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The Public Health Significance of Leishmaniasis: An Overview

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Abstract
This seminar paper documents an overview on the current knowledge of Leishmania parasites of animals with emphasis on the public health significance and points to the wealth of information available for leishmaniasis, in contrast to the numerous gaps in our understanding of the public health significance and associated factors for the spread of the disease. Leishmaniasis is a parasitic zoonosis caused by protozoans of the genus Leishmania transmitted by insects known as phlebotomines, which are found in wild or urban environments. It affects domestic and wild animals and transmission to man happens by accident. The disease occurs in tropical and sub-tropical areas, mainly in Asia, Europe, Africa, and America There are three forms of leishmaniasis, visceral (VL), cutaneous (CL), and mucocutaneus (MCL). Of the three forms, VL is the most prevalent and severe in eastern Africa. In Ethiopia, the disease affects people living in a significant portion of the country. Leishmaniasis is difficult to control, causing epidemic outbreaks, thus being an important public health problem. Due to lesions caused by the mucocutaneous type and the severity of those caused by the visceral type in humans, leishmaniasis is one of the main public health concerns. Infection can cause symptoms that vary in severity and duration, depending on the health of the infected person and the particular strain of the parasite. The disease affects the rural poor community and usually outbreak occurs during harvesting seasons.
Keywords: Leishmania, leishmaniasis, Public health

INTRODUCTION

Leishmaniasis is a major vector-borne metazoonosis disease caused by obligate intramacrophage protozoa of the genus *Leishmania* (Azevedo e*t al*., 2012). The parasite is of greatmedical and veterinary public health significance. It infects numerous mammal species, including humans. Leishmaniasis is transmitted by the bite of phlebotominefemale sand flies of the genera *Phlebotomus* and *Lutzomyia*in the old and new worlds, respectively (Kakarsulemankhel *et al*., 2011). Leishmaniasis is a neglected tropical disease (NTD) caused by protozoan parasites of the Leishmania genus, and transmitted by sand fly bites from about 30 species that are proven vectors. There are three forms of leishmaniasis, visceral (VL), cutaneous (CL), and mucocutaneus (MCL). Of the three forms, VL is the most prevalent in eastern Africa, followed by CL and MCL (Desjeux, 2004).

Visceral leishmania (VL) is the most severe form of leishmaniasis, almost always fatal if untreated. Over 90% of the estimated annual incidence of 500,000 VL cases worldwide occurs in just six countries: - Bangladesh, India, Nepal, Sudan, Ethiopia and Brazil. Eastern Africa has the second highest number of VL cases, after the Indian Subcontinent, and the disease is endemic in parts of Eritrea, Ethiopia, Kenya, Somalia, North Sudan, Southern Sudan and Uganda (Desjeux, 2004 and Chappuis *et al.,* 2007).

The primary reservoir hosts of *Leishmania s*pecies are sylvatic mammals, such as; forest rodents, hyraxes, wild canids. Among domesticated animals; dogs are the most important species in the epidemiology of this disease. In addition to becoming ill, dogs are reservoir hosts for *L.infantum*, one of the two most important organisms in human visceral leishmaniasis (Pal, 2005). Currently, leishmaniasis has a wider geographical distribution pattern than before and it is considered to be a growing public health concern for several countries. The increase in leishmaniasis worldwide incidence is mainly attributed to the increase of several risk factors that are clearly man made and include massive migration, deforestation, urbanization, immune suppression, malnutrition and treatment failure (Desjeux, 2001).

Man made changes to the environment, as well as the population movements may lead to alterations in the range and density of the vectors and reservoirs and consequently may increase human exposure to infected sand flies (Zavitsanou *et al*., 2008). In Ethiopia, the disease affects people living in a significant portion of the country. Recurrent epidemics of visceral leishmaniasis (VL) have occurred in Metema and Humera. Following agricultural development in the region a large number of labor migrants from the highlands were moved to the endemic areas in the late 1970 for cropharvesting. This led to out breaks of VL, which resulted in high morbidity and mortality (Abyot *et al*., 2005).

A recent study investigating risk factors associated with the outbreak in Libo Kemkemidentified dog ownership and habitual outdoor-sleeping to be risk factors for infection (Bashaye *et al*., 2009). The cutaneous form was first described in Ethiopia in 1913 and is common in highland areas of altitude ranging from 1,700 to 2,700 meter above sea level. The majority of cutaneous Leishmaniasis (CL) cases in Ethiopia are caused by *L.aethiopica* (Gebre-Michael *et al*., 2004).

Leishmaniasis is one of the opportunistic infections that attack HIV-infected individuals. Recently more notice has been taken of Leishmania/HIV co-infection. The VL has a mortality rate as high as 100% if left untreated

and is spreading in several areas of the world due to increase number of AIDS victims (Pal, 2005). Leishmania and HIV co-infections have been reported in 35 out of 88 countries in which leishmaniasis are endemic, emerging disease and as many as 70 percent adults with VL also have HIV infection (TerHorst *et al*., 2008). Approximately 30%of VL patients are estimated to have HIV (TerHorst *et al*., 2008). Lack of a vaccine is one of the strongest drawbacks in controlling VL in endemic regions (Hide *et al*., 2007). As a result leishmaniasis is causing a great impact on the health of both human being and animals. Therefore the objectives of this paper are:-

- To highlight its public health significance and give an overview of the occurrence of leishmaniasis in Ethiopia.
- To compile the causes of the disease, the transmitting vector and reservoir hosts
- To indicate some risk factors for the spreading of the disease in humans.

LEISHMANIASIS

In 1903, Leishmania and Donovan separately described a protozoan parasite found in thesplenictissue of patients in India. Their simultaneous discovery of the protozoan now called *Leishmania donovani* first alerted the scientific community to the life threatening disease of leishmaniasis (Aynalem,2003).Now a century later, hundreds of millions are still at risk of infection and millions are afflicted by Leishmania (also known as "kala-azar", "black fever" or "black sickness"). It is an illness recognized for its complexity and diversity of symptoms and effects. It is endemic to most ecological regions of the world ranging from the rainforests ecosystems to desert land scapes, afflicting both rural and urban communities. About 21 different species of Leishmaniasis have been identified, and are categorized "underits primary syndromes; ***cutaneous***, ***mucocutaneos*** and ***visceral***, which result from parasite multiplication in macrophages in the skin, nasal and oral mucosa; and internal organs, respectively" (Aynalem, 2003).

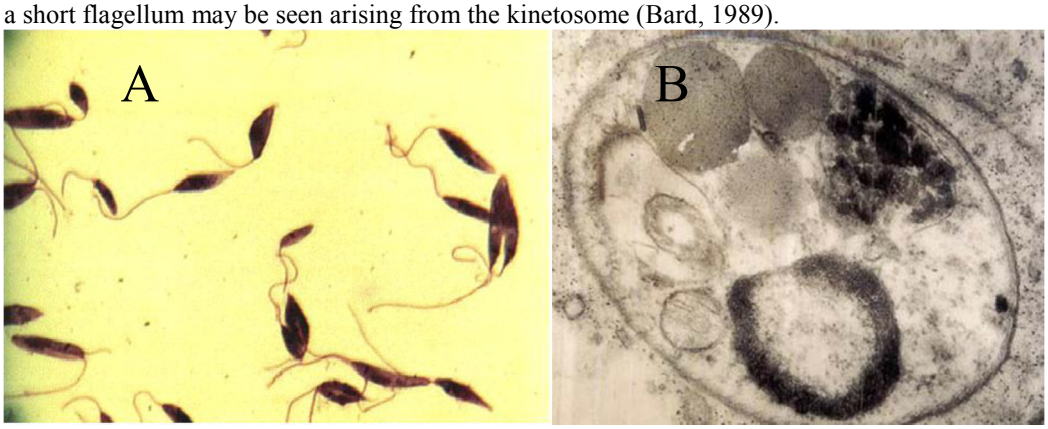
Leishmania

Species of the genus *Leishmania* (Kinetoplastida, Trypanosomatidae) are the causative organisms of leishmaniasis or leishmaniosis and these parasitic unicellular protozoans are usually transmitted between vertebrate hosts by the bite of blood sucking female phlebotomine (Diptera, Psychodidae) sand flies (WHO,2010). 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 develop in the guts of competent sand fly vectors, which inoculate them into mammalian skin. There; they are ingested by cells of the mononuclear phagocyte system (formerly the reticuloendothelial system), transform into rounded amastigote forms with the loss of the free flagellum, and then can multiply in the phagolysosomes of recruited macrophages to cause a primary lesion at the site of the sand fly bite. 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*). Any parasite causing cutaneous leishmaniasis can visceralize (*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 (Read ,2013).

The protozoan leishmania is an obligatory intracellular parasite which exists in two distinctive forms. In man and other hosts it occurs as a non-flagellaramastigote form, while in culture and guts of sand flies the flagellar or the promastigote form is seen. They are neither found in the peripheral blood nor in any visceral organ (Chang *et al.,* 1985).

The amastigote are small, round to oval, bodies which measure about 2-5µm and found only in the macrophages of infected vertebrate hosts. They are colorless, have a homogenous cytoplasm and are surrounded by a pellicle. The nucleus is centrally located, anterior to which is the kinetoplast. The kinetoplast is a section of the mitochondrion in which the mitochondrial DNA is arranged in regular arrays of fine fibrils (Chang *et al.,* 1985*).*The nucleus and a kinetoplast are easily visible in routine hematoxylin-eosin staining and rarely a short intra-cytoplasmic portion of the flagellum is also visible. The flagellar or the promastigote a form are seen in the culture media and in the gut of the sand fly, mosquitoes and bugs but it is only in sand fly that the parasite reaches the buccal cavity which becomes the insect vector of the parasite(Peters *et al.,*1985). They are motile, slender, organisms measuring 10-15 micrometer in length with a single anterior flagellum (Figure 1A). Rosette or clusters of promastigotes may also be seen. The electron microscopic picture (Figure 1B) shows that amastigotes have a double membrane supported by a layer of subpellicular fibrils (Stinson *et al.,* 1989).They run a spiral course from the region of the flagellar base towards the posterior apical end. One of the membranes is lost when the transformation of the amastigotes takes place, the fibrils are however retained. Amastigotes lack the flagellum but

a short flagellum may be seen arising from the kinetosome (Bard, 1989).



Figures 1: Promastigotes and Amastigote stages of Leishmania A) Flagellated *Leishmania* parasites; B) Electron microscopic picture of leishmania amastigote (Morsy, 1996)

Biology and Life cycle of Leishmaniosis

In nature, Leishmania are alternatively hosted by the insect (flagellated promastigotes) and by mammals (intracellular amastigotes). When a female sand fly takes blood meal from an infected mammal; the insect ingests intracellular amastigotes. Inside the fly amastigotes are transformed in to flagellated promastigotes in the midgut. The promastigotes migrate into the anterior portion of the mid gut. The bite of an infected sand fly deposits infective promastigotes in the mammals' skin, which are rapidly phagocytosed by the cells of mononuclear-phagocyte system. The intracellular rparasites change into amastigotes, which multiply by binary fission (Abyot *et al*., 2005). Figure two shows the life cycle of leshimania parasite.

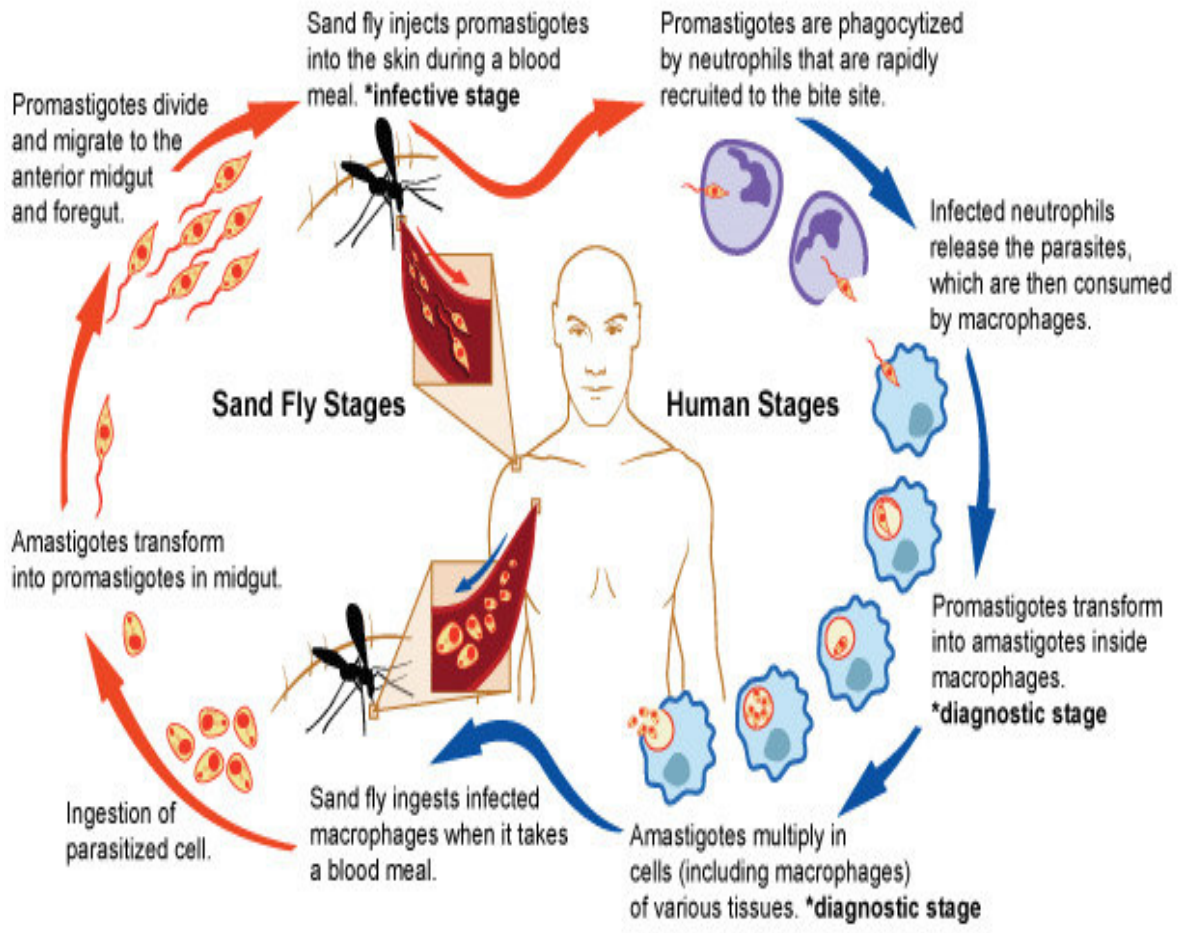


Figure 2: Biology and Life cycle of leishmaniasis in sandy flies and human (Morsy,1996).

Epidemiology

Human and animal leishmaniasis shows a wider geographic distribution than previously known leishmaniasis is widely distributed around the world. They range over inter tropical zones of America, Africa and extend in to temperate regions of South America, southern Europe and Asia. Their extension limits are latitude 45° north and 32° south. The burden of VL remains unknown since several cases are not diagnosed (Kolaczinski *et al*., 2008). It has been estimated that there are approximately half a million new cases of VL annually worldwide, with more than 50,000 associated deaths. More than 90% of VL cases occur in just six countries, namely India, Nepal, Bangladesh, Sudan, Ethiopia and Brazil (Chappuis *et al*., 2007**)**. However, it is also an important disease in several other East African countries, with an incidence rate of 30,000 cases per year and a mortality rate of 4,000 deaths per year (Musa *et al*., 2012**).**

During the last two decades, emergence of resistance to pentavalent antimonial had a huge impact on the epidemiology of leishmaniasis (Alvar *et al*., 2006). Epidemiology of new world CL is found in Mexico, Central America and South America-from Northern Argentina to Southern Texas and southern Europe. Many such patients develop unusual cutaneous manifestations (Arfan *et al*., 2006). Old world CL occurs in Asia, Middle East and Africa. Zoonotic cutaneous leishmaniasis (rural, wet type) is caused by *L.aethiopica* and *L.major* in most part of the Central Asia, Middle East and North Africa and transmission of infection is maintained in wild rodent/gerbil colonies. The estimated annual incidence is 1-1.5 million cases of CL in the old world over 90% of annual cases occur in Afghanistan, Algeria, Iran, Iraq, Saudi Arabia and Syria (Desjeux, 2004).

Several studies have definitively demonstrated that VL occurs in northwestern Ethiopia (Humera, Metema), Segen and Woito valleys in Gemu Gofa. Sporadic cases ofVL have been diagnosed from Wolkayit Tsegede (Gondar), Gibdo, Raya, and Kobo (Wello), Kijawa (Gambella) and Gelana (Sidamo) and Genale (Bale) river basins. Recently a devastating epidemic occurred in Humera with an estimated annual incidence of 1,500-2,000 cases. Due to high mortality, occurrence of epidemics, and high incidence of the disease in 15-45 age group leishmaniasis has become one of the leading health problems in Ethiopia. Cutaneous leishmaniasis (CL) occurs in highlands of Ethiopia. Transmission occurs in Cuttaber (Dessie), Aleku (Wellega), and Ochollo (GemuGofa). In Ochollo the overall prevalence of localized CL was 3.6-4.0%, with a peak value of 8.55 in the 0-10 year's old age group. Sporadic cases of CL have been diagnosed from many localities in the northern, central, and southern high lands of Ethiopia (Abyot *et al.*, 2005).

The vector Phlebotomi is mostly found in rural areas between 100 and 800mabove sea level and spends their life in a limited area, not exceeding1.5 km around their birth place. In some endemic areas of the Mediterranean the incidence of sero positivity is about 5–15% of the canine population (Siqueira-Neto *et al*, 2012). Of sero-positive dogs, 20–40% is asymptomatic carriers and may represent an unrecognized reservoir of the infection for other dogs and human beings (Siqueira-Neto *et al.*, 2012). The endemic area, and the total number of affected animals, has been increasing in recent years (Kakarsulemankhel, 2011**)**. This could reflect greater mobility of dog owners with their pets and/or a change in the climatic conditions that favor the sand flies' survival in new areas. *Leishmania chagasi* which causes the visceral disease in dogs and humans in the new world is inoculated by the bloodsucking sand fly *Lutzomyia* species and survival in new areas. Its distribution includes central and south America and small endemic areas in North America (Bravo *et al*., 1993).

Table 1: Parasitological information and its vectors

***Leishmania* species**	**Clinical form**	**Vector species**	**Reservoirs**
L. aethiopica	ZCL, DCL, ML	*P. longipes,* *P. pedifer*,* *P. sergenti*	*Procaviacapensis,* *Heterohyrax brucei*
L. major	ZCL	*P. dubosqui*	*Arvicanthisniloticus*
L. tropica	CL	*P. sergenti,* *P. saeveus*	
L. donovani	VL, PKDL	*P. orientalis,* *P. martini, H. celiae*	

Source:- (Alvar *et al*., 2008)

Transmissionof Leishmania

Leishmaniasis is a vector borne disease. It is mainly transmitted from the reservoir host to the healthy individual by the bite of female phlebotomus sand fly. The inoculation of promastigotes through the sand fly bite is the usual method of leishmaniasis transmission. In visceral leishmaniasis, a few cases of congenital and of blood transfusion transmission have been reported (Abyot *et al.,* 2005). Kala-azar is transmitted by members of subfamily of blood-feeding sand flies, *Phlebotomus martini*. When a fly bites a human being, it transmits the parasite in to his or her blood stream, where it multiplies. Infection can cause symptoms that vary in severity and duration, depending on the health of the infected person and the particular strain of the parasite. In East Africa, most cases of kala-azar are caused by a parasite called *Leishmania donovani*; in Ethiopia, this parasite is most often transmitted by a sand

fly called *Phlebotomus orientalis* (Berman, 2006*)*. Kala-azar is endemic in some regions in the north western lowlands and southern Ethiopia. In these regions it is transmitted by the sand fly from one human to another; usually in the period before Ethiopia's yearly major rainy season. In Ethiopia, in 2008, a national kala-azar task force was initiated, with the aim of eliminating kala-azar from the country by 2015 (Handman, 2001).

Pathogenesis of Leishmania

The bite of an infected sand fly results in the intradermal inoculation of promastigote stage of Leishmania. Within the dermis of mammalian skin, promastigotes escape complement activation and they are phagocytized by macrophages where they transform toamastigotes. Inside macrophages they have the capacity to resist intracellular digestion and they divide mitotically. When the intracellular development of the amastigotes remains localized at the inoculation site, various cytokines are released and cell reactions are generated, resulting in the development of localized lesion of CL. In other instances the parasite spread to the organs of the mononuclear phagocytic system, giving rise to VL. Amastigotes may also spread to other cutaneous sites as in diffuse cutaneous leishmaniasis (DCL) or to mucosal sites in the case of mucocutaneous leishmaniasis (MCL) (Abyot *et al*., 2005).

The localization of the parasite to the various organs of the patient results in the clinical expression of the disease. It is directly related to the tropism of the parasite species. In that sense, the genus leishmania can be divided broadly in to viscerotropic (*L.donovani*, *L.infantum*) and dermotropic (roughly all the other species). *L.braziliensis* and more rarely *L.panamensis* are known for their secondary mucosal spread. In spite of this general tropism of species some exceptions occur. Thus, well established viscera-tropic species can occasionally be responsible for limited cutaneous lesion and vice versa. The clinical expression of leishmaniasis depends not only on tropism of the parasite but also on immune status of the patient. Species responsible for localized cutaneous leishmaniasis can cause visceral or diffuse cutaneous leishmaniasis in immune compromised patients (Abyot *et al*., 2005).

Leishmania organisms are able to live in the endothelial reticulum of host cells because they neutralize the host cell's pH and detoxify oxygen metabolites. In macrophages the parasites multiply by binary fission until they rupture the cell and spread to other macrophages. It has recently been recognized that langerhans cells and other dendritic cells may also be infected by the parasite (Saint-André, 1997). These cells process and present parasitic antigens on their surface and are able to prime naive T helper (Th) cells and direct their response to the infection (Fondevila, 1997).

Locomotory problems are not very frequent and include shifting leg lameness, due to immune-mediated polyarthritis, polymiositis, and bone lesions, in which parasites are found in granulomatous inflammatory groups (Denerolle, 1996). There are also chronic ulcerative colitis with large bowel diarrhea and melena (Ferrer *et al.*, 1991); as well as acute fatal hemorrhagic enteritis (Denerolle, 1996). Histopathologically, two types of renal lesions have been described:- membranous glomerulonephritis and an extra-membranous glomerulonephritis, both a consequence of immune complex depositions. Proliferative lesions have rarely been seen (Diaz-Espineira *et al*., 1997).

PUBLIC HEALTH SIGNIFICANCE OF LEISHMANIASIS

People can carry some species of *Leishmania* asymptomatically for long periods, without becoming ill. In humans, the reported incubation period for cutaneous leishmaniasis can be as short as 1-2 weeks or as long as several months when it is caused by New World species, and up to three years when Old World species are involved. The incubation period for visceral leishmaniasis is 10 days to several years; most cases seem to become apparent in two to six months (Saint-André, 1997).

Two forms of leishmaniasis, cutaneous and visceral, are seen in humans. Some texts also distinguish a muco-cutaneous form, while others consider it to be a subset of cutaneous leishmaniasis. All types of leishmaniasis are growing health problems in the world, with endemic areas that are continually spreading. The form of the disease and the usual clinical signs vary with the species of leishmania. Some infections remain asymptomatic (Ferrer *et al.*, 1991).

Forms of Leishmaniasis in human

Cutaneous Leishmaniasis

Cutaneous leishmaniasis often involves only the skin, and may be characterized by one to dozens of lesions. Depending on the species of leishmania, ulcers, smooth nodules, flat plaques or hyperkeratotic wart-like lesions may be seen. The initial lesions, which occur on skin that was exposed to sand flies, are usually papules. Many lesions remain localized, but in some cases, the parasites may spread via the lymphatics and produce secondary lesions on the skin, or occasionally the mucosa, of other parts of the body. Regional lymphadenopathy sometimes occurs. Cutaneous leishmaniasis is usually painless unless the lesions become secondarily infected, and except in the ear, the ulcers tend to remain confined to the skin and do not affect the subcutaneous tissues. Most skin lesions heal spontaneously; however, the speed of healing varies with the species of leishmania. In some cases, it may

take several months to a year or longer. Some forms leave permanent scars. HIV-infected individuals can have unusually severe cases, and the disease is more difficult to cure. Steroid treatment or other forms of immune-suppression can also result in unusually severe disease. They may cause damage to deep tissues, and can persist indefinitely. The diffuse form can be incurable in some cases. Figure 3 shows lesions of cutaneous leishmaniasis on the face and back of a human.

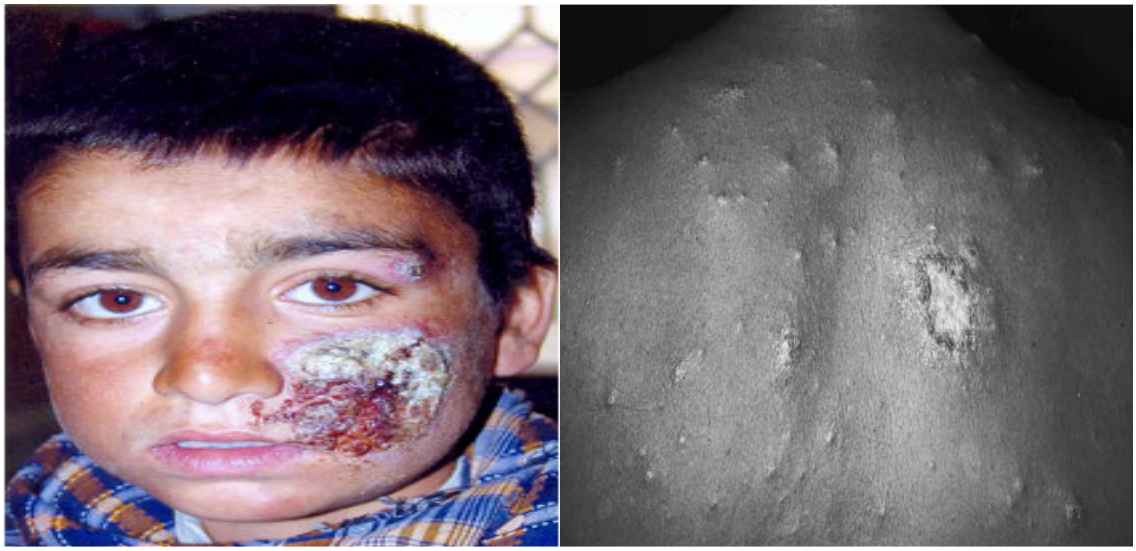


Figure-3: Lesion of cutaneous leishmaniasis (Couppie *et al*., 2004)

MucocutaneousLeishmaniasis (espundia)
Mucocutaneous leishmaniasis usually occurs in Latin America, where it is caused by *L.braziliensis braziliensis* and, less often, by *L.panamensis/L.guyanensis* (Arfan *et al*., 2006). Mucocutaneous leishmaniasis tends to occur 1 to 5 years after cutaneous leishmaniasis caused by these organisms has healed, but it can also be seen while skin lesions are still present. The initial signs are erythema and ulcerations at the nares, followed by destructive inflammation that can spread to involve the nasal septum, and in some cases, the pharynx or larynx. Frequent nosebleeds can be an early sign. The inflammation may perforate the nasal septum, cause severe disfigurement of the face, or block the pharynx or larynx. In some cases, the genitalia may also be involved. Mucocutaneous leishmaniasis does not heal spontaneously.

Visceral Leishmaniasis
Visceral leishmaniasis is usually an insidious, chronic disease among the inhabitants of endemic areas; however, the onset may be acute in travelers from *Leishmania*-free areas. In some cases (especially in Africa), a primary granuloma appears on the skin before the systemic signs. The most common symptoms of visceral leishmaniasis are a prolonged undulant fever, weight loss, decreased appetite, signs of anemia, and abdominal distension with splenomegaly and hepatomegaly. Thrombocytopenia may cause bleeding tendencies, including petechiae or hemorrhages on the mucous membranes, and leukopenia can result in increased susceptibility to other infections. Other symptoms may include coughing, chronic diarrhea, darkening of the skin, lymphadenopathy, and in many cases, signs of chronic kidney disease. Mild cases with only a few symptoms may resolve spontaneously (Kolaczinski *et al*., 2008**)**. Unless they are treated, most other cases are eventually fatal, often from secondary infections and other complications. Fulminant disease or atypical cases can also occur, especially in patients co-infected with HIV. People with successfully treated infections continue to carry the parasite, and the disease may recur if they become immune suppressed. Similarly, asymptomatically infected individuals may later develop clinical signs (Abyot *et al*., 2005).

Risk Factors for Humans
Socio-Economic Factors
Poverty increases the risk for leishmaniasis in many ways. Poor housing and per domestic sanitary conditions (e.g. lack of waste management, open sewerage) may increase sand fly breeding and resting sites, as well as their access to humans (Cortes *et al*., 2007)
Malnutrition
Poor protein, energy, iron, vitamin A and zinc nutritional status increases the risk that an infection will progress to clinically manifest visceral leishmaniasis. Recent experiments in protein, energy, zinc and iron deficient mice

suggest that this effect is mediated primarily through functional failure of the lymph node barrier and increased early visceralization of the parasite. Protein-energy malnutrition has also been associated with an increased risk for muco-cutaneous leishmaniasis (MCL), (Malaria Consortium, 2010).

Population Movements

Epidemics of both visceral and cutaneous leishmaniasis in both the old and the new world are often associated with migration and the introduction of non-immune people into areas with existing endemic or enzootic transmission cycles. Prediction of such outbreaks depends on the availability of ecological information and on evaluation of development areas before implementation of projects or population movements (Kakarsulemankhel, 2011). Seasonal labor movements may also spread the disease, with the return of migrants to non-endemic areas, as appears to have occurred in the highlands of Ethiopia in the 2000s. Individual who has behaviors such as sleeping outside under acacia trees and living in houses constructed of grassy material appears to increase risk for the disease (WHO, 2010).

Environmental Changes

In most endemic regions leishmaniasis is characterized by a patchy distribution with discrete transmission foci. This focal distribution of leishmaniasis transmission sites is due to micro ecological conditions that affect the vector, the parasite and there servoir host (Kakarsulemankhel, 2011). Environmental changes that can affect the incidence of leishmaniasis include urbanization, domestication of the transmission cycle and the incursion of agricultural farms and settlements into forested areas (WHO, 2010).

Climate Change

Leishmaniasisis is a climate-sensitive disease, occupying a characteristic climate space that is strongly affected by changes in rainfall, atmospheric temperature andhumidity (Malaria Consortium, 2010). Global warming and land degradation together are expected to affect the epidemiology of leishmaniasis by a number of mechanisms. First, changes in temperature, rainfall and humidity can have strong effects on the ecology of vectors and reservoir hosts by altering their distribution and influencing their survival and population sizes (Maltezou, 2008). Secondly, drought, famine and flood resulting from changes in climate conditions could lead to massive displacement and migration of people to areas with transmission of leishmaniasis and poor nutrition could compromise their immunity (WHO, 2010).

HIV co-Infection

The human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome-pandemic had also an impact on the epidemiology of VL (TerHorst *et al*., 2008). Due to deficient diagnostic capacities and surveillance, the burden of VL-HIV-co-infection in Africa remains grossly unknown; however HIV-co-infection is emerging in thiscontinent. In North West Ethiopia up to 30% of VL cases are HIV/co-infected (Maltezou, 2008).

IMPACT AND STATUS OF LEISHMANIASIS IN ETHIOPIA

In addition to public health significance; leishmaniasis imposes a negative impact on the economy of the people in particular and the country in general. These economic impacts are due to high cost of treatment, and time lost during hospitalization. The disease affects the rural poor community and usually outbreak occurs during harvesting seasons (Bashaye *et al*., 2009). Its prevalence is steadily rising in northern Ethiopia posing a public health challenge in the region (Custodio *et al*., 2012). The estimated annual burden of VL was to be between 4,500 and 5,000 cases. While there is currently no reliable estimate of the prevalence of CL, it has been estimated that the number of CL cases significantly exceeds that of VL (FMoH, 2006).

Several studies have definitively demonstrated that VL occurs in north western Ethiopia (Humera and Metema), Segen and Woito valleys in Gemu Gofa. Sporadic cases of VL have been diagnosed from WolkayitTsegede, Gibdo, Raya Kobo, Kijawa (Gambella) and Gelana (Sidamo) and Genale (Bale) river basins. Recentlya devastating epidemic occurred in Humera with an estimated annual incidence of 1,500-2,000 cases. Due to high mortality, occurrence of epidemics and high incidence of the disease in 15-45 age group leishmaniasis has become one of the leading health problems in Ethiopia (Malaria Consortium, 2010). In 2005, an outbreak of VL in Libo Kemkem woreda, a highland area of Amhara regional state, was identified. By 2007, around 2,450 primary cases and 120 deaths had been reported since the outbreak began in 2003 (Bashaye *et al*., 2009).

Table 2: Leishmania in skin test positivity in the Middle Awash (2 years report), Ethiopia

Locality	Site	Tested	No. positive	Positive in %
Melkasadi	4th camp	105	32	31
	Halaysumale	77	53	69
Melkawerer	Mahdol	66	37	56
	Hassoba	103	64	62
Amibara	Idolekore	118	47	40
	Old Gewane	68	16	23
	Medema	128	18	14
Gawena	Meteka	114	19	17
Total		**779**	**286**	**36.71**

Source: Oliva *et al*., 1995

CONCLUSION AND RECOMMENDATIONS

Leishmaniasisis caused by a protozoan parasite. The parasite is transmitted from one host to another throughthe bite of female sand fly. The leishmaniasisis zoonosis and the human infection is incidental. The disease has public health significance and both VL and CL are endemic. Taking into consideration the lack of a commercially available vaccine, the lack of access to efficient drug therapy mainly in the developing countries, the limited local resources of the affected countries, it is concluded that elimination of the disease is still a challenge for the international health community. Therefore the following points are recommended for future control of the disease:

- Priority should be given to the establishment of control programs and governments should take the lion share to empower and support concerned institutions to address control programs.
- The breeding and resting sites of the vector, control of hyraxes and rodents in the proximity of human dwellings should also be implemented.
- Policy should be formulated to control leishmaniasis in the direction of to eliminate stray and feral dogs.
- Extensive research in epidemiology of leishmaniasis should also be conducted in non-endemic areas too.

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