Int. J. Adv. Res. Biol.Sci. 2(1): (2015): 53–65

International Journal of Advanced Research in Biological Sciences
ISSN : 2348-8069
www.ijarbs.com

Review Article

A Systematic Review on Neglected Important Protozoan Zoonoses

Yibeltal Muhie Mekonen[1, 2] and Simenew Keskes Melaku[1, 3, 4]*

[1]College of Veterinary Medicine and Agriculture, Addis Ababa University, Debre Zeit, Ethiopia
[2]College of Veterinary medicine, Jigjiga University, Jigjiga, Ethiopia
[3]College of Agriculture and Natural Resources, Dilla University, Dilla, Ethiopia
[4]College of Veterinary Medicine, Animal Resources and Biosecurity, Makerere University, Kampala, Uganda
*Corresponding Authors: *drsimenew@yahoo.com/simenew.keskes@aau.edu.et/simenew.keskes@gmail.com*

Abstract

Infectious protozoan parasites are transmitted to humans through several routes, including contaminated food and water, inadequately treated sewage/sewage products, and livestock and domestic pet handling. Several enteric protozoa cause severe morbidity and mortality in both humans and animals worldwide. In developed settings, enteric protozoa are often ignored as a cause of diarrheal illness due to better hygiene conditions, and as such, very little effort is used toward laboratory diagnosis. Although these protozoa contribute to the high burden of infectious diseases, estimates of their true prevalence are sometimes affected by the lack of sensitive diagnostic techniques to detect them in clinical and environmental specimens. Despite recent advances in the epidemiology, molecular biology, and treatment of protozoan illnesses, gaps in knowledge still exist, requiring further research. There is evidence that climate-related changes will contribute to their burden due to displacement of ecosystems and human and animal populations, increases in atmospheric temperature, flooding and other environmental conditions suitable for transmission, and the need for the reuse of alternative water sources to meet growing population needs. This review discusses the common enteric protozoa from a public health perspective, highlighting their epidemiology, modes of transmission, prevention and control and epidemiological pictures in Ethiopia. It also discusses the potential impact of climate changes on their epidemiology and the issues surrounding waterborne transmission and suggests a multidisciplinary approach to their prevention and control.

Keywords: Ethiopia, integrated approach, protozoan zoonoses, neglected, poverty, production loss

Introduction

Approximately 60 percent of all human pathogens are zoonoses of microbes that are naturally transmitted between animals and humans. Neglect of their control persists because of a lack of information and awareness about their distribution, a lack of suitable tools and managerial capacity for their diagnosis, and a lack of appropriate and sustainable strategies for their prevention and control. Furthermore, many of the most affected countries have poor or non-existent veterinary public health infrastructures. This situation has marginalized control of zoonoses to the gap between veterinary responsibilities and medical needs, generating a false perception that their burden and impact on society are low. As a result, neither the human and animal health resources nor the research needed for their control are available spawning a category of non zoonotic diseases (Choffnes and Relman, 2011).

The neglected tropical diseases (NTDs) are the most common conditions affecting the poorest 500 million people living in sub-Saharan Africa (SSA), and together produce a burden of disease that may be equivalent to up to one-half of SSA’s malaria disease burden and more than double that caused by tuberculosis (Hotez and Kamath, 2009).Starting with an initial set of 13-15 diseases, there are now over 40 helminth, protozoal, bacterial, viral, fungal and ectoparasitic infections covered under the brand-name of the neglected tropical diseases. Gaps in our understanding of the epidemiology and control of many of the neglected tropical diseases remain, which calls for additional funding for innovative research (Jürg et al, 2012).

The health and socioeconomic impacts of zoonotic parasitic and related food-borne diseases are growing continuously and increasingly being felt most particularly by developing countries. Apart from causing human morbidity and mortality, they hamper agricultural production, decrease availability of food, and create barriers to international trade (Solaymani-Mohammadi and Petri, 2006). The problem of zoonoses has spread from predominantly restricted rural areas into regional and, in some cases, worldwide epidemics. This is due to the great changes of the previous decades, especially the increasing urbanization, most of which is inadequate planned. In addition, large movements of populations, opening up of badly needed new areas for food production, the increasing trade in meat, milk and other products of animal origin, the increasing number and speed of vehicles, and even tourism have contributed to expanding the impact of zoonotic diseases (Solaymani-Mohammadi and Petri, 2006). The challenges of food-borne, waterborne, and zoonotic protozoan diseases associated with climate change are expected to increase, with a need for active surveillance systems, some of which have already been initiated by several developed countries (Hall et al, 2002; Jayhus et al, 2009). However, very little effects are attempting in the developing world which actually are the main victims.

Parasitic zoonoses (PZs) pose a significant but often neglected threat to public health, especially in developing countries. In order to obtain a better understanding of their health impact, summary measures of population health may be calculated, such as the Disability-Adjusted Life Year (DALY). However, the data required to calculate such measures are often not readily available for these diseases, which may lead to a vicious circle of under-recognition and under-funding (Devleesschauwer et al, 2014).

Cryptosporidiosis, giardiasis and amebiasis are the common cause of human diarrhoeal disease worldwide, and lead to significant morbidity and mortality in the world, particularly in developing nations. It occurs both in immuneocompetent and in immunocompromised individuals. It is generally believed that although these protozoan parasitic infections are distributed worldwide, their prevalence is higher in developing compared to developed countries. However, the relative importance of zoonotic infections especially in developing countries has not been studied in detail. The prevalence rates are generally higher in immunodeficient compared to immune-competent patients. However, most studies on prevalence have been carried out in developed countries where the laboratory and clinical infrastructure are more easily available. Protozoan pathogens and HIV interact in their host, modifying the immunopathology of disease and complicating therapeutic intervention. Disease prevalence and distribution and population movements impact greatly on HIV/protozoan parasite co-infections (Andeani et al, 2012).

In Ethiopia there are little reports regarding protozoan zoonoses. However, there are still reports from clinics and hospitals where these diseases are becoming major issues of concern. This review will examine published data on the neglected protozoan pathogens in Ethiopia and analyses their current importance to public health. Data on the prevalence of these infections in domestic and wild animals and evidence on zoonotic sources of transmission will be discussed too.

Important but Neglected Protozoan Zoonoses Dealt in this Critical Review

Amoebiasis

This disease is caused by a single cell protozoan parasite called *Entamoeba* spp. *(E. histolytica, E. polecki).* Invasive amebiasis is one of the world most prevalent and fatal infectious diseases. Around 500 million people are infected worldwide while 75,000 die of the disease annually. Behind malaria and schistosomiasis, amebiasis ranks third on the list of parasitic causes of death worldwide. The infection is common in developing countries and predominantly affects individuals with poor socioeconomic conditions, non hygienic practices, and malnutrition (Stanley, 2003).

In human most infections are asymptomatic. Clinical manifestations vary from intermittent periods of mucoid or bloody diarrhea to severe dysentery accompanied by pyrexia. With hepatic invasion, hepatomegaly, and abdominal pain and the case fatality rate in severe cases, and with hepatic abscessation-up to 100% without treatment. In animals usually subclinical, but colitis and diarrhea can occur, particularly in nonhuman primates (Hugh-Jone et al, 1995). Its occurrence is worldwide but most common in tropical areas with poor sanitation. It is common in nonhuman primates, especially in Asia and Africa. E. *polecki* is a parasite of swine (Solaymani-Mohammadi and Petri, 2006).

Amebiasis in Ethiopia

A number of survey and routine diagnosis in Ethiopia indicate that amebiasis is one of the most widely distributed diseases. In a countrywide survey of amebiasis in 97 communities, the overall prevalence of *Entamoeba histolytica* infections, as measured by rate of cyst-passers, in schoolchildren and non-school communities were 15.0% and 3.5%, respectively (Erko et al, 1995). A study conducted on the prevalence of *Entamoeba histolytica/dispar* among children in Legedini, Adada and Legebira, Dire-Dawa administrative region was 33.7% (Dawit, 2006 Unpublished MSc Thesis).

The mail transmission is by ingestion of food or water contaminated with cysts from asymptomatic individuals. (Trophozoites, passed by acute cases, are too fragile to survive outside the host (Hugh-Jone et al, 1995). Microscopic examination of fresh feces from diarrheic patients for trophozoites or cysts in feces of asymptomatic individuals is a confirmatory test at laboratory levels. Treatment with an amebicide is diagnostic when clinical signs are reduced. Radiology and/or hemagglutination tests of sera for evidence of extra intestinal infection can also be used to screen the disease.

Giardiasis

Giardiasis is caused by *Giardia lamblia* (also known as *Giardia duodenalis* or *G. intestinalis*) is a unicellular, flagellated intestinal protozoan parasite of humans isolated worldwide and is ranked among the top 10 parasites of man (Farthing and Kelly, 2005). Its occurrence is worldwide (Figure 1) and prevalence very high in areas with poor sanitation and in institutions. Human infections usually originate from other humans but may result from contact with dogs, cats, rodents, beavers, or nonhuman primates. The prevalence of the disease varies from 2% to 5% in developed to 20% to 30% in developing countries. The variation in prevalence might be attributed to factors such as the geographical area, the urban or rural setting of the society, the age group composition and the socio-economical conditions of the study subject.

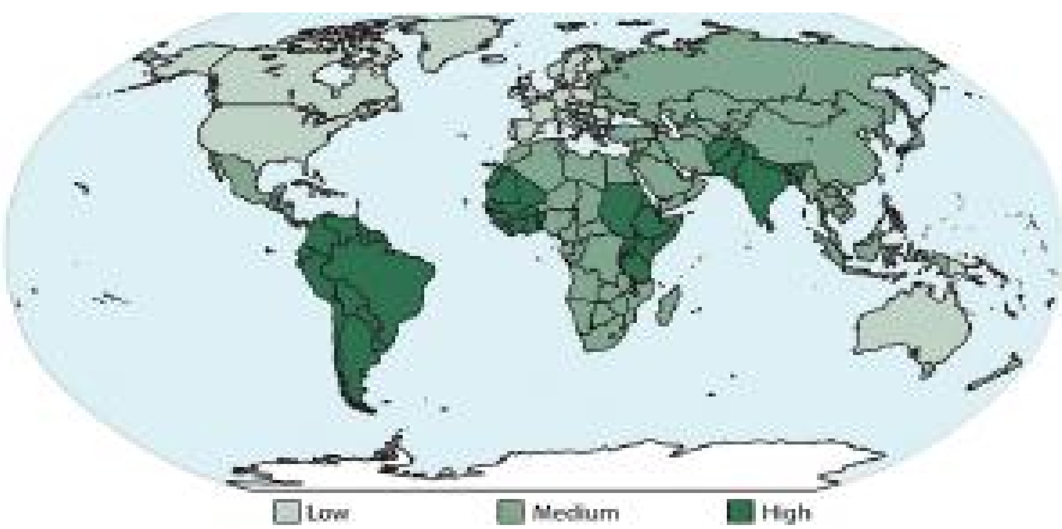

Figure 1: Risk of disease caused by *Giarda* species with different degrees
Source: Esch and Petersen (2013)

Giardiasis in Ethiopia

According to Birrie and Erko (1995) based on a countrywide survey of giardiasis, the overall prevalence among school children and residents were 8.9% and 3.1%, respectively and that of the non-school children were 4.4%. Recent report indicates that the prevalence of *Giardia lamblia* among diarrhoea patients referred to EHNRI (Ethiopian Health and Nutrition Research Institute) was 8.6% (Endeshaw et al, 2004). In a study conducted in South Western Ethiopia, the prevalence of Giardiasis was 13.7% (Ali et al, 1999). A study conducted for the determination of Prevalence of Giardiasis and Cryptosporidiosis among children in relation to water sources in selected Village of Pawi Special District in Benishangul-Gumuz Region, Northwestern Ethiopia sowed that out of the 384 children examined, 102 (26.6%) for giardiasis (Tigabu et al, 2010).

Ingestion of cysts (trophozoites are too fragile to survive outside host) on food, or by direct fecal-oral transfer are main means of transmissions and commonly waterborne, including municipal outbreaks (Hugh-Jone et al, 1995). Human infections with *G. duodenalis* belong to two genotypes, A and B based on specific signal sequences in the 5' end of the small subunit (16S) ribosomal RNA gene. As they are found in faecal samples from domestic and wild animals and in the environment, these two genotypes are widespread and possibly zoonotic. It is now shown that the re-emergence of zoonotic human giardiasis corresponds mainly to the genotypes A-I and to a lesser extent to genotype B. There may also be a correlation between these genotypes and pathogenicity. The major source of infection is through faecal-oral route although large outbreaks have been through contaminated water (Thompson et al, 2008).

Humans are the principal reservoir of infection, but *Giardia lamblia* can infect dogs, cats, beavers, and other animals. These animals can contaminate water with feces containing cysts that are infectious for humans. The transmission of *Giardia* to humans is dependent upon the ingestion of cysts excreted in the feces of infected persons or animals (Thompson, 1994). Laboratory-confirmed giardiasis shall be defined as the detection of *Giardia* organisms, antigen, or DNA in stool, intestinal fluid, tissue samples, biopsy specimens, or other biological sample. Tinidazole, metronidazole, nitazoxanide, paromomycin, furazolidone, and quinacrine are known to be effective in treating giardiasis. Because making a definitive diagnosis is difficult, empiric treatment can be used in patients with the appropriate history and typical symptoms.

Leishmaniasis

Leishmaniasis is an ancient disease caused by protozoans from the *Leishmania* genus and transmitted by the bite of a sand fly. It has four subtypes of varying severity, which include cutaneous and visceral infections. Cutaneous infection results in the formation of disfiguring lesions which frequently occur on the face, arms and legs. Lesions may last anywhere from a few weeks to over a year; secondary lesions may also occur years after the initial lesion has healed (Heymann, 2004). Visceral cases can result in anaemia, fever, debility and death if left untreated (Kindhauser, 2002).

The causative organisms of leishmaniasis or leishmaniosis are species of the genus *Leishmania* (Kinetoplastida, Trypanosomatidae) (WHO, 2010) and these parasitic unicellular protozoans are usually transmitted between vertebrate hosts by the bite of blood sucking female phlebotomine sand flies (Diptera, Psychodidae) (Ready, 2013). Parasites of the subgenus *Sauroleishmania* are considered to be restricted to lizards, and most lizard-feeding sand flies do not usually bite humans. About 20 species of *Leishmania* infect mammals and many of them can cause human leishmaniasis. Motile infective forms of the parasite (metacyclic promastigotes with a long free flagellum) develop in the guts of competent sand fly vectors, which inoculate them into mammalian skin. Infections can spread, often via the lymphatic system, to cause secondary dermal lesions with forms and tissue tropisms in humans that show some parasite species specificity.

Human cutaneous leishmaniasis is caused by most *Leishmania* species in the subgenus *Leishmania*, which occur in most subtropical and tropical regions (for example *Leishmania (Leishmania) major* from Africa and Asia, and *Leishmania (Leishmania) mexicana* from Central and South America), and by many species in the subgenus *Viannia*, which are restricted to Latin America (for example *Leishmania (Viannia) brasiliensis*) (Figure 2). Any parasite causing cutaneous leishmaniasis can visceralize (for example *Leishmania (Leishmania) tropica*, which normally causes Oriental sore), but only two species of the subgenus *Leishmania* routinely do so, and these are the causative agents of most human visceral leishmaniasis (VL) worldwide (Ready, 2014).

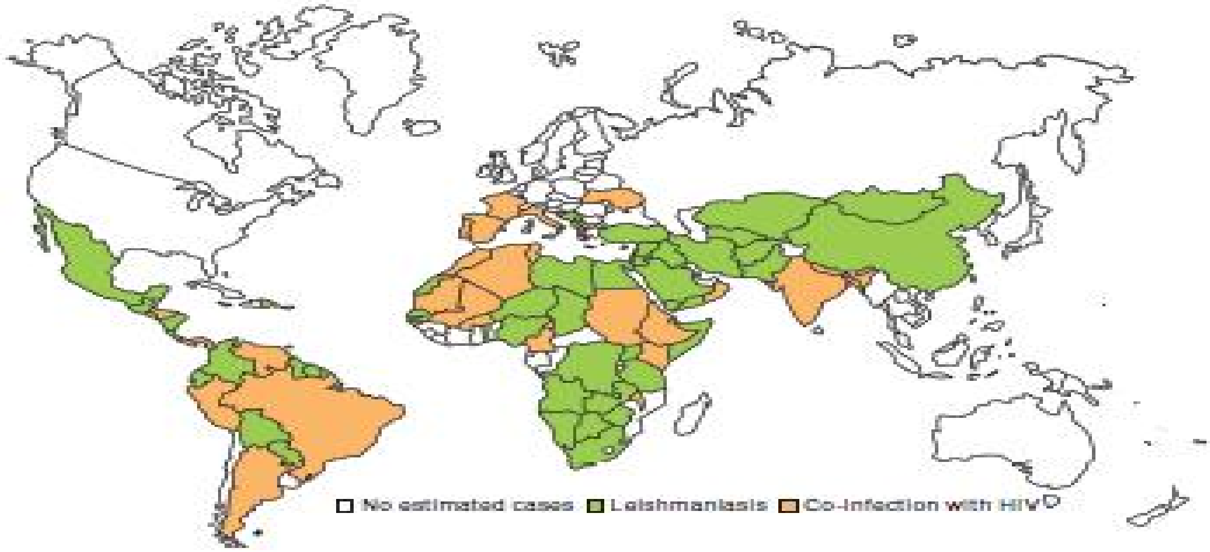

Figure 2: Global burden of *Leishmania* as adapted from the "Leshimaniases and *Leishemania* HIV co-infection" WHO fact sheet No. 116, May 2000.

In human cutaneous-erythematous papule on exposed portions of body which develops into a slowly healing ulcer. Visceral-Progressive weakness, intermittent fever, splenomegaly, anorexia, weight loss, hair loss, bleeding from gums and mucous membranes. In animals frequently the disease is subclinical. Skin lesions have been observed on dogs and horses. Old World type usually not fatal but New World type may result in face lesions involving mucocutaneous junction with secondary bacterial infection resulting in death. Visceral type if untreated is usually fatal (Hugh-Jone et al, 1995).

The occurrence of Cutaneous (Old World) leishmaniasis is mainly in Africa, Asia and southern Europe. However, the New World visceral leishmaniasis is in Latin America, southern United States. Of course the visceral form also is common in Asia, Africa, Europe and Latin America. Dermal leishmanoid is a condition in which dermal lesions can be heavily parasitized in a subset of patients successfully treated for VL, and it occurs mainly in India and Sudan in patients infected by *L. donovani* (John and Petri, 2006; Mandal and Khan, 2011; McGwire and Satoskar, 2014). They remain asymptomatic for months to years and then develop a progressive proliferation of parasites within the skin, giving rise to diffuse macular, maculopapular, or nodular lesions (McGwire and Satoskar, 2014). Lesions appear anywhere on the body, but they often occur on the face (Barrett and Croft, 2012).

Leishmaniasis in Ethiopia

Both VL and CL are important endemic vector-borne diseases in Ethiopia. The Federal Ministry of Health (FMoH) estimates the annual burden of VL to be between 4,500 and 5,000 cases (FMoH Ethiopia, 2006 unpublished). Known VL endemic foci are in the arid south-west, and the Humera and Metema lowlands in the north-west. The definitive reservoir of VL in Ethiopia remains unknown, although transmission is assumed to be anthroponotic (WHO, 1998). CL is confined mainly to the highlands, although occasional cases have occurred in the Omo Valley. About 2-12% of all visceral leishmaniasis cases involve HIV co-infections underlines the synergic aspect of both diseases; such proportions may reach 40%, as in Humera, northwest Ethiopia (WHO, 2007), where co-infections have increased two-fold in the last decade (Andreani et al, 2012).

In 2005, an outbreak of VL in Libo Kemkem woreda, a highland area of Amhara regional state, was identified by MSF-Greece. The outbreak began in Bur district in 2003, with cases peaking in 2005 and occurring mainly in Libo Kemkem and Fogera woredas, ultimately becoming a low-incidence endemic area by 2007 (Herrero et al, 2009). By 2007, around 2,450 primary cases and 120 deaths had been reported since the outbreak began in 2003, with the majority of cases treated at Addis Zemen health centre (Bashaye et al, 2009).

The southwest foci include the Omo plains, Aba Roba plains and Weyto River Valley in Southern Nations and Nationalities People's Region (SNNPR) all areas of lowland savannah with low rainfall. The main focus in the southwest occurs in the lower course of the Rift Valley, most notably the Segen (Aba Roba focus) and Weyto valleys in the drainage basin of the Chew Bahir Lake, near Konso woreda. Cases in this area peak during the wet seasons, from February to May, and September to October (Gebre-Michael and Lane, 1996). From January 1999 to July 2004, there were 8,049 admissions and 1,334 deaths within the medical wards of Gondar University Hospital. Of these, 221 were visceral leishmaniasis cases, accounting for about 2.8% of the admissions and 52(3.9%) deaths in the medical wards (Mengistu and Ayele, 2007).

Medical records from patients with Leishmania attending the Italian Dermatological Centre of Mekele in the period 2005-2008 were retrospectively reviewed and 471 patients were affected by clinical form of cutaneous leishmaniasis (86%); climatic and environmental changes occurring in this region and land degradation are discussed as factors influencing leishmaniasis distribution (Morrone et al, 2011). CL due to *L. aethiopica* manifests itself in three forms, all of which are found in Ethiopia: localised cutaneous (LCL), ML and diffused cutaneous leishmaniasis (DCL) (Gebre-Michael et al, 2004). Infection is largely transmitted by *P. longipes*, although *P. pedifer* is also known to transmit the infections. CL has been extensively studied in the western highlands and lake areas of the Rift Valley. The main areas of transmission include the Ochollo focus in the Rift Valley escarpment above Lake Abaya, the Kutaber area in the eastern Ethiopian plateau near Dessie, the Aleku area of Wollega zone, the south-west highlands of Bale and Sidamo, and the Sebeta area near Addis Ababa. The Ochollo focus is highly endemic for the disease, with higher prevalence in younger age groups. An outbreak in Silti woreda, SNNPR, 150km south of Addis Ababa, was reported in 2005. A CL prevalence of 4.8% was recorded, which exceeded prevalence rates previously reported from Ochollo (Negera et al, 2008; Dawit et al, 2012).

Leishmaniasis may be diagnosed by microscopic visualization of the amastigotes (Leishman-Donovan bodies) in marrow, spleen, lymph nodes or skin lesions (Sundar & Rai, 2002). Enzyme-linked immunosorbent assay (ELISA), antigen coated dipsticks, and the direct agglutination tests (DAT) are available but have variable sensitivity and specificity. DAT has been found to be a sensitive inexpensive test but requires careful manipulation and eight hours of incubation which limits its application. Nucleic acid amplification methods have been developed but are not readily available in developing countries.

Effective drugs have been used in humans. A fundamental issue is the difficulty to capture and treat stray dogs- Drugs currently exist (pentavalent antimonials, miltefosine), which are used in humans, but there is reduced efficiency in dogs (Evans and Kedzierski, 2012) leading to the need for standardisation of treatment protocols for dog treatment. There are also perceived opportunities where the stray dog problem can be controlled (Reithinger and Davies, 2002). Drug combinations should be considered.

WHO-recommended mitigation strategies include treatment of infected humans, culling of seropositive dogs and use of insecticides to reduce household sand fly burdens. Two vaccines are available in Brazil for immunization of dogs and available data suggest that one of these delivers an additive effect, in conjunction with dog culling, on the incidence of disease in children (Palatnik-de-Sousa et al, 2009; Costa et al, 2011). A promising human vaccine is in phase II/III clinical trials, which raises the prospect of a vaccine strategy targeting both humans and the reservoir host (Naigill and Kaur, 2011; Evans & Kedzierski, 2012).

Cryptosporidiosis

The causes of this disease are *Cryptosporidium* spp. *(C. parvum,* possibly others). In humans, abdominal pain, nausea, watery diarrhea lasting 3-4 days. In immune-deficient or immune-suppressed people, the disease is severe, with persistent diarrhea (6-25 evacuations per day) and malabsorption of nutrients. In normal persons the disease is self-limiting. In immune-compromised individuals, disease is severe and case fatality rate may be high. In animals normally a clinical disease can be seen only among young neonates. In ruminants, gastroenteritis and diarrhea are common. A respiratory syndrome among chicken and turkey were also reported. Its occurrence is worldwide and common in domestic livestock and birds (Hugh-Jone *et al*., 1995).

Cryptosporidiosis in Ethiopia

The disease burden of Cryptosporidium parvum and role of zoonotic transmission in cryptosporidiosis epidemiology are poorly understood in developing countries. Current report showed 26.9% of HIV/AIDS patients tested for cryptosporidiosis are positive (Adamu et al, 2014) which is close prevalence with report of 25.9% by Endeshaw et al (2004). A study also conducted for the determination of Prevalence of Giardiasis and Cryptosporidiosis among children in relation to water sources in selected Village of Pawi Special District in Benishangul-Gumuz Region, Northwestern Ethiopia sowed that out of the 384 children examined, 31 (8.1%) for cryptosporidiosis (Tigabu et al, 2010).

In Ethiopia HIV/AIDS patients with low CD4+ cell counts had an extremely high occurrence of Cryptosporidium infection, even when they were on highly active antiretroviral therapy HAART. Although the majority of cryptosporidiosis cases were caused by *C. parvum*, there was a high diversity of Cryptosporidium species, with a significant number of cases caused by the newly recognized *C. viatorum*. These *Cryptosporidium* spp. and *C. parvum* subtypes were linked to different clinical manifestations. Therefore, improved hygiene and avoidance of calf contact among this population should be advocated to reduce the occurrence of Cryptosporidium infections, especially those caused by *C. parvum* IIa subtypes of calves (Adamu et al, 2014). Oocysts are infective when passed in feces. The disease is not species specific (strain from one animal can infect many other species). Fecal-oral transmission from infected animals or humans is the route. A confirmatory test is Microscopic examination of fresh feces for the identification of oocysts (Hugh-Jone et al, 1995).) Improved hygiene and avoidance of calf contact should be advocated to reduce cryptosporidiosis transmission in HIV/AIDS patients in the study settings (Adamu et al, 2014).

Toxoplasmosis

Toxoplasmosis is among the global major zoonotic diseases (Torgerson and Macpherson, 2011) and the third leading cause of food-related deaths in the USA. It is caused by Toxoplasma gondii, an Apicomplexa protozoan parasite, with cats as the definitive host, and warm-blooded animals as intermediate hosts. Cats are considered the key in the transmission of Toxoplasma gondii to humans and other animals because they are the only hosts that can excrete the environmentally resistant oocysts in their feces (Dubey, 2010).

Humans get infections with *T. gondii* after ingesting raw or undercooked meat, by ingesting cat-shed oocysts via contaminated soil, food or water; or congenitally by transplacental transmission of tachyzoites (Tenter et al, 2000; Dubey, 2010; Torgerson and Macpherson, 2011). Infection with *T. gondii* during pregnancy can result in fetal death, neonatal death or various congenital defects, such as hydrocephalus, central nervous system abnormalities and chorioretinitis (Dubey, 2004; Dubey, 2010).Toxoplasmosis is also a serious problem in immunocompromised patients (Dubey, 2004; Jones et al, 2009). In addition, recent studies have indicated that toxoplasmosis is a plausible risk factor for personality shifts and increased likelihood of reduced intelligence or schizophrenia. Recently, highly virulent genetically atypical strains of *T. gondii* have been incriminated with pneumonia, even in immunocompetent individuals (Leal et al, 2007). Serological screening for *T. gondii* antibodies should be one in women of child-bearing age as it allows identification of women at risk of acquiring infection and is part of a strategic approach for prevention of congenital toxoplasmosis (Remington *et al*., 2006). The seroprevalence of *T. gondii* infection among women of child-bearing age in different countries (Figure 3) ranges from 4 to 100%. In Africa, prevalence ranges from 25% in Burkina Faso to 75% in Sao Tome and Principe (Hugh-Jone et al, 1995; Torgerson and Macpherson, 2011).

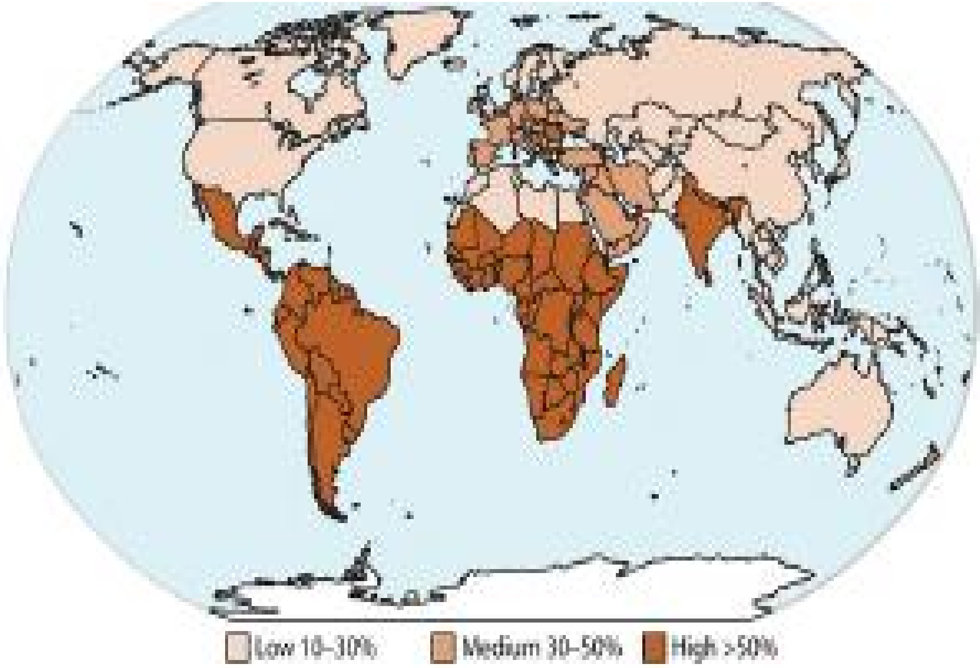

Figure 3: Human seroprevalece of *Toxoplasma gondii*
Source: Esch and Petersen (2013)

Toxoplasmosis in Ethiopia

In Ethiopia there are few studies under taken in different parts of the country on different species of animals and human. The highest prevalence was recorded in human (90%) as shown in table 1 below.

Table 1: Prevalences of *T. gondii* in Ethiopia

Study area	Prevalence (%) of different study subjects					Test used	References
	Cattle	Sheep	Goat	Cat	Human		
Central Ethiopia	6.6	11.9	22.9	-	-	MDAT	Bekele and Kasali (1989)
Debre Birhan	-	35	34	-	-	MDAT	Tilaye and Getachew (2002)
Nazareth		52.6	24	-		MDAT	Negash et al (2004)
East and West Shewa Zones			19.7			ELISA	Endrias et al (2013)
Nazareth					60		Negash et al (2008)
Addis Ababa					90	ELISA	Teshale et al (2009)
South Omo, North Omo and East Shewa Zones			74.9				Teshale et al (2007)
Jimma town					83.6	ELISA	Endalew et al (2012)
Centeral Ethiopia					81.4	ELISA	Endrias et al (2013)

Source: Yibeltal and Simenew (2014)

There is also a report on isolation of viable *Toxoplasma gondii* from tissues and feces of cats from Addis Ababa, Ethiopia. In total, viable *T. gondii* was isolated from 27 of the 36 cats, and these isolates were designated TgCatEt1 to TgCatEt27 and this is the first report of isolation of viable T. gondii from any host in Ethiopia (Dubey et al, 2013). In addition to this there is Four genotypes were recognized, including ToxoDB #1 (Type II clonal, nine isolates), ToxoDB #2 (Type III, five isolates), Toxo DB #3 (Type II variant, ten isolates), and ToxoDB #20 (nine isolates) study conducted from tissues and feces of 27 cats from Ethiopia (Dubey et al, 2013).

Generalities of these Protozoan Zoonoses

The clinical impact of zoonotic enteric protozoan infections is greatest in the developing world where inadequate sanitation, poor hygiene and proximity to zoonotic reservoirs, particularly companion animals and livestock are greatest (Figure 4). In such circumstances, it is not surprising that infections with more than one species of enteric protozoan are common, and in fact single infections are rare (Thompson and Smith, 2011). Unfortunately the impact of polyparasitism in the context of enteric protozoan infections has not been considered and has not been adequately taken into account (Ouattara et al, 2008).

Several reports signify that zoonotic disease and poverty are interrelated entities. There are complex relationship between zoonotic disease and poverty (Figure 5). Diseases of livestock and zoonoses affect production and reduce market access. Interventions against these diseases, whether they are aimed at reducing losses or reducing human health effects, may be expensive to producers. All of these may contribute to maintaining producer communities in poverty. Moving to the right of this diagram, animal diseases may also affect health, by reducing the nutritional benefits of animal products, whose micronutrients are particularly important in child development, and by causing disease in humans, with its consequences of morbidity and mortality, reduced labour and income, and the costs of treating these diseases. Both agricultural effects and health effects therefore may contribute to the persistence of poverty in poor populations associated with animal production.

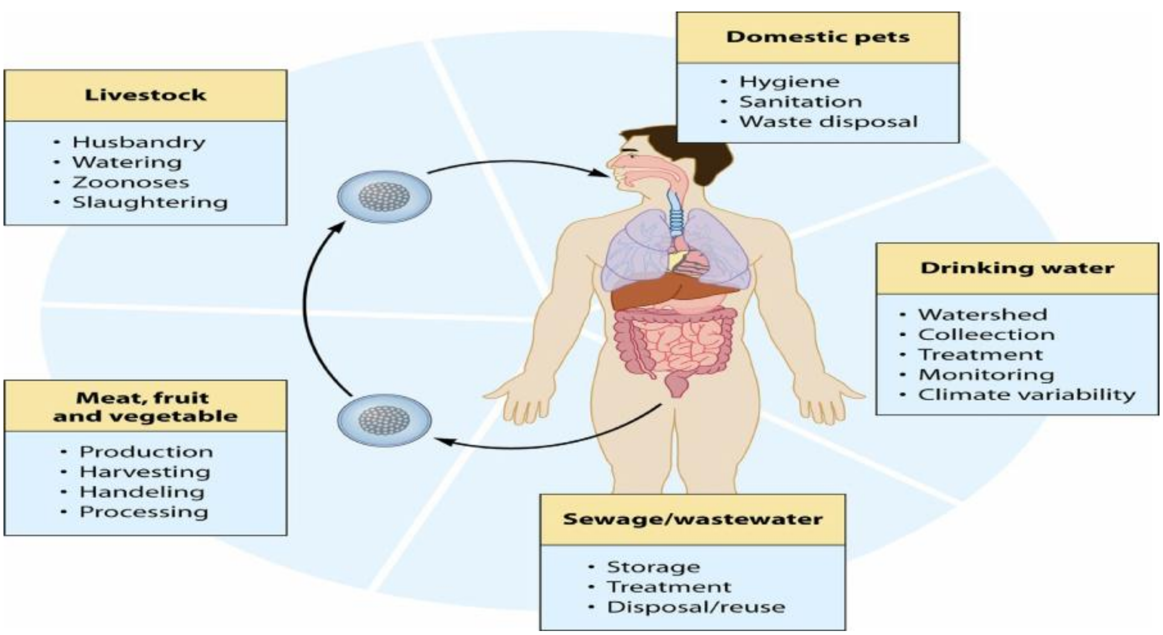

Figure 4: Complex interactions in the transmission and control of enteric protozoan infections. Infectious parasites are transmitted to humans through several routes, including contaminated food and water, inadequately treated sewage/sewage products, and livestock and pet handling. Prevention and control can be implemented at different levels of food production, liquid waste management water quality control, and careful livestock and pet handling process.

Source: Stephanie et al (2012)

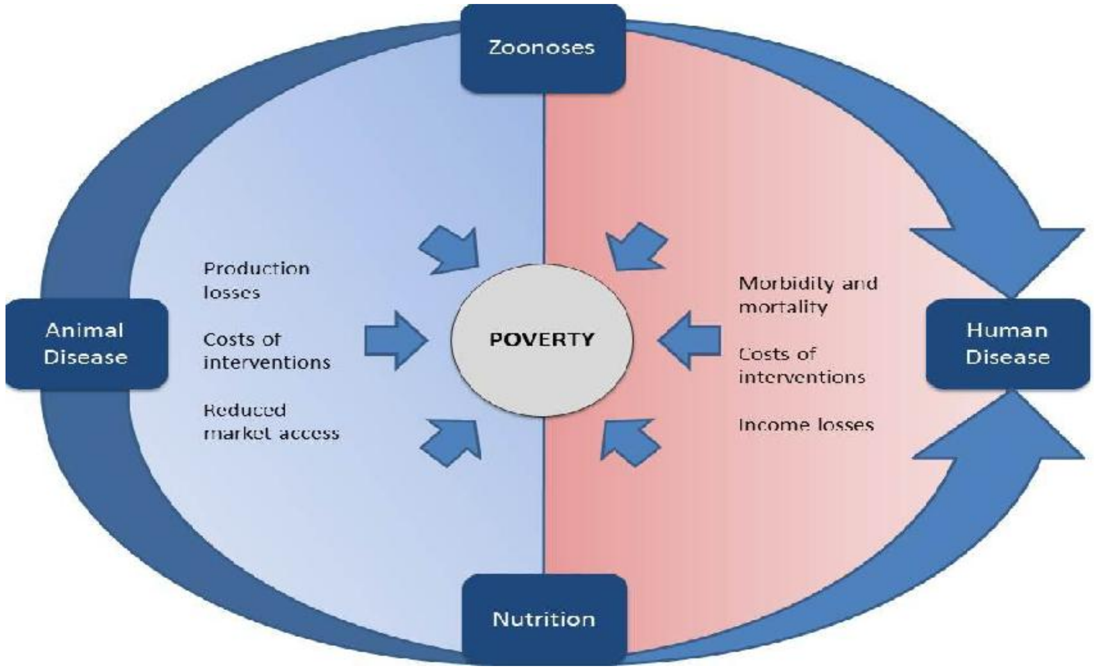

Figure 5: Impact of animal disease on human health

Source: Kock et al (2012)

Conclusion

The protozoan zoonoses circulating in Ethiopia are major burden on public health and wellbeing. The magnitude and scope of this burden varies for each of the protozoan parasites discussed in this manuscript. Apart from causing human morbidity and mortality, they hamper agricultural production, decrease availability of food, and create barriers to international trade. It is generally believed that although these parasitic infections are distributed worldwide, their prevalence is higher in developing compared to developed countries. However, the relative importance of zoonotic infections especially in developing countries has not been studied in detail including. These protozoan zoonoses are the most neglected but very important in terms of human health and veterinary concerns. The main share belongs to cryptosporidiosis; giardiasis, toxoplasmosis, leishmaniasis and amebiasis are some of the major ptorozoan zoonoses. Despite their significant effect in human and animal health and economic loses, very few studies have been conducted in Ethiopia. Treatment inefficiency, greater drug resistance, misdiagnoses and the exacerbation of both parasitemia and AIDS progression in co-infected individuals all call for a greater focus on these emerging HIV opportunistic diseases. Epidemiological surveillance may be a critical component of public health and is essential not only for the early identification of emerging diseases and trends but also for resource planning and measuring the impact of control strategies. Strengthened communication and cooperation among professionals in human, animal and environmental health areas would be particularly valuable as we seek to predict, recognize, and mitigate the impact infectious disease, including food borne illness. Promotion of such cooperation worldwide would result in holistic, multi-disciplinary solutions to difficult problems for which a single discipline may not address. Such an initiative of “One Health” concept has already been launched in the US. “There is no scientific barrier nor should there be, between Veterinary medicine and Human medicine” (Saunders, 2000). Community-based surveillance systems and the strengthening of existing health care infrastructures will be important steps to take in dealing with the neglected tropical diseases as clearly articulated by WHO (2007) of their global plan to combat neglected tropical diseases from 2008-2015. The new Pan-African, “One Health platform on neglected zoonotic diseases (OH NZD)” currently being initiated as the platform of Advocacy for Neglected Zoonotic Diseases (ADVANZ) will hopefully play great roles in Africa among the poor people.

References

Adamu, H., B. Petros, G. Zhang, H. Kassa, S. Amer, J. Ye, Y. Feng and Xiao, L. 2014. Distribution and Clinical Manifestations of Cryptosporidium Species and Subtypes in HIV/AIDS Patients in Ethiopia. *PLoS Negl. Trop. Dis*. **8(4):** e2831.

Ali, I., G. Mekete and Wodajo, N. 1999. Intestinal parasitism and related risk factors among students of Asendabo Elementary and Junior Secondary school, south western Ethiopia. *Ethiop. J. Health Dev.* **13(2)**: 1-5.

Andreani, G., R. Lodge, D. Richard and Tremblay, M. J. 2012. Mechanisms of interaction between protozoan parasites and HIV. *Curr. Opin. HIV AIDS*. **7(3):** 276-282.

Barrett, M.P., and Croft, S.L. 2012. Management of trypanosomiasis and leishmaniasis. *Br Med Bull*.**104: 1**75-196.

Bashaye, S., N. Nombela, D. Argaw, A. Mulugeta, M. Herrero, J. Nieto, C. Chicharro, C. Canavate, P. Aparicio, I. D. Velez, J. Alvar and Bern C. 2009. Risk factors for visceral leishmaniasis in a new epidemic site in Amhara Region, Ethiopia. *Am J Trop Med Hyg.* **81:** 34-39.

Bekele, T., and Kasali, O.B. 1989. Toxoplasmosis in sheep, goats and cattle in central Ethiopia, *Veterinary Research Communications*, **13 (5):** 371-375.

Birrie, H., and Erko, B. 1995. Giardiasis in Ethiopia. *Ethiop. J. Health Dev.* **9(1)**: 77-80.

Choffnes, E. R., and Relman, D. A. 2011. The Causes and Impacts of Neglected Tropical and Zoonotic Diseases: Opportunities for Integrated Intervention Strategies: Workshop Summary. pp. 1-605.

Costa, C. H. N., N. C. Peters, S. R. Maruyama, E. C. de Brito, and Santos, F. D. M. 2011. Vaccines for the leishmaniases: proposals for a research agenda. *PLoS neglected tropical diseases*. ***5*(3):** e943.

Dawit, G., Z. Girma, and Simenew, K. 2012. A Review on Biology, Epidemiology and Public Health Significance of Leishmaniasis. *Acta Parasitologica Globalis.* **3(3):** 43-53.

Devleesschauwer, B., A. Ale, P. Torgerson, N. Praet, C. Maertens de Noordhout, D.P. Basu, B.P. Sher, L. Rob, V. Jozef, D.J.Durga, H. H. Arie, D. Luc, D. Pierre and Niko, S. 2014. The Burden of Parasitic Zoonoses in Nepal: A Systematic Review. *PLoS. Negl. Trop. Dis*. **8(1):** 1-13.

Dubey J.P., 2004. Toxoplasmosis- a waterborne zoonosis. *Vet Parasitol*. **126:** 57-72.

Dubey J.P., 2010. Toxoplasmosis of animals and humans. 2nd edition. Beltsville, Maryland: U.S.A. CRC Press.

Dubey, J. P., C. Darrington, N. Tiao, L. R. Ferreira, S. Choudhary, B. Molla, W. J. A. Saville, G. Tilahun, O.C.H. Kwok and Gebreyes, W. A. 2013. Isolation of Viable Toxoplasma gondii from Tissues and Feces of Cats from Addis Ababa, Ethiopia. *J. Parasitol*. **99(1):** 56-58.

Dubey, J.P., S. Choudhary, G. Tilahun, N. Tiao, W.A. Gebreyes, X. Zou and Su, C. 2013. Genetic diversity of Toxoplasma gondii isolates from Ethiopian feral cats. *Vet. Parasitol.* **196(1-2):** 206-208.

Endalew, Z.**,** Y. Delenasaw, A. Solomon, B. Tariku, S. Abdi and Ahmed, Z. 2012. Seroprevalence of *Toxoplasma gondii* and associated risk factors among pregnant women in Jimma town, Southwestern Ethiopia. *BMC Infectious Diseases***, 12(337):** 1-6.

Endeshaw, T., H. M. Mohamod, and Tilahun, W. 2004. *Cryptosporidium parvum* and other intestinal parasites among diarrheal patients referred to EHNRI in Ethiopia. *Ethiopian Medical Journal*. **42**:195-198.

Endrias, Z. G., H.A. Anteneh, S.T. Tesfaye, D.T. Kassu, M. Girmay, M. Vitale, V. Di Marco, E. Cox and Dorny, P. 2013. Seroepidemiology of Toxoplasma gondii infection in women of child-bearing age in central Ethiopia. BMC Infectious Diseases. **13(101):** 1-9.

Endrias. Z., A. Abebe, T.S. Tesfaye, T. Getachew, G. Medhin, M. Vitale, V. Di Marco, E. Cox, J. Vercruysse and Dorny, P. 2013. Seroepidemiological study of caprine toxoplasmosis in East and West Shewa Zones, Oromia Regional State, Central Ethiopia. *Res. Vet. Sci*. http://dx.doi.org/10.1016/j.rvsc.2012.07.020.

Erko, B., H. Birrie, and Tedla, S. 1995. Amebiasis in Ethiopia. *Trop. Geogr. Med.* **47(1),**30-32

Esch, J. K., and Peteresen, C.A. 2013. Transmission and Epidemiology of Zoonotic Protozoal Diseases of Companion Animals. *Clinical Microbio. Rev.* **26(1):** 58-85.

Evans, K. J., and Kedzierski, L. 2012. Development of Vaccines against Visceral Leishmaniasis. *Journal of tropical medicine*. Hindawi Publishing Corporation. pp.1-14.

Farthing, M.J.G., and Kelly, P. 2005. Protozoal gastrointestinal infections. *Medicine*. **33**: 81-83.

Gebre-Michael, T., and Lane, R. P. 1996. The roles of Phlebotomus martini and P.celiae (Diptera: Phlebotominae) as vectors of visceral leishmaniasis in the Aba Roba focus, southern Ethiopia. *Med. Vet. Entomol.* **10:** 53-62.

Gebre-Michael, T., M. Balkew, A. Ali, A. Ludovisi and Gramiccia, M. 2004. The isolation of Leishmania tropica and *L. aethiopica* from Phlebotomus (Paraphlebotomus) species (Diptera: Psychodidae) in the Awash Valley, northeastern Ethiopia. *Trans. R. Soc. Trop. Med. Hyg.* **98;** 64-70.

Hall, G.V., R.M. D'Souza and Kirk, M. D. 2002. Foodborne disease in the new millennium: out of the frying pan and into the fire? *Med. J. Aust.* **177**: 614-618.

Herrero, M., G. Orfanos, D. Argaw, A. Mulugeta, P. Aparicio, F. Parreno, O. Bernal, D. Rubens, J. Pedraza, M. A. Lima, L. Flevaud, P. P. Palma, S. Bashaye, J. Alvar and Bern, C. 2009. Natural history of a visceral leishmaniasis. *Am J Trop Med Hyg.* **81(3):** 373-377.

Heymann, D.L., 2004. Control of communicable diseases manual (18th ed.). Washington, DC: American Public Health Association.

Hotez, P.J., and Kamath, A. 2009. Neglected Tropical Diseases in Sub-Saharan Africa: Review of Their Prevalence, Distribution, and Disease Burden. *PLoS. Negl. Trop. Dis.* **3(8):** 1-10.

Hugh-Jones, M. E., W. T. Hubbert and Hacstad, H. V. 1995. Zoonoses: Recognition, control, and prevention. *A Blackwell Publishing Company* intestinal parasites among diarrhoeal patients referred to EHNRI in Ethiopia. *Ethiop. Med. J.* **42**:195-198.

Jaykus, L-A., M. Woolridge, J. M. Frank, M. Miraglia, A. McQuatters-Gollop, C. Tirado, R. Clarke and Friel, M. 2009. Climate change: implications for food safety. FAO, Rome, Italy. pp.1-49.

John, D.T., and Petri, W.A., 2006 *Markell and Voge's Medical Parasitology*. 9th ed. Missouri: Saunders/Elsevier Inc.pp.127-139.

Jones J.L., V. Dargelas, J. Roberts, C. Press, J.S. Remington and Montoya J.G. 2009. Risk factors for *Toxoplasma gondii* infection in the United States. Clin. Infect. Dis. **49**: 878-884.

Jürg, U., L. B. Sören, K. Stefanie, B. Johannes, L. N. Andreas, K. Jennifer and Christoph, F. H. 2012. Neglected tropical diseases: diagnosis, clinical management, treatment and control. *Current opinion.* **142**: 1-24.

Kindhauser, M.K., 2002. Communicable diseases: Global defence against the infectious disease threat. Geneva, Switzerland: World Health Organization.

Kock, R., S. Croft, M. Dixon, C. Fletcher, L. Good, J. Guzman, D. Heymann, R. Liyanage, D. Mckeever, R. McNerney, R. Peeling, M. Moran, D. Pfeiffer, J. Waage, and Wu, L. 2012. 'Prioritising the need for new diagnostics, medicine, vaccines and management practices of zoonoses which have significant impact in the developing world': DFID ZOONOSES REPORT 6. Pp. 1-126.

Leal F.E., C.L. Cavazzana, H.F. de Andrade, A.J. Galisteo, J.S. de Mendonc and Kallas E.G. 2007. Toxoplasma gondii pneumonia in immunocompetent subjects: case report and review. *Clin. Infect. Dis*. **44**: e62–e66

McGwire, B.S., and Satoskar, A.R., 2014. Leishmaniasis: clinical syndromes and treatment (Sir William Osler Medicine Master Class). *Q J Med*. **107**: 7-14.

Mengistu, G., and Ayele, B. 2007. Visceral Leishmaniasis and HIV co-infection in patients admitted to Gondar university hospital, northwest Ethiopia. *Ethiop. J. Health Dev*.**21(1),**53-60

Mondal, D., and Khan, M.G., 2011. Recent advances in post-kala-azar dermal leishmaniasis. *Curr Opin Infect Dis*. **24**: 418-422.

Nagill, R., and Kaur, S. 2011. Vaccine candidates for leishmaniasis: a review. *International J. immunopharmacology*. **11(10):** 1464-1488.

Negash, T., G. Tilahun and Medhin, G. 2008. Seroprevalence *Toxoplasma gondii* in Nazareth town, Ethiopia, *East African journal of public health*. **5:**123-126.

Negash, T., G. Tilahun, S. Patton, F. Prevot and Dorchies, P .H. (2004). Serological survey on toxoplasmosis in sheep and goats in Nazareth, Ethiopia, *Revue. Med. Vet*. **155(10**): 486-487.

Negera, E., E. Gadisa , L. Yamuah, H. Engers , J. Hussein, T. Kuru , A. Hailu, L. Gedamu and Aseffa, A. 2008. Outbreak of cutaneous leishmaniasis in Silti woreda, Ethiopia: risk factor assessment and causative agent identification. *Trans. R. Soc. Trop. Med. Hyg.* **102(9**): 883-90.

Ouattara, M., K.D. Silue, A.N. N'Guessan, A. Yapi, M. Barbara, G. Raso, J. Thompson, R.C.A., C.S. Palmer and O'Handley, R. 2008. The public health and clinical significance of Giardia and

Cryptosporidium in domestic animals. WHO Collaborating Centre for the Molecular Epidemiology of Parasitic Infections, Australia. *The Veterinary Journal*. **177:** 18-25.

Palatnik-de-Sousa, C. B., I. Silva-Antunes, A. D. A. Morgado, I. Menz, M. Palatnik and Lavor, C. 2009. Decrease of the incidence of human and canine visceral leishmaniasis after dog vaccination with Leishmune in Brazilian endemic areas. *Vaccine*. **27(27)**: 3505-12.

Ready, P.D., 2013. Biology of phlebotomine sand flies as vectors of disease agents. *Annu Rev Entomol*. **58**:227-250.

Ready, P.D., 2014. Epidemiology of visceral leishmaniasis. *Clinical Epidemio.* **6**: 147-154.

Reithinger, R., and Davies, C. R. 2002. Canine leishmaniasis: novel strategies for control. *Trends in parasitology*. **18(7):** 289-90.

Remington, J.S., R. McLeod, P. Thulliez and Desmonts G. 2006. Toxoplasmosis. In Infectious diseases of the fetus and newborn infant. Edited by Remington, J.S., J.O. Klein, C.B. Wilson and Baker, C.J. Philadelphia: Elsevier Saunders. pp. 947-1081.

Saunders, L.Z., 2000. Virchow's contributions to veterinary medicine celebrated then, forgotten now. *Vet. Pathol*. **37:** 199-2007.

Solaymani-Mohammadi, S., and Petri, J.W.A. 2006. Zoonotic implications of the swine-transmitted protozoal infections. *Vet. Parasitol.* **140**: 189-203.

Stanley, S.L., 2003. Amoebiasis. *Lancet.* **361**: 1025-1034.

Stephanie, M. F., D. Star, J. Harkness and Ellisa, J. 2012. Enteric Protozoa in the Developed World: a Public Health Perspective. *Clinical Microbiology Reviews.* **25(3):** 420-449.

Sundar, S., and Rai, M. 2002. Laboratory Diagnosis of Visceral Leishmaniasis. Laboratory Diagnosis of Visceral Leishmaniasis. *Clinical and vaccine immunology*. **9(5):** 951-958.

Tenter, A.M., A.R. Heckeroth and Weiss, L.M. 2000. *Toxoplasma gondii*: from animals to humans. *Int. J. Parasitol*. **30**: 1217-1258.

Teshale, S., A. Dumetre, M.L. Darde, B. Merga and Dorchies, P.H. 2007. Serological survey of caprine toxoplasmosis in Ethiopia. *Parasite.* **14**: 155-159.

Teshale, S., T. Mekashew, T. Endale, T. Belete and Ashenafi, T. 2009. Seroprevalence of latent *Toxoplasma gondii* infection among HIV infected and HIV uninfected people in Addis Ababa, Ethiopia. *BMC research notes*. **213:** 1186-1189.

Thompson, R.C.A., and Smith, A. 2011. Zoonotic enteric protozoa. *Veterinary Parasitology*. **182**: 70-78.

Thompson, S.C., 1994. Infectious diarrhoea in children: Controlling transmission in a childcare setting. *J. Paediatr. Child Health*. **30**: 210-219.

Tigabu, E., B. Petros and Endeshaw, T. 2010. Prevalence of Giardiasis and Cryptosporidiosis among children in relation to water sources in Selected Village of Pawi Special District in Benishangul-Gumuz Region, Northwestern Ethiopia. *Ethiop. J. Health Dev.* **24(3):** 205-213.

Tilaye, D., and Getachew, T.2002. Study on toxoplasmosis in sheep and goats in Debre Berhan and the surrounding areas in central Ethiopia. *Bulletin of Animal Health and Production in Africa*. **50**:138-147.

Torgerson P. R., and Macpherson C.N.L. 2011. The socioeconomic burden of parasitic zoonoses: Global trends. *Vet. Parasitol.* **182**: 79-95.

WHO, 1998. The World Health Organization Report, Geneva, Switzerland.

WHO. 2007. Global plan to combat neglected tropical diseases 2008–2015. Geneva, Switzerland: World Health Organization. http://whqlibdoc.who.int/hq/2007/WHO_CDS_NTD_2007.3_eng.pdf.

WHO. 2010. Control of the leishmaniasis: report of a meeting of the WHO Expert Committee on the Control of Leishmaniases, Geneva, March 22–26, 2010. *World Health Organ Tech Rep Ser*. **949**: 1-186.

Yibeltal, M., and Simenew, K. 2014. Toxoplasmosis: Emerging and reemerging zoonosis. *Afr. J. App. Microbiol. Res.* **3(1):** 1-11.