Journal of Biology, Agriculture and Healthcare
ISSN 2224-3208 (Paper) ISSN 2225-093X (Online)
Vol.7, No.23, 2017

Review on Leptospirosis in Animals and Its Public Health Importance

Jalel Negero
Jimma university college of Agriculture and veterinary medicine, school of veterinary medicine. P. O. box 307, Jimma, Oromia, Ethiopia

Summary
Leptospirosis is one of the major zoonotic bacterial infections that have a potential to affect all mammals including human and it has been documented as emerging zoonotic infectious disease. It has a worldwide distribution and caused by spiral shaped gram negative bacteria called Leptospira. The infection is primarily transmitted from animal to human through direct contact with urine of infected animal and ingestion of feed and water contaminated by Leptospira. Although found globally, leptospirosis is endemic in tropical and subtropical areas of the world particularly, where there is heavy rainfall and humidity. The clinical pictures of leptospirosis do not vary greatly with the species of animals. In human; individual working in agriculture and sewage, veterinarians and individuals in close contact with animals are at risk. Direct microscopic examination, serology and molecular techniques are commonly used for the diagnosis of the disease. Treatment is possible with specific serum therapy and by administration of antibiotics like tetracycline, chloramphenicol and penicillin. Applying of a good sanitary measures, vaccination, quarantine and rodent control are the most important control options of the leptospirosis.
Keywords: Leptospirosis, Zoonosis, Leptospira.

INTRODUCTION

Leptospirosis is identified as an emerging/re-emerging, worldwide, contagious, bacterial zoonotic disease that affects all mammals, including humans, livestock and wildlife (Bharti *et al.*, 2003). It is caused by different pathogenic species of the genus Leptospira which consist a various group of pathogenic and saprophytic spirochetes, currently classified into 17 genomospecies according to DNA– DNA hybridisation studies (Levett, 2001). In animals, it was recorded as the major causes of reproductive loss (Tilahun *et al*., 2013). Although distributed globally, the disease is particularly occurred in tropical and subtropical regions of the world, where environmental conditions are favorable for the survival and transmission of the organism (Pappas, 2008).

In the transmission of organism, rodents are the most important source of infection for humans and animals (Zavitsanou and Babatsikou, 2008). Once infection occurs, Leptospira species lives for a long time in the kidney tubules of an infected animal from where they are excreted through the urine (Sambasiva *et al*., 2003). Humans acquire the infection either through direct contact with the urine or other biological materials from the infected animals or indirect contact with water, soil and vegetation polluted with urine from animals harboring pathogenic leptospirosis (Adler and Moctezuma, 2010). In humans, Leptospirosis is known by different names like Weil's disease, Pretibial fever, Fort Bragg fever, Pea picker's fever, rice field fever, sugar cane cutter fever, swine herder's disease (Mohit and Umapathy, 2015).

In animals the disease is characterized by a broad range of clinical signs and acute or sub-acute stage of the disease is observed in the leptospiremic phase and characterized by septicemia and haemmorhagic syndrome while chronic infections are usually associated with reproductive losses (Petrakovsky *et al*., 2014). A serological assay and the Microscopic Agglutination Test (MAT) are considered as the gold standard for the diagnosis of leptospiral infection and its treatment is possible by using antibiotics (Heymann, 2004). Control measures of leptospirosis are aimed at limiting the occurrence of clinical disease based on integrated actions in several links of transmission chain (Lucheis and Ferreira, 2011) and it possible to prevent the disease through applying good sanitary measures (Tilahun *et al.,* 2013). Even if leptospirosis is identified as a mutual disease in tropical area with important zoonosis and mortality in both animals and humans it is not well reviewed and in general there is lack of well documented information about the disease. Hence, the objective of this review paper is to overview the information regarding leptospirosis in animals and its public health significance.

Etiology

Leptospirosis is caused by pathogenic spirochaetes of genus leptospira, occurring in nearly all the mammalian species (Dhanze *et al*., 2013). Genus leptospira is classified under Order Spirochaetales, Family Leptospiraceae, Class Spirochaetes and it is divided into two species: L. interrogans, embracing all pathogenic strains and L. biflexa, comprising the saprophytic strains isolated from the environment (Sharma and Yadav, 2008). L. interrogans contain over 212 serovars arranged into 23 serogroups. Common serovars of L. interrogans are serovar pomona, canicola, bratislava, graphityphosa, hardjo and interohemorrhagie (Radostits *et al*., 2007).

Morphology of Leptospira

Morphologically, leptospira is unique among other spirochetes bacteria, in that they have characteristic hooked ends and are tightly coiled Doern, (2000) (Figure 1). In tissue and within phagocytes, the organism has a spherical

or granular appearance. It has two periplasmic flagella, one attached sub terminally at each end that extend toward the cell's center without overlapping (Plank and Dean, 2000).

Epidemiology of Leptospirosis

Host range and distributions

Leptospirosis has the capacity to infect all mammalian species, but cattle, sheep, goats, dogs, horses and pigs are more likely affected and cats are rarely affected (Levett., 2001). It has a worldwide distribution but most common in developing countries and warm climate where contact with infected animals or water contaminated with their urine is likely to occur (Ko *et al*., 2009). Leptospira can survive in ponds, rivers, surface water, moist soil and mud when environmental temperature is warm. Serovars of L. interohaemorrhagiae, L. canicola, L. pomonai, L. hardijo and L. gripotyphosa occur in all continents and outbreaks have been reported followed by natural disaster like flooding and hurricane (WHO, 2003). Survival of the organism in the environment depends on variations in soil and water conditions in contaminated area. The organism is susceptible to drying and pH < 6 or > 8. Ambient temperatures < 7.1 °C or > 34 °C are detrimental for the survival of the organism (Radiostats *et al*., 2007).

Source of infection and Reservoir Hosts

A wide range of hosts especially, small mammalian species like rodents, insectivores and domestic animals (Acha and Azyfres, 2003) may serve as a source of human infection. Similarly, any infected animal can be a source of infection to others of own kind or to other species including human. Each leptospira to be associated with particular serovar species of natural maintenance host; however there are many exceptions to this rule as one may to be carried by different hosts and one animal may act as host for different serovars. In addition serovar may adapt to new host species which may become a natural reservoir for this infection (Radiostats *et al*., 2007).

Infection with leptospirae is maintained within a population of natural maintenance hosts by vertical and horizontal transmission, such population of natural animal host from the infection reservoir. The natural maintainace host ensures the continues circulation of a particular leptospiral serovar in a geographical area without the need for others, while incidental hosts to be involved maintenance hosts may carry a particular certain leptospira in their kidney and shed with their urine for long period of time and sometimes for animal life (Everard *et al*., 2001).

Transmission of the disease

Leptospirosis can be transmitted from one carrier animal to another healthy animal through direct or indirect contact with urine or other body fluids that contain viable Leptospira. Congenital transmission is also possible depending on the virulence of the organism and host. A viable infected neonate can harbour the infection for several weeks after birth and can act as a source of infection. In rats, pigs and dogs sexual transmission of has been also reported. Transmission by natural breeding or artificial insemination can occur but it is not common (Radiostats *et al*., 2007). Human acquire the infection directly through the handling of infected animal tissues and ingestion of contaminated food and water, sexual intercourse, trans-placental from infected mother to the foetus and through breast milk (WHO, 2003).

Risk factors of leptospirosis in animals

Animals of all age groups can be affected by leptospirosis, but young animals are more susceptible to the infection than adult animals. Certain management factors that pose risks of infection are introduction of infected animal into herd, common grazing with infected ones, access to contaminated water supplies such as streams, rivers, flood or drainage water and purchasing or loan of infected male animals for natural insemination (Radostits *et al*., 2007). Leptospirosis is termed as a seasonal disease in most cases in that it occurs commonly during summer season and fall in temperate region. In tropical climate the peak incidence occurs during the rainy season (Adler and Moctezuma, 2010).

Risk factors of leptospirosis in humans

Leptospirosis affects risk groups that are exposed to animal reservoirs or contaminated environments, such as abattoir and sewage workers, salver workers, coal mines, plumbers, farm workers, veterinarians, pet shop owners, meat handlers, military personnel, slaughter house workers and workers in fishing industry (Katz *et al*., 2002). Recreational activities that increase the risk of leptospirosis are gardening and water sports such as canoeing, swimming and white water rafting residents of some urban areas (Radostits *et al*., 2007). Compared to women, men are more frequently diagnosed with leptospirosis and this has been traditionally attributed to the over representation of men in high-risk occupations (Pavli and Maltezou, 2008).

Pathogenesis

After the bacterium enters the body, reaches blood streams through lymphatic vessels (Adler and Moctezuma, 2010). In the bloodstream, the bacteria will multiply and spread to some organs like kidneys, spleen, central nervous system, liver, eyes and reproductive organs. Once systemic circulation, three possible pathways are there. If the animal has a high and adequate antibody titer the body will be cleared from leptospires and no clinical signs can be seen. Animal with a moderate antibody can present with a mild or short leptospiremia followed by mild clinical signs. The leptospires are then eliminated through the kidneys and after the elimination the animal will not continue to shed leptospires. If the animal has a low or absent antibody titer there will be a multiplication of leptospires in the bloodstream which results in chronic shedding of leptospires in especially, in the urine for days

to months, even years (Greene *et al*., 2006).

Clinical signs of leptospirosis in animals

Leptospirosis is characterized by a broad range of clinical symptoms in livestock with minor difference between species affected: acute, subacute or chronic. Clinical signs of acute or sub-acute disease are observed in the leptospiremic phase and it is characterized by septicaemia, high fever, anorexia, petechiation of mucosa, depression and acute haemolyticanaemia with haemoglobinuria, jaundice and pallor of the mucosa (Petrakovsky *et al*., 2014).

Chronic infections are usually associated with reproductive losses through abortion, stillbirth, infertility and mastitis and milk drop syndrome. Abortion is common during the last trimester of pregnancy (Radostits *et al*., 2007). Although, other non-specific signs are common, anorexia, lethargy and vomiting were the three most common clinical signs in dogs with leptospirosis (Greenlee *et al*., 2004). In equine, severe forms of the disease is characterized by conjunctival suffusion, jaundice, anaemia, petechial haemorrhages on the mucosa and general depression. In foal's renal failure and in pregnant mares, placentitis, abortion and stillbirths may also present (Verma *et al*., 2013).

At necropsy, leptospirosis is characterized by the development of vasculitis, endothelial damage and inflammatory infiltrates composed of monocytic cells, plasma cells, histiocytes and neutrophils. On gross examination, petechial hemorrhages are common and organs are often discolored due to the degree of icterus (Levett, 2001). The histopathology is most marked in the liver, kidneys, heart, and lungs but other organs may also be affected according to the severity of the individual infection. In liver, intrahepatic cholestasis, hypertrophy and hyperplasia of Kupffer cells is evident while in the kidneys, interstitial nephritis is the major finding accompanied by an intense cellular infiltration composed of neutrophils and monocytes (Radostits *et al*., 2007).

Clinical signs of Leptospirosis in Human

Most human infections are asymptomatic and self-limited. Clinical leptospirosis typically manifests with a biphasic course, with an acute phase (anicteric form) followed by the immune phase characterized by antibody production and leptospiruria (Pavliand Maltezou, 2008). Only a minority of patients develop biphasic illness. Patients typically present with fever of abrupt onset, headache, conjuctival suffusion, photophobia, nausea and vomiting. Conjuctival suffusion and myalgias are considered to be the pathognomonic sign of leptospirosis. During the immune phase, fever may recurrent after 3 to 4 days of defervescence, accompanied by headache and myalgia and occasionally by cerebrospinal fluid pleocytosis. Aseptic meningitis develops up to 25% of leptospirosis cases (Bharti *et al*., 2003). Icteric leptospirosis (Weil's disease) develops in 5% to 10% of clinical leptospirosis cases (Pavli and Maltezou, 2008).

Diagnosis

Different laboratory tests available for the detection of leptospira are microscopic evaluation, culture, molecular method, serology and animal inoculation (Ahmad *et al*., 2005).

Microscopic Evaluation

During the first 10 days of the infection, leptospires may be seen on microscopic evaluation of blood, urine, CSF and peritoneal or pleural exudate (Levett, 2001). Dark field microscopy is required as the leptospires are very small (Zuerner, 2010), however more than 10000 organisms/ml are required to be able to see them. The method is insensitive and has a low specificity. Before choosing a body fluid considered about the pathogenesis and stage of the disease is important because, blood can only be used in the acute stage of the disease (Levett, 2001). Dark field microscopy must be followed by serology or cultural diagnostic methods if it is desirable to specify the serovar. The leptospires can be seen with light microscopy if using either Giemsa stain or silver impregnation on air-dried smears (Greene *et al*., 2006).

Culture

Even though the organism is isolated from body fluids, tissues like kidney, liver, lungs, brain from dead animals are giving a greater opportunity of a successful isolation. For abortion suspected cases, isolation could be attempted from non autolysed abortion materials or tissue samples from a freshly aborted fetus (Adler and Moctezuma, 2010). Isolation requires expensive and properly prepared and kept culture media. Inoculated media are incubated at 28-30 ^{0}C for several weeks or months. Cultures are incubated in dark and quite environment. Time of incubation depends on the serovar. Serovars such as pomona and grippotyphosa require the least time incubation up to 10 days. Regardless of time required for isolation, the inoculated culture media must be protected from contamination, thus require the addition of antimicrobial agents selected to inhibit growth of contaminants (ko *et al*., 2010).

Molecular method

DNA amplification using PCR and DNA primers have become an excellent diagnostic tool for detecting the presence of Leptospira in animal tissues and fluids and it can be applied to blood, urine, CSF and tissue samples ante or post mortem (Levett, 2001). Modern methods such as fragment length polymorphism (FLP), pulse field gel electrophoresis (PFGE) and other methods are currently being assessed (Adler and Moctezuma, 2010).

Serology

The microscopic agglutination test (MAT) is the standard serological test for the diagnosis of lepsopirosis

particularly for acute disease associated with host adapted serovars but it is less useful in the diagnosis of chronic infection (Radiostats *et al*., 2007). In animals which survive infection, leptospira can be readily diagnosed on the bases of demonstrating rising antibody titre in acute or convalescent sera. ELISA is more accurate than other tests with many advantages due to its high specificity and sensitivity, convenient technical feature including automation and can be used efficiently as serenity test for large number of serum samples (Hirsh *et al*., 2004).

Animal inoculation

A sensitive technique for the isolation of Leptospira consists of the intraperitoneal inoculation of young guinea pig with fresh plasma or urine, within few days spirochetes become demonstrated in the peritoneal cavity. On the death of the animal haemorrhagic lesions with spirochetes are found in many organs (Radiostats *et al*., 2007).

Public health Importance of Leptospirosis

Human leptospirosis is associated with some syndromes from asymptomatic to severe biphasic ilness. The first phase (acute, septicemic) is characterized by nonspecific signs like fever, chills, headache, conjunctival suffusion, photophobia, lymphadenopathy abdominal pain, nausea, vomiting, a sore throat, cough and jaundice may be also seen in more severe infection (Murray *et al*., 2002). The second phase (immune phase) is characterized by the development of anti leptospiral antibodies and excretion of leptospiral organisms with urine and also two forms of the disease are seen in this phase; Icteric and anicteric forms (Katz *et al.*, 2000). Most infections are anicteric form which is associated with aseptic meningitis.

The icteric form is more severe and it occurs in 5-10% of all patients, often rapidly progressive and associated with multi organs failure (kidney, liver and central nervous system) (Pavli and Maltezou, 2008). In this form some patients may have pulmonary symptoms with clinical signs ranging from cough, dyspnoea, chest pain and mild to severe haemoptysis. Severe gastrointestinal bleeding, adrenal or subarachnoid haemorrhage and pulmonary haemorrhages can occur. Death can occur from kidney failure, cardiac involvement, pulmonary haemorrhage or other hserious organ dysfunction. Anterior Uveitis occurs up to a year after re-covering in 2-10% of cases. Iridocyclitis and chorio retinitis can also be complications and may persist for years. Abortion, fetal death and rare congenital infections in new born have been reported (Radostits *et al*., 2007).

Prevalence of leptospirosis in Ethiopia

In Ethiopia, although climatic condition, socioeconomic and other factors are highly favorable for the occurrence and spread of the disease, few information's are available about leptospirosis in animals and humans. So far, Moch *et al*. (1975) reported the prevalence of 91.2%, 70.7%, 57.1%, 47.3%, 43.4%, 15.4% and 8.3% in horses, cattle, pigs, goats, sheep, camels and dogs respectively. In human, Eshetu *et al.* (2004) reported that from a total of 59 febrile patients attending the Wonji Hospital, 47.46% of them were found to positive for anti-antibodies of leptospira and the occurrence of the disease was more common in males compared to females. According to Tsegay *et al*. (2016) a total of 184 out of 418 horses had antibody titres of 1:100 or greater to at least one of 16 serovars, demonstrating the presence of 16 serovars of Leptospira species in Central and Southern Ethiopian horses. This means, 44% of the sampled horses were seropositive to at least one serovar.

Treatment

In animals, treatment is possible using dihydrostreptomycin after signs appeared to control the infection before severe damage to the liver and kidneys occurs (Levett, 2001) and control the leptospiruria of 'carrier' animals and render them safe to remain in the group (Radostits *et al*., 2007). Antibiotics like tetracycline, penicillin, ampicillin, doxycycline, streptomycin and the erythromycin were also used to treat the disease. Fluid therapy, blood transfusions and other supportive care may also be necessary (Heymann, 2004).

Prevention and Control

Understanding the epidemiology of leptospirosis is necessary to reduce the risk of the transmission (Levett, 2001). In domestic animals, leptospirosis can be controlled through vaccination, prophylactic treatment of exposed animals with antibiotics, quarantine newly introduced animals for at least 4 weeks, rodent control, regular serological testing, improved environmental hygiene, separating young animals from adults and safe artificial insemination (Dhanze *et al*., 2013). Whereas, in human it can be controlled through occupational hygiene, taking care of animal bite, vaccination, drinking clean water, early treatment, prophylactic therapy, awareness creation for people living in high risk areas (WHO, 2003).

CONCLUSION AND RECOMMENDATIONS

Leptospirosis is one of the most important bacterial zoonosis with the capacity to infect both animals and human worldwide. Infections with Leptospira are maintained within a population of natural maintenance hosts by vertical and horizontal transmission and rodents have been recognized as the most important and widely distributed reservoirs of leptospiral infection. Human leptospiral infections result primarily from direct or indirect exposure to urine of infected animals. Handling of infected animals and their tissues, ingestion of contaminated land water, sexual intercourse and trans-placental transmission are other modes of infection. Mortality in human with leptospirosis remains significant because of delay in diagnosis due to lack of diagnostic infrastructure. Applying of good sanitary measures, vaccination, quarantine and rodent control are the most important control measures of

the disease. At the end the following recommendations were forwarded; proper hygiene and prevention of contamination from urine and body fluids of infected animals should be practiced both at animal and herd level, awareness creation for risk groups and further studies to know about status and associated risk factors of leptospirosis in both animals and human in Ethiopia.

REFERENCES

Acha, P.N. and B. Azyfres, 2003. Zoonoses and communicable diseases common to man and animals. 3rd ed. USA, 1: 157-168.

Adler, B., and Moctezuma, A. P. (2010). Leptospira and Leptospirosis, journal of Veterinary Microbiology, 140: 287-296.

Ahmad, S.N., Shah, S., Ahmad, F.M. (2005).Laboratory diagnosis of leptospirosis.Journal of. Post graduate Medical, 51:195-200.

Bharti,A.R., Nally, J.E., Ricaldi, J.N., Matthias, M.A., Diaz, M. M., and Lovett, M.A. (2003). Leptospirosis, a zoonotic disease of global importance, journal of Lancet Infectious Disease, 3:757-771.

Bunnell, J.E., Hice, C.L., Watts, D.M., Montrueil, V., Tesh, R.B., Vinetz, J.M. (2000), Detection of pathogenic Leptospiraspecies,Infections among mammals captured in the Peruvian Amazon basin region, American Journal of Tropical Medicine Hygiene, 63(5-6): 255-8.

Dhanze, H., M.Kumar, Suman., and B.G.Mane., (2013). Epidemiology of leptospirosis, An Indian perspective, Journal of food borne and zoonotic diseases, 1(1) : 6-13.

Doern, G.V., (2000). Detection of selected fastidious bacteria, journal of Clinical Infectious Diseases, 30 : 166-173.

Eshetu, Y., Simone, K., Tsehaynesh, M., Dawit, W., Bethelehem, N., Neway, G., Belachew, D., Eduard, J, Sanders., (2004). Human leptospirosis in Ethiopia, a pilot study in Wonji, Ethiopian Journal of Health Developmet, 18 (1):48-51

Greene, J.E., Sykes, A.B., Cathy. and K, Hartmann., (2006). Infectious disease of the dog and cat, 3rd edn, Canada, Saunders, pp: 402-417.

Greenlee, J.J., Bolin, C.A., Alt, D.P., Cheville, N.F., and Andreasen, C.B., (2004). Clinical and pathologic comparison of acute leptospirosis in dogs caused by two strains of Leptospira kirschneri serovar grippotyphosa, American Journal of Veterinary Research,65 (8) : 1100-1107.

Heymann, D., (2004), Contol of Communicable Diseases, Manual, American Public Health Association, 18th ed, 306-309.

Katz, A.R., Ansdell, V.E., Effler, P.V., Middleton, C.R., Sasaki, D.M., (2002), Leptospirosis in Hawaii, 1974–1998, Epidemiologic analysis of 353 laboratory-confirmed cases, American Journal of Tropical Medicine Hygiene, 66 : 61.

Ko, A.I., Goarant, C., and Picardeau, M., (2009). Leptospira, the dawn of the molecular genetics era for an emerging zoonotic pathogen, journal of Nature Reviews Microbiology, 7 : 736-47.

Levett, P.N., (2001). Leptospirosis, Clinical Microbiology Reviews, journal of American society of microbiology, 14 (2) : 296-326.

Lucheis, S.B., Ferreira, J., (2011). Ovine leptospirosis in Brazil, The Journal of Venomous Animals and Toxins including Tropical Diseases, 17 (4) : 394-405.

Moch, R.W., Ebner, E.E., Barsoum, I.S., Botros, BAM., (1975). Leptospirosis in Ethiopia, A serological survey in domestic and wild animals, Journal of Tropical medicine and Hygiene, 78(2) : 38-42.

Mohit, Bhatia., B.L, Umapathy., (2015) ,Desiphering leptospirosis-a diagnostic mystery, an insight, International Journal of Medical Research and Health Sciences, 4(3):693-701.

Murray, P.R., K.S.K., Rosenthal, G.S. Obayashi., and M.A, Paller., (2002). Medical Microbiology, 4edn, USA, pp: 390-394.

Pappas, Georgios., Papadimitriou, G.P., Siozopoulou, V., Christou, L., and Akritidis N., (2008), The globalization of leptospirosis, worldwide incidence trends,International Journal of Infectious Diseases,12 (4) : 351-357.

Pavli, Androula., Helena, C. Maltezou, (2008). Travel-Acquired Leptospirosis, Journal of Travel Medicine, 15(6) : 447–453.

Petrakovsky, Jessica., Alejandra, Bianchi., Helen, Fisun., Patricia, Nájera-Aguilar., and Martha Maria, Pereira., (2014). Animal Leptospirosis in Latin America and the CaribbeanCountries, Reported Outbreaks and Literature Review (2002–2014), International journal of Environmental Research andPublic Health,11(10) : 10770-10789.

Plank R., and Dean.D., (2000). Overview of the epidemiology, microbiology and pathogenesis of Leptospiraspp. in humans, Journal of Microbes and Infection, 2(10) : 1265- 1276.

Radostits, O. M., Gay, C. C., Hinchcliff, K. W., and Constable, P.D., (2007).Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs and goats, London, Saunders, Elsevier Health Sciences, pp: 1094-1110.

Sambasiva RR, Naveen G, Bhalla P, Agarwal SK. Leptospirosis in India and the rest of the World. Braz J Infect Dis. 2003; 7(3): 178–193.

Sharma, M., & Yadav, A. (2008). Leptospirosis: epidemiology, diagnosis, and control, journal of Infectious Disease and Antimicrobial Agents, 25 (2) : 93-103.

Tilahun, Z., Reta, D., & Simenew, K. (2013). Global epidemiological overview of leptospirosis.International Journal of Microbiology Research, 4(1) : 09-15.

Tsegay, K., Potts, A. D., Aklilu, N., Lötter, C., &Gummow, B. (2016). Circulating serovars of Leptospira in cart horses of central and southern Ethiopia and associated risk factors. Journal of Preventive Veterinary Medicine, 125 : 106–115.

Verma, A., Stevenson, B., and Adler, B. (2013).Leptospirosis in horses. Journal of Veterinary microbiology, 167(1) :61-66.

World Health Organization. (2003). Human leptospirosis: guidance for diagnosis, surveillance and control. Pp.1-107.

Zavitsanou A, Babatsikou F. Leptospirosis: Epidemiology and Preventive measures. Health Sci J. 2008; 2: 75–82.

Zuerner, R. L, (2010). Genus Leptospira, Bergey's Manual of Systematic Bacteriology, 2nd ed, 4 : 232-242, Springer,New York.

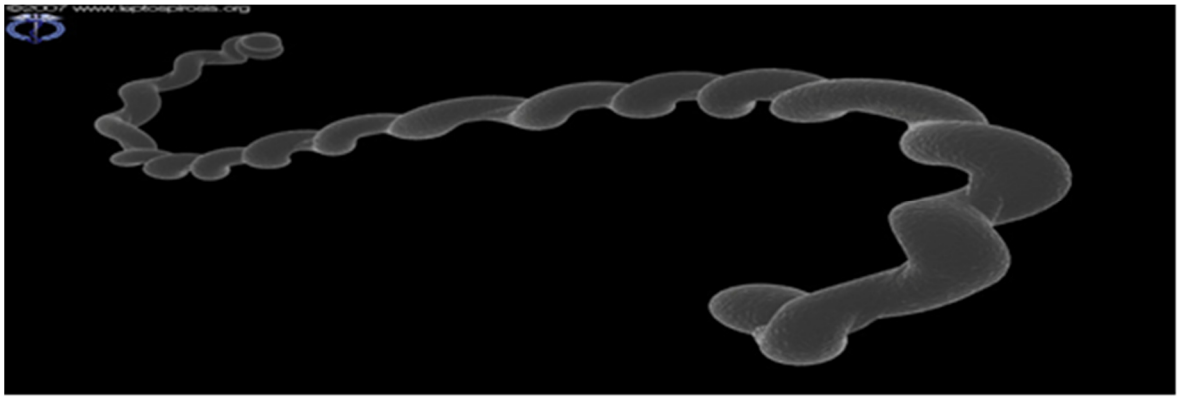

Figure 1. Corkscrew structure of leptospira,
Source: Dhanze *et al*., 2013