiply in infected cells and affect different tissues, depending in part on which Leishmania species is involved (4).

These differing tissue specificities cause the differing clinical manifestations of the various forms of leishmaniasis. Sandflies become infected during blood meals on infected hosts when they ingest macrophages infected with amastigotes (5, 6). In the sandfly’s midgut, the parasites differentiate into promastigotes (7), which multiply, differentiate into metacyclic promastigotes, and migrate to the proboscis (an elongated appendage from the head of an animal, either a vertebrate or an invertebrate) (8). Leishmaniasis is caused by infection with the pathogen Leishmania. The genomes of three Leishmania species (L. major, L. infantum, and L. braziliensis) have been sequenced and this has provided much information about the biology of the parasite. For example, in Leishmania, protein-coding genes are understood to be organized as large polycistronic units in a head-to-head or tail-to-tail manner; RNA polymerase II transcribes long polycistronic messages in the absence of defined RNA pol II promoters, and

Leishmania has unique features with respect to the regulation of gene expression in response to changes in the environment.

The new knowledge from these studies may help identify new targets for urgently needed drugs and aid the development of vaccines.[1] Leishmaniasis is diagnosed in the haematology laboratory by direct visualization of the amastigotes (Leishman-Donovan bodies). Buffy-coat preparations of peripheral blood or aspirates from marrow, spleen, lymph nodes, or skin lesions should be spread on a slide to make a thin smear and stained with Leishman's or Giemsa's stain (pH 7. 2) for 20 minutes. Amastigotes are seen with monocytes or, less commonly in neutrophils, of peripheral blood and in macrophages in aspirates. They are small, round bodies 2–4 µm in diameter with indistinct cytoplasm, a nucleus, and a small, rod-shaped kinetoplast. Occasionally, amastigotes may be seen lying free between cells. However, the retrieval of tissue samples is often painful for the patient and it can be difficult to identify the infected cells. For these reasons, other indirect immunological methods of diagnosis are developed.

These methods include the enzyme-linked immunosorbent assay (ELISA), antigen coated dipsticks, and the direct agglutination test (DAT). Although these tests are readily available, they are not the standard diagnostic tests due to their insufficient sensitivity and specificity. Over the years, several different Polymerase Chain Reaction (PCR) assays are made into use for the detection of Leishmania DNA. With the PCR assay, a specific and sensitive diagnostic procedure is finally possible. Currently, no vaccines are in routine use. However, the genomic sequence of Leishmania has provided a rich source of vaccine candidates. Genome-based approaches have been used to

screen for novel vaccine candidates. One study screened 100 randomly selected genes as DNA vaccines against L. major infection in mice. Fourteen reproducibly protective, novel vaccine candidates were identified. A separate study used a two-step procedure to identify T cell antigens. Six unique clones were identified: glutamine synthetase, a transitional endoplasmic reticulum ATPase, elongation factor 1gamma, kinesin K-39, repetitive protein A2, and a hypothetical conserved protein.

The 20 antigens identified in these two studies are being further evaluated for vaccine development. There are two common therapies containing antimony (known as pentavalent antimonials): meglumine antimoniate (Glucantime) and sodium stibogluconate (Pentostam). It is not completely understood how these drugs act against the parasite; they may disrupt its energy production or trypanothione metabolism. Unfortunately, in many parts of the world, the parasite has become resistant to antimony when treating for visceral or mucocutaneous leishmaniasis,[5] but the level of resistance varies according to species.[6]

Amphotericin (AmBisome) is now the treatment of choice;[7] its failure in some cases to treat visceral leishmaniasis (Leishmania donovani) has been reported in Sudan, but this may be related to host factors such as co-infection with HIV or tuberculosis rather than parasite resistance.[8] Hypoxanthine-guanine phosphoribosyl transferase (HGPRT; EC 2. 4. 2. 8) is a central enzyme in the purine recycling pathway. Parasitic protozoa (Leishmania donovani) cannot synthesize purines de novo and utilize the salvage pathway to produce purine bases. Thus, this enzyme is targeted in drug discovery and development. The model of the monomeric L. donovani

HGPRT showed that this enzyme is an α/β type protein with a PRTase type I folding pattern.

Among all of the computationally screened compounds, pentamidine, 1, 3-dinitroadamantane, acyclovir and analogs of acyclovir had higher binding affinities than the real substrate (guanosine monophosphate). Amino acids of HGPRT that are frequently involved in the binding of these compounds are Lys 66, Asp 74, Arg 77, Asp 81, Val 88, Tyr 182, Arg 192 and Arg 194. It is predicted that patients suffering from both HIV and visceral leishmaniasis (VL) may benefit if they are treated with acyclovir or pentamidine in conjunction with first-line antileishmanial therapies such as miltefosine and AmBisome.[9] Miltefosine (Impavido), is a new drug for visceral and cutaneous leishmaniasis. The cure rate of miltefosine in phase III clinical trials is 95%; studies in Ethiopia show it is also effective in Africa. In an observational study of 34 Dutch soldiers with Leishmania major infection who had failed to respond to intralesional antimony, 30 responded to miltefosine.[10]

In HIV-immunosuppressed people who are coinfected with leishmaniasis, even in resistant cases, 2/3 of the people have been shown to respond to this new treatment. Clinical trials in Colombia showed a high efficacy for cutaneous leishmaniasis. In mucocutaneous cases caused by L. brasiliensis, it has shown to be more effective than other drugs. Miltefosine received approval by the Indian regulatory authorities in 2002 and in Germany in 2004. In 2005, it received the first approval for cutaneous leishmaniasis in Colombia. Miltefosine is also currently being investigated as treatment for mucocutaneous leishmaniasis caused by Leishmania braziliensis in

Colombia,[5] and preliminary results are very promising. It is now registered in many countries and is the first orally administered breakthrough therapy for visceral and cutaneous leishmaniasis.[11][12] In October 2006, it received orphan drug status from the US Food and Drug Administration. The drug is generally better tolerated than other drugs.

Main side effects are gastrointestinal disturbance in the 1–2 days of treatment, which does not affect the efficacy. Because it is available as an oral formulation, the expense and inconvenience of hospitalisation is avoided, which makes it an attractive alternative. However, there are problems associated with the use of miltefosine that arise from its teratogenicity and pharmacokinetics: In a Dutch study by Thomas P. C. Dorlo in 2008, miltefosine was shown to be much slower eliminated from the body than previously thought and was therefore still detectable in human plasma samples taken five months after the end of treatment. The presence of subtherapeutic miltefosine concentrations in the blood beyond five months after treatment might contribute to the selection of resistant parasites and, moreover, the measures for preventing the teratogenic risks of miltefosine must be reconsidered. This led to some reluctance to taking Miltefosine by affected populations.

The antifungal drug, fluconazole 200 mg daily, has been shown to be significantly more effective in the treatment of cutaneous leishmaniasis compared to the placebo in a trial done in Saudi Arabia. In another randomized clinical trial from Iran, fluconazole 400 mg daily was shown to be significantly more effective than fluconazole 200 mg daily in the treatment of cutaneous leishmaniasis.[13] The Institute for OneWorld Health has

reintroduced the drug paromomycin for treatment of leishmaniasis, results with which led to its approval as an orphan drug. The Drugs for Neglected Diseases Initiative is also actively facilitating the search for novel therapeutics. A treatment with paromomycin will cost about $10.

The drug had originally been identified in 1960s, but had been abandoned because it would not be profitable, as the disease mostly affects poor people. [14] The Indian government approved paromomycin for sale in August 2006. [15] A 21-day course of paromomycin produces a definitive cure in > 90% of patients with visceral leishmaniasis.[16] Drug-resistant leishmaniasis may respond to immunotherapy (inoculation with parasite antigens plus an adjuvant), which aims to stimulate the body’s own immune system to kill the parasite.[17] Two weeks of topical treatment with 0. 1% cantharidin ointment was an effective method for treating cutaneous leishmaniasis in infected BALB/c mice.[

Amoebiasis

Amoebiasis, or Amebiasis, refers to infection caused by the amoeba Entamoeba histolytica. The term Entamoebiasis is occasionally seen but is no longer in use; it refers to the same infection. Likewise amoebiasis is sometimes incorrectly used to refer to infection with other amoebae, but strictly speaking it should be reserved for Entamoeba histolytica infection. Other amoebae infecting humans include: Parasites

Dientamoeba fragilis, which causes Dientamoebiasis

Entamoeba dispar

Entamoeba hartmanni

Entamoeba coli

Entamoeba moshkovskii

Endolimax nana and

Iodamoeba butschlii.

Except for Dientamoeba, the parasites above are not thought to cause disease. A gastrointestinal infection that may or may not be symptomatic and can remain latent (incubation period) in an infected person for several years, amoebiasis is estimated to cause 70, 000 deaths per year worldwide. Symptoms can range from mild diarrhea to dysentery with blood and mucus in the stool. E. histolytica is usually a commensal organism. Severe amoebiasis infections (known as invasive or fulminant amoebiasis) occur in two major forms. Invasion of the intestinal lining causes amoebic dysentery or amoebic colitis.

If the parasite reaches the bloodstream it can spread through the body, most frequently ending up in the liver where it causes amoebic liver abscesses. Liver abscesses can occur without previous development of amoebic dysentery. When no symptoms are present, the infected individual is still a carrier, able to spread the parasite to others through poor hygienic practices. While symptoms at onset can be similar to bacillary dysentery, amoebiasis is not bacteriological in origin and treatments differ, although both infections can be prevented by good sanitary practices. Transmission

Amoebiasis is usually transmitted by the fecal-oral route, but it can also be transmitted indirectly through contact with dirty hands or objects as well as by anal-oral contact. Infection is spread through ingestion of the cyst form of the parasite, a semi-dormant and hardy structure found in feces. Any non-

encysted amoebae, or trophozoites, die quickly after leaving the body but may also be present in stool: these are rarely the source of new infections. Since amoebiasis is transmitted through contaminated food and water, it is often endemic in regions of the world with limited modern sanitation systems, including México, Central America, western South America, South Asia, and western and southern Africa. Amoebic dysentery is often confused with “ traveler’s diarrhea” because of its prevalence in developing nations. In fact, most traveler’s diarrhea is bacterial or viral in origin. Prevention

To help prevent the spread of amoebiasis around the home :
Wash hands thoroughly with soap and hot running water for at least 10 seconds after using the toilet or changing a baby’s diaper, and before handling food. Clean bathrooms and toilets often; pay particular attention to toilet seats and taps. Avoid sharing towels or face washers.

To help prevent infection:
Avoid raw vegetables when in endemic areas, as they may have been fertilized using human feces. Boil water or treat with iodine tablets.
Avoid eating Street Foods especially in public places where others are sharing sauces in one container Good sanitary practice, as well as responsible sewage disposal or treatment, are necessary for the prevention of E. histolytica infection on an endemic level. E. histolytica cysts are usually resistant to chlorination, therefore sedimentation and filtration of water supplies are necessary to reduce the incidence of infection. Diagnosis of human illness

Immature E. histolytica/E. dispar cyst in a concentrated wet mount stained with iodine. This early cyst has only one nucleus and a glycogen mass is visible (brown stain). Asymptomatic human infections are usually diagnosed by finding cysts shed in the stool. Various flotation or sedimentation procedures have been developed to recover the cysts from fecal matter and stains help to visualize the isolated cysts for microscopic examination. Since cysts are not shed constantly, a minimum of three stools should be examined. In symptomatic infections, the motile form (the trophozoite) can often be seen in fresh feces. Serological tests exist and most individuals (whether with symptoms or not) will test positive for the presence of antibodies. The levels of antibody are much higher in individuals with liver abscesses. Serology only becomes positive about two weeks after infection. More recent developments include a kit that detects the presence of amoeba proteins in the feces and another that detects ameba DNA in feces. These tests are not in widespread use due to their expense. Amoebae in a colon biopsy from a case of amoebic dysentery.

Microscopy is still by far the most widespread method of diagnosis around the world. However it is not as sensitive or accurate in diagnosis as the other tests available. It is important to distinguish the E. histolytica cyst from the cysts of nonpathogenic intestinal protozoa such as Entamoeba coli by its appearance. E. histolytica cysts have a maximum of four nuclei, while the commensal Entamoeba coli cyst has up to 8 nuclei. Additionally, in E. histolytica, the endosome is centrally located in the nucleus, while it is usually off-center in Entamoeba coli. Finally, chromatoidal bodies in E. histolytica cysts are rounded, while they are jagged in Entamoeba coli.

However, other species, Entamoeba dispar and E. moshkovskii, are also commensals and cannot be distinguished from E. histolytica under the microscope. As E. dispar is much more common than E. histolytica in most parts of the world this means that there is a lot of incorrect diagnosis of E. histolytica infection taking place.

The WHO recommends that infections diagnosed by microscopy alone should not be treated if they are asymptomatic and there is no other reason to suspect that the infection is actually E. histolytica. Typically, the organism can no longer be found in the feces once the disease goes extra-intestinal. Serological tests are useful in detecting infection by E. histolytica if the organism goes extra-intestinal and in excluding the organism from the diagnosis of other disorders. An Ova & Parasite (O&P) test or an E. histolytica fecal antigen assay is the proper assay for intestinal infections. Since antibodies may persist for years after clinical cure, a positive serological result may not necessarily indicate an active infection. A negative serological result however can be equally important in excluding suspected tissue invasion by E. histolytica. Relative frequency of the disease

In older textbooks it is often stated that 10% of the world’s population is infected with Entamoeba histolytica. It is now known that at least 90% of these infections are due to E. dispar. Nevertheless, this means that there are up to 50 million true E. histolytica infections and approximately seventy thousand die each year, mostly from liver abscesses or other complications. Although usually considered a tropical parasite, the first case reported (in 1875) was actually in St Petersburg in Russia, near the Arctic Circle. Infection

is more common in warmer areas, but this is both because of poorer hygiene and the parasitic cysts surviving longer in warm moist conditions. Giardiasis

Giardiasis — popularly known as beaver fever — is a parasitic disease caused by the flagellate protozoan Giardia lamblia (also sometimes called Giardia intestinalis and Giardia duodenalis). The giardia organism inhabits the digestive tract of a wide variety of domestic and wild animal species, as well as humans. It is a common cause of gastroenteritis in humans, infecting approximately 200 million people worldwide. Giardia lamblia is a flagellated protozoan parasite that colonizes and reproduces in the small intestine, causing giardiasis. The parasite attaches to the epithelium by a ventral adhesive disc, and reproduces via binary fission. Giardiasis does not spread via the bloodstream, nor does it spread to other parts of the gastrointestinal tract, but remains confined to the lumen of the small intestine. Giardia trophozoites absorb their nutrients from the lumen of the small intestine, and are anaerobes.

If the organism is split and stained, its characteristic pattern resembles the familiar “ smiley face” symbol. Chief pathways of human infection include ingestion of untreated sewage, a phenomenon particularly common in many developing countries; contamination of natural waters also occurs in watersheds where intensive grazing occurs. The life cycle begins with a noninfective cyst being excreted with the faeces of an infected individual. The cyst is hardy, providing protection from various degrees of heat and cold, desiccation, and infection from other organisms. A distinguishing characteristic of the cyst is four nuclei and a retracted cytoplasm. Once ingested by a host, the trophozoite emerges to an active state of feeding and

motility. After the feeding stage, the trophozoite undergoes asexual replication through longitudinal binary fission.

The resulting trophozoites and cysts then pass through the digestive system in the faeces. While the trophozoites may be found in the faeces, only the cysts are capable of surviving outside of the host. Distinguishing features of the trophozoites are large karyosomes and lack of peripheral chromatin, giving the two nuclei a halo appearance. Cysts are distinguished by a retracted cytoplasm. This protozoan lacks mitochondria, although the discovery of the presence of mitochondrial remnants (organelles) in one recent study " indicate that Giardia is not primitively amitochondrial and that it has retained a functional organelle derived from the original mitochondrial endosymbiont". This organelle is now termed a mitosome. Signs and symptoms

Symptoms include loss of appetite, diarrhea, hematuria (blood in urine), loose or watery stool, stomach cramps, upset stomach, projectile vomiting (uncommon), bloating, flatulence, and burping (often sulphurous). Symptoms typically begin one to two weeks after infection and may wane and reappear cyclically. Symptoms are caused by Giardia organisms coating the inside of the small intestine and blocking nutrient absorption. Most people are asymptomatic; only about a third of infected people exhibit symptoms. Untreated, symptoms may last for six weeks or longer.

Symptomatic infections are well recognised as causing lactose intolerance, which, while usually temporary, may become permanent. Although hydrogen breath tests indicate poorer rates of carbohydrate absorption in those

asymptomatically infected, such tests are not diagnostic of infection. It has been suggested that these observations are explained by symptomatic giardia infection allowing for the overgrowth of other bacteria. Some studies have shown giardiasis should be considered as a cause of vitamin B12 Deficiency as a result of the problems caused within the intestinal absorption systems Transmission

Giardiasis is passed via the fecal-oral route. Primary routes are personal contact and contaminated consumables. The more susceptible are institutional or day-care workers, travelers, those eating improperly treated food or drink, and people who have contact with individuals already infected. It is a particular danger to people hiking or backpacking in wilderness areas worldwide, especially if they have no immediate access to medical supplies. Giardia is also suspected to be zoonotic—communicable between humans and other animals. Major reservoir hosts include beavers, dogs, cats, horses, humans, cattle and birds. Diagnosis

The mainstay of diagnosis of giardiasis is stool microscopy. This can be for motile trophozoites or for the distinctive oval G. lambliacysts. The entero-test uses a gelatin capsule with an attached thread. One end is attached to the inner aspect of the patient’s cheek, and the capsule is swallowed. Later, the thread is withdrawn and shaken in saline to release trophozoites which can be detected microscopically. A new immunologic test, enzyme-linked immunosorbent assay (ELISA), is now available. These tests are capable of a 90% detection rate or more. Because Giardia lamblia is difficult to detect, often leading to misdiagnoses, several tests should be conducted over a one-week period. Treatment

Drugs used to treat adults include metronidazole, albendazole and quinacrine. Furazolidone and nitazoxanide may be used in children. Treatment is not always necessary, as the body can defeat the infection by itself. The drug tinidazole can treat giardiasis in a single treatment of 2000 mg, instead of the longer treatment of the other medications listed. The shorter duration of treatment may also cause patient less distress. Tinidazole is now approved by the FDA and available to US patients.