ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

LEPTOSPIROSIS IN RUMINANTS: AN UPDATED REVIEW ON EPIDEMIOLOGY, PATHOGENESIS, DIAGNOSIS AND ONE HEALTH CONTROL STRATEGIES

Rajratna Landge, Chandrakant D. Bhong, Vikas S. Waskar and Rahul P. Kolhe

Department of Veterinary Public Health and Epidemiology, KNP College of Veterinary Science, Shirwal, Dist. Satara, Maharashtra, India (Maharashtra Animal and Fishery Sciences University, Nagpur)

Corresponding Author: *Dr. Chandrakant D. Bhong*

Article DOI: https://doi.org/10.36713/epra32078
DOI No: 10.36713/epra32078

ABSTRACT

Leptospirosis is a globally distributed bacterial zoonosis caused by pathogenic spirochaetes of the genus Leptospira, and ruminants are among its most economically important hosts. In cattle, buffaloes, sheep and goats, the disease ranges from acute haemolytic illness in young stock to a chronic, largely subclinical reproductive syndrome marked by abortion, stillbirth, weak neonates, infertility and milk drop. Host-adapted serovars, particularly L. borgpetersenii and L. interrogans serovar Hardjo in cattle, persist in the kidneys and genital tract and are shed in urine and uterine discharges for months, which sustains herd infection and creates continuous occupational exposure for farmers, milkers, veterinarians and abattoir workers. Incidental serovars such as Pomona, Icterohaemorrhagiae, Grippotyphosa and Canicola, acquired from rodents, pigs, dogs and wildlife, cause more severe acute disease. Tropical climate, monsoon flooding, waterlogged paddy fields, communal grazing, mixed species husbandry and rodent infestation make leptospirosis endemic in India, where serosurveys repeatedly record substantial seropositivity in bovines and small ruminants with reproductive disorders. The microscopic agglutination test remains the reference serological test, while real-time PCR targeting lipL32, isothermal assays such as LAMP, and culture provide direct evidence of infection and carrier status. Control rests on detection and treatment of carriers with streptomycin or long-acting oxytetracycline, vaccination with locally relevant serovars, rodent and water management, biosecurity and worker protection. Because the same environmental and animal reservoirs drive human infection, leptospirosis in ruminants is best addressed through an integrated One Health framework linking veterinary, medical and environmental surveillance. This review summarises current knowledge on the aetiology, epidemiology, pathogenesis, clinical and pathological features, diagnosis, treatment, prevention and public health significance of leptospirosis in ruminants, with emphasis on the Indian context, and identifies priority research gaps.

KEYWORDS: *Leptospirosis, Leptospira, Ruminants, Cattle, Small ruminants, Serovar Hardjo, Reproductive failure, Microscopic agglutination test, Zoonosis, One Health*

1. INTRODUCTION

Leptospirosis is considered the most widespread zoonosis in the world and a re-emerging infection of humans and animals in tropical and subtropical regions (Bharti et al., 2003; Levett, 2001). Human leptospirosis is estimated to cause about 1.03 million cases and 58,900 deaths each year (Costa et al., 2015), with a burden of roughly 2.9 million disability-adjusted life years (Torgerson et al., 2015). Almost every mammal can harbour pathogenic leptospires, but livestock, rodents and dogs are the reservoirs that matter most for human exposure in agrarian societies (Adler & de la Peña Moctezuma, 2010; Cilia et al., 2020).

In ruminants, leptospirosis is primarily a disease of production. Acute infection causes fever, haemolytic anaemia, haemoglobinuria, jaundice and death, especially in calves and lambs. The larger losses, however, arise from chronic infection of the kidney and reproductive tract, which leads to abortion, stillbirth, weak offspring, repeat breeding, prolonged calving intervals and agalactia (Ellis, 1994; Grooms, 2006). Because chronically infected animals often appear healthy, the disease is under-diagnosed and its economic impact is consistently underestimated (Lilenbaum & Martins, 2014).

India has the largest bovine population in the world and a large population of sheep and goats, most of which are kept by smallholders under conditions that favour leptospiral transmission. Heavy monsoon rainfall, flooding, paddy cultivation, shared water sources, close contact between livestock, dogs and rodents, and limited diagnostic access make the disease endemic in states such as Maharashtra, Gujarat, Kerala, Tamil Nadu, Karnataka and the Andaman and Nicobar Islands (Balamurugan et al., 2018; Karpagam & Ganesh, 2020). Ruminants infected with leptospires also contaminate the environment shared with people, so animal leptospirosis is directly linked to the human disease burden.

This review consolidates current knowledge on leptospirosis in cattle, buffaloes, sheep and goats. It covers

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

aetiology, epidemiology, pathogenesis, clinical and pathological features, diagnosis, treatment, prevention and control, and the public health dimension, and it outlines research priorities relevant to India and other tropical livestock systems.

2. AETIOLOGY AND TAXONOMY

Leptospires are thin, helical, highly motile spirochaetes of the family Leptospiraceae, order Spirochaetales. Cells measure about 6–20 µm in length and 0.1 µm in diameter, with characteristic hooked ends and two periplasmic flagella that give corkscrew motility through viscous media and tissues (Levett, 2001). They are obligate aerobes that grow slowly at 28–30 °C in Ellinghausen–McCullough–Johnson–Harris (EMJH) medium, using long-chain fatty acids as their carbon and energy source. Being too thin to be seen by ordinary light microscopy, they are visualised by dark-field microscopy, immunofluorescence or silver impregnation.

Two classification systems are in use. The **serological system** groups leptospires into more than 300 serovars arranged in about 24 to 30 serogroups on the basis of lipopolysaccharide (LPS) antigens; it remains the basis of the microscopic agglutination test (MAT) and of vaccine formulation. The **genomic system** is based on DNA relatedness and whole-genome analysis. Vincent et al. (2019) described 64 *Leptospira* species divided into a pathogenic clade (P), with subclades P1 (highly virulent) and P2 (intermediate), and a saprophytic clade (S). Serovar and species do not correspond one-to-one; for example, serovar Hardjo exists as *L. interrogans* serovar Hardjo (type Hardjoprajitno) and *L. borgpetersenii* serovar Hardjo (type Hardjobovis), which differ in virulence and geographical distribution (Ellis, 2015).

Pathogenic leptospires survive for weeks to months in moist soil, mud and surface water at neutral to slightly alkaline pH and moderate temperature. They are rapidly killed by desiccation, acidic pH (below about 6), temperatures above 50 °C, and common disinfectants. The outer membrane lipoprotein LipL32 is abundant and conserved in pathogenic species, which makes it a key target for molecular diagnosis and recombinant antigen development (Picardeau, 2017; Haake & Levett, 2015).

Table 1. Principal *Leptospira* serovars of importance in ruminants

Serovar (serogroup)	Common species	Main maintenance host	Status in ruminants	Main clinical consequence
Hardjo, type Hardjobovis (Sejroe)	*L. borgpetersenii*	Cattle	Host-adapted	Chronic renal and genital carriage, abortion, infertility, milk drop
Hardjo, type Hardjoprajitno (Sejroe)	*L. interrogans*	Cattle	Host-adapted	As above, generally more virulent
Pomona (Pomona)	*L. interrogans*	Pigs, wildlife	Incidental	Acute haemolytic disease in calves and lambs, late abortion
Icterohaemorrhagiae / Copenhageni (Icterohaemorrhagiae)	*L. interrogans*	Rats	Incidental	Severe acute disease with jaundice and haemoglobinuria
Grippotyphosa (Grippotyphosa)	*L. kirschneri*	Rodents, wildlife	Incidental	Acute disease, abortion
Canicola (Canicola)	*L. interrogans*	Dogs	Incidental	Acute disease, abortion
Australis, Autumnalis, Hebdomadis (respective serogroups)	*L. interrogans*	Rodents, wildlife	Incidental	Frequently detected in Indian serosurveys; reproductive disorders

Sources: Ellis (2015); Adler & de la Peña Moctezuma (2010); Balamurugan et al. (2018).

3. EPIDEMIOLOGY

3.1 Maintenance and incidental hosts

The epidemiology of leptospirosis is shaped by the relationship between a serovar and its host. In a **maintenance host**, the serovar is transmitted efficiently within the species, causes relatively mild disease, persists in the kidney and genital tract, and is shed for long periods while serological responses are often low. In an **incidental (accidental) host**, transmission between animals of that species is inefficient, but clinical disease is more severe and kidney carriage is shorter (Ellis, 2015). Cattle are the maintenance host for serovar Hardjo, and sheep can also maintain Hardjo, which matters where the two species graze together (Martins & Lilenbaum, 2014). Infections of ruminants with Pomona, Icterohaemorrhagiae, Grippotyphosa, Canicola and Australis are incidental and originate from pigs, rodents, dogs and wildlife (Adler & de la Peña Moctezuma, 2010).

3.2 Global and Indian distribution

Bovine leptospirosis has been reported from every continent except Antarctica, and Hardjo is the most common serovar in cattle worldwide (Ellis, 2015). In tropical countries, a broader range of serogroups is involved and herd-level exposure is often very high (Lilenbaum & Martins, 2014; Zarantonelli et al., 2018). In India, serological surveys of cattle and buffaloes, especially animals with abortion, repeat breeding and other reproductive disorders, consistently detect antibodies against serogroups Sejroe (Hardjo), Australis, Pomona, Icterohaemorrhagiae, Hebdomadis, Grippotyphosa and Autumnalis. Seroprevalence varies widely between studies with

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

region, species, sampling frame and MAT cut-off, but leptospirosis is recognised as an important, under-diagnosed cause of reproductive loss in endemic states (Balamurugan et al., 2018). Sheep and goats in India are also seropositive, and small ruminants under extensive grazing are exposed to the same contaminated water and pasture as bovines (Martins & Lilenbaum, 2014).

3.3 Sources of infection and routes of transmission

The main source of infection is urine of carrier animals, which may contain very large numbers of leptospires. A meta-analysis found that the leptospiral load in cattle urine can be comparable to that of rats, indicating that livestock are important sources of environmental contamination in rural communities (Barragan et al., 2017). Other sources include placental fluids, aborted foetuses, uterine discharges, semen and, less importantly, milk.

- **Direct transmission:** contact with infected urine, placental material or aborted foetuses; venereal transmission through natural service or contaminated semen; transplacental infection of the foetus; and neonatal infection.
- **Indirect transmission:** entry of leptospires through mucous membranes of the eye, nose and mouth, or through softened or abraded skin, during contact with contaminated water, mud, pasture or bedding.

Within cattle herds, Hardjo spreads mainly by direct urinary and venereal routes, so a few chronic shedders can maintain infection indefinitely (Loureiro & Lilenbaum, 2020).

3.4 Risk factors

Animal-level risk factors include age (exposure accumulates with age, while severe acute disease is more common in the young), pregnancy and breeding status. Herd and environmental risk factors include:

- High rainfall, monsoon flooding and waterlogged fields, which favour leptospiral survival and dispersal (Lau et al., 2010; Mwachui et al., 2015)
- Access to ponds, streams, canals and stagnant water; communal grazing and watering
- Co-grazing of cattle with sheep, and contact with pigs, dogs, rodents and wildlife
- Natural service with untested bulls, introduction of purchased or replacement animals without quarantine or testing
- Poor drainage, inadequate disposal of placentas and aborted foetuses, and rodent infestation of feed stores and sheds
- Absence of vaccination and of routine reproductive health monitoring

In Indian smallholder systems, many of these risks occur together. Paddy-livestock farming, sugarcane cultivation with irrigation, shared village water sources and free-roaming dogs and rodents create continuous opportunities for exposure across species, including humans.

4. PATHOGENESIS

Leptospires penetrate mucous membranes or damaged skin and, aided by their motility and outer membrane proteins, reach the bloodstream within hours. A **leptospiraemic phase** follows, lasting about 4 to 7 days, during which organisms multiply in blood and spread to the liver, kidneys, lungs, spleen, brain, eyes and, in pregnant animals, the uterus and placenta (Adler & de la Peña Moctezuma, 2010). Adhesion to host cells and extracellular matrix, resistance to complement, and acquisition of iron are important virulence attributes (Haake & Levett, 2015; Ko et al., 2009).

Tissue damage arises mainly from injury to the endothelium of small blood vessels, which produces vasculitis, capillary leakage, haemorrhage and ischaemia. In incidental infections such as those caused by Pomona or Icterohaemorrhagiae, haemolysins and phospholipases contribute to intravascular haemolysis, causing anaemia, haemoglobinuria and jaundice, particularly in calves and lambs. Hepatocellular damage and cholestasis add to the icterus, and acute interstitial nephritis can lead to renal failure.

Specific agglutinating and opsonising antibodies appear from around the end of the first week and clear leptospires from the blood and most organs. The organisms persist, however, in sites with limited antibody access: the lumen of the proximal renal tubules, the pregnant uterus and the genital tract of both sexes, and occasionally the eye. Colonisation of the renal tubules results in prolonged urinary shedding, which in Hardjo-infected cattle can continue for many months (Ellis, 2015). Persistence in the uterus and oviduct of non-pregnant cows and in the seminal vesicles of bulls underlies the genital form of the disease, now described as bovine genital leptospirosis (Loureiro & Lilenbaum, 2020).

In pregnant animals, leptospires cross the placenta and infect the foetus. Foetal death results from direct foetal infection, placentitis, and, in acute maternal infection, maternal fever and haemolysis. Depending on the stage of gestation and the serovar, the outcome may be early embryonic death, abortion (typically in the last trimester), stillbirth, or the birth of weak, infected neonates (Ellis, 1994; Grooms, 2006). Reduced milk yield in acute Hardjo infection is attributed to direct involvement of the mammary gland during leptospiraemia.

5. CLINICAL MANIFESTATIONS

5.1 Cattle

Acute form. Seen mainly in calves and in adults infected with incidental serovars. Signs include high fever (40.5–41.5 °C), anorexia, depression, haemolytic anaemia, haemoglobinuria ("red water"), icterus, petechial haemorrhages on mucosae and, occasionally, meningitis. Mortality in young calves can be high.

Milk drop syndrome. Acute Hardjo infection in lactating cows causes a sudden fall in milk yield, often affecting many cows in a herd over a few weeks. The udder is flaccid, and all four quarters produce thick, yellowish, colostrum-like milk, sometimes blood-tinged, with a high somatic cell count but without obvious udder inflammation. Yield usually recovers within two to three weeks.

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

Reproductive (chronic) form. This is the most economically important form. It includes abortion, usually in the last trimester and several weeks after the initial infection, stillbirth, premature birth and weak calves with high neonatal mortality. Chronic genital infection is associated with repeat breeding, embryonic loss, extended calving intervals and reduced conception rates, often in herds with no history of acute illness (Grooms, 2006; Loureiro & Lilenbaum, 2020).

Subclinical carrier state. Many infected cattle show no clinical signs but shed leptospires in urine and genital secretions, acting as a reservoir for other animals and for people.

5.2 Buffaloes

Buffaloes are highly susceptible and are frequently exposed because of wallowing behaviour in ponds and canals. Clinical features are broadly similar to those in cattle, including fever, haemoglobinuria, jaundice, abortion and reduced milk yield. Their prolonged contact with surface water makes buffaloes a significant, and often overlooked, source of environmental contamination in mixed bovine herds.

5.3 Sheep and goats

Small ruminants were traditionally regarded as relatively resistant, but serological and molecular studies show that infection is common in tropical regions (Martins & Lilenbaum, 2014). Acute disease, typically with Pomona, Icterohaemorrhagiae or Grippotyphosa, presents as fever, anorexia, haemoglobinuria, jaundice, anaemia and sudden death, particularly in lambs and kids. Goats appear more prone to acute disease than sheep. Reproductive signs include abortion, stillbirth, weak neonates and infertility. Sheep may act as maintenance hosts for Hardjo and can shed the organism in urine for long periods.

Table 2. Differential diagnosis of leptospirosis in ruminants

Clinical presentation	Conditions to differentiate
Haemoglobinuria, anaemia, jaundice	Babesiosis, anaplasmosis, theileriosis, bacillary haemoglobinuria, post-parturient haemoglobinuria, chronic copper poisoning, plant toxicities
Abortion and stillbirth	Brucellosis, bovine viral diarrhoea, infectious bovine rhinotracheitis, neosporosis, trichomoniasis, campylobacteriosis, listeriosis, Q fever, mycotic abortion
Sudden milk drop	Mastitis (especially environmental or coliform), ketosis, other acute febrile diseases
Repeat breeding, embryonic loss	Nutritional deficiencies, endocrine disorders, campylobacteriosis, trichomoniasis, uterine infections

6. PATHOLOGY

6.1 Gross lesions

In **acute** cases, carcasses show generalised icterus, pale mucous membranes, thin watery blood and petechial or ecchymotic haemorrhages on serosal surfaces. The liver is enlarged, friable and yellow-brown. Kidneys are swollen, dark and may show petechiae on the capsule and cortex. The urinary bladder often contains dark red to brown urine because of haemoglobinuria. Pulmonary oedema and haemorrhage may be present.

In **chronic** cases, kidneys show multiple small, raised or depressed greyish-white foci, 1 to 5 mm in diameter, scattered through the cortex ("white-spotted kidney"), which correspond to foci of interstitial nephritis and may progress to fibrosis.

Aborted foetuses are usually autolysed, which limits diagnosis. When fresh, they may show icterus, subcutaneous oedema, serosanguinous fluid in body cavities, and pale foci in the kidneys. The placenta may be oedematous with avascular, yellowish-brown cotyledons.

6.2 Histopathological lesions

The characteristic renal lesion is **interstitial nephritis**, with infiltration of lymphocytes, plasma cells and macrophages around tubules and glomeruli, tubular degeneration and necrosis, and, in chronic cases, interstitial fibrosis. Leptospires can be demonstrated in the lumen of the proximal convoluted tubules by silver staining (Warthin–Starry or Levaditi) or, more specifically, by immunohistochemistry. The liver shows dissociation of hepatic cords, centrilobular necrosis, Kupffer cell hyperplasia, erythrophagocytosis and bile stasis. Foetal tissues may show non-suppurative interstitial nephritis, hepatitis and placentitis. Immunohistochemistry and PCR on foetal kidney, liver and lung are useful in confirming leptospiral abortion (Ellis, 2015).

7. DIAGNOSIS

Diagnosis of leptospirosis in ruminants should combine history, clinical signs, herd reproductive records, pathology and laboratory tests, because no single test detects all stages of infection. Laboratory methods fall into two groups: tests that detect the organism or its DNA, and tests that detect antibodies (WOAH, 2021; Musso & La Scola, 2013).

7.1 Sample selection

During the acute leptospiraemic phase (first week), blood (EDTA or heparin) and milk are the samples of choice for organism or DNA detection, together with serum for baseline serology. After the first week and in chronic carriers, urine (collected mid-stream, preferably after diuretic administration) is the most useful sample. In abortion cases, foetal kidney, liver, lung, thoracic and abdominal fluid, stomach contents and placenta should be submitted, together with maternal serum. Genital samples such as vaginal fluid, uterine flushings and semen are useful for genital

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

carriers (Loureiro & Lilenbaum, 2020). Samples for culture should be inoculated within hours or kept chilled; those for PCR can be frozen.

7.2 Direct detection

Microscopy. Dark-field microscopy of urine or body fluids is rapid but has poor sensitivity and specificity because artefacts resemble leptospires. Immunofluorescence and immunohistochemistry on tissues are more reliable and remain useful on partially autolysed foetuses.

Culture. Isolation in EMJH medium (with 5-fluorouracil or other selective agents for contaminated samples) is the only way to obtain the serovar for typing and vaccine development. It is slow, with cultures incubated at 28–30 °C and examined regularly for up to 12 to 26 weeks, and its sensitivity is low, particularly for fastidious Hardjo strains (WOAH, 2021; Zarantonelli et al., 2018).

Molecular methods. Conventional and real-time PCR have become the most practical tools for direct detection in blood, urine, milk, semen and tissues. Commonly used targets include *lipL32*, *secY*, *lfb1* and 16S rRNA genes. A TaqMan real-time PCR targeting *lipL32* detects pathogenic leptospires specifically and sensitively (Stoddard et al., 2009), and other validated real-time assays are available (Ahmed et al., 2009). Urinary PCR is especially valuable for identifying carrier animals that are seronegative or have low MAT titres (Hamond et al., 2014). Loop-mediated isothermal amplification (LAMP) offers a simpler, low-cost, field-adaptable alternative that requires only a heating block and visual read-out (Sonthayanon et al., 2011). Sequencing and multilocus sequence typing of PCR products allow species and genotype identification without culture.

7.3 Serological tests

Microscopic agglutination test (MAT). MAT is the reference serological test. Serial dilutions of serum are reacted with a panel of live leptospiral cultures representing serovars present in the region, and agglutination is read under dark-field microscopy (Goris & Hartskeerl, 2014). A titre of 1:100 or more is generally taken as evidence of exposure, while a four-fold or greater rise between acute and convalescent sera taken two to four weeks apart confirms recent infection (WOAH, 2021). MAT is valuable at herd level but has important limitations for individual animals. Titres may be low or absent in chronic Hardjo carriers and in animals that abort weeks after infection, vaccination titres interfere with interpretation, cross-reactions occur between serogroups, and the test requires maintenance of live cultures. The panel should include locally prevalent serogroups, as indicated by Indian serosurveys.

Enzyme-linked immunosorbent assay (ELISA). IgM and IgG ELISAs using whole-cell antigens or recombinant proteins such as LipL32 and LigA/LigB are suitable for screening large numbers of samples, including bulk-tank milk for herd surveillance. They do not identify the infecting serovar and may need confirmation by MAT.

Table 3. Comparison of diagnostic methods for leptospirosis in ruminants

Method	Target	Best sample and stage	Main advantages	Main limitations
Dark-field microscopy	Organism	Urine, body fluids	Rapid, inexpensive	Low sensitivity and specificity; artefacts
Culture (EMJH)	Live organism	Blood (acute), urine, kidney, foetal tissue	Definitive; allows serovar typing	Very slow (weeks to months); low sensitivity; contamination
FAT / immunohistochemistry	Antigen in tissue	Kidney, liver, foetal tissue	Works on autolysed tissue; shows lesion association	Needs specific conjugates; moderate sensitivity
Conventional / real-time PCR	DNA (*lipL32*, 16S rRNA, *secY*)	Blood (acute), urine, milk, semen, foetal tissue	Rapid, sensitive, detects carriers	Does not give serovar; cost and laboratory requirements
LAMP	DNA	As for PCR	Simple, low-cost, field-adaptable, visual read-out	Risk of carry-over contamination; fewer validated veterinary assays
MAT	Agglutinating antibodies	Serum (paired samples); herd level	Reference test; indicates serogroup	Labour-intensive; live cultures; low titres in carriers; vaccine interference
ELISA (IgM/IgG)	Antibodies	Serum, bulk milk	High throughput; suitable for surveillance	No serovar information; variable specificity

Sources: WOAH (2021); Musso & La Scola (2013); Sykes et al. (2022).

A practical approach combines MAT on a representative group of animals (herd serology) with PCR on urine and genital samples to identify active shedders. Interpretation should consider vaccination history, the serovar panel used and the reproductive history of the herd (Sykes et al., 2022).

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

8. TREATMENT

Treatment has two aims: to control acute disease in clinically affected animals, and to eliminate renal and genital carriage so that shedding stops. Early antimicrobial therapy shortens leptospiraemia and limits organ damage, but it cannot reverse established foetal infection or tissue injury.

Acute disease. Penicillin–streptomycin combinations, streptomycin or oxytetracycline are commonly used in acute cases. Supportive care is essential and may include intravenous fluids, haematinics, and blood transfusion in animals with severe haemolytic anaemia.

Elimination of carrier state. In experimentally infected cattle carrying *L. borgpetersenii* serovar Hardjo, a single intramuscular dose of streptomycin (25 mg/kg), long-acting oxytetracycline (20 mg/kg) or tilmicosin (10 mg/kg, subcutaneously) eliminated urinary shedding (Alt et al., 2001). Strategic whole-herd treatment combined with vaccination is often recommended during outbreaks of abortion or when introducing animals into a herd, so that both existing carriers and susceptible animals are dealt with at the same time (Martins & Lilenbaum, 2017).

Antimicrobial use should follow national regulations, label directions and prescribed withdrawal periods for milk and meat. Tilmicosin must be given only by a veterinarian because accidental injection is dangerous to humans. Given rising concern over antimicrobial resistance, treatment should be guided by a confirmed diagnosis and integrated with vaccination and management measures rather than used as a substitute for them.

9. PREVENTION AND CONTROL

Control of leptospirosis in ruminants depends on breaking the cycle between carrier animals, the environment and susceptible hosts. Because maintenance and incidental infections behave differently, a control plan should start by identifying the serovars involved and their likely sources (Martins & Lilenbaum, 2017; Ellis, 2015).

9.1 Vaccination

Inactivated whole-cell bacterins are used in many countries for cattle, and sometimes for sheep and goats. Protection is serovar-specific, lasts about a year and requires a primary course of two doses followed by annual or, in high-risk herds, six-monthly boosters. Vaccines differ in their ability to prevent renal colonisation and urinary shedding; a monovalent *L. borgpetersenii* serovar Hardjo vaccine has been shown to prevent colonisation and shedding after challenge (Bolin & Alt, 2001). For tropical conditions, vaccines should contain the serovars that actually circulate locally, ideally autochthonous strains, because imported vaccines may not match local serogroups (Martins & Lilenbaum, 2017). In India, ruminant leptospirosis vaccines are not widely available, which makes development of vaccines based on locally prevalent serogroups a priority.

9.2 Herd health and biosecurity

- Test and quarantine newly purchased animals; treat them with an effective antimicrobial and vaccinate before introduction
- Use artificial insemination with semen from tested bulls, or test breeding bulls before natural service
- Investigate every abortion and dispose of aborted foetuses, placentas and discharges by deep burial or incineration
- Maintain reproductive records so that repeat breeding, embryonic loss and abortion clusters are detected early
- Separate cattle from sheep, pigs and dogs where practical, and avoid co-grazing in high-risk seasons

9.3 Environmental management

- Restrict access to stagnant ponds, flooded pastures and canals; provide clean, piped or trough drinking water
- Improve shed drainage, maintain dry floors and bedding, and manage effluent and urine safely
- Conduct sustained rodent control in sheds, feed stores and fodder stacks, and keep stored feed rodent-proof
- Disinfect contaminated premises with suitable disinfectants after outbreaks

9.4 Surveillance

Regular serological screening of breeding herds, bulk milk ELISA where available, and PCR testing of urine from animals with reproductive problems help track infection and measure the impact of control measures. Linking these data to rainfall, flooding and human case reports allows early warning before the monsoon season (Sykes et al., 2022).

10. PUBLIC HEALTH SIGNIFICANCE AND ONE HEALTH APPROACH

Infected ruminants are an important source of human leptospirosis, particularly for dairy farmers, milkers, livestock owners, veterinarians, paravets, inseminators and abattoir workers. Exposure occurs through splashes of urine during milking, handling of aborted foetuses and placentas, assisting calvings, and working barefoot in contaminated sheds, fields and water. Serovar Hardjo is a recognised occupational infection of dairy workers, and human infections caused by serovars carried by cattle have been documented in tropical settings (Zarantonelli et al., 2018). Human disease ranges from a mild influenza-like illness to severe Weil's disease with jaundice, renal failure and pulmonary haemorrhage (Bharti et al., 2003; Haake & Levett, 2015).

In India, human leptospirosis is endemic in coastal and high-rainfall states, including Maharashtra, and outbreaks commonly follow monsoon flooding (Karpagam & Ganesh, 2020). Since rodents, dogs, livestock and humans share the same contaminated environment, control in any one species alone has limited impact. A One Health approach should include:

- Joint surveillance by animal husbandry, public health and environmental agencies, with sharing of serovar data from animals and humans

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

- Inclusion of leptospirosis in investigations of abortion and reproductive failure in livestock, and in human fever surveillance during the monsoon
- Worker protection through gloves, gumboots, aprons and eye protection, covering of skin wounds, hygiene after handling animals, and awareness of early symptoms
- Community education for farmers on the link between animal urine, flooding and human illness
- Coordinated rodent control, water management and drainage at village level
- Climate-informed early warning using rainfall and flood forecasts (Lau et al., 2010)

Veterinary public health professionals are well placed to lead this integration because they work across livestock health, food safety and zoonotic disease control.

11. RESEARCH GAPS AND FUTURE DIRECTIONS

Several gaps limit effective control of leptospirosis in ruminants, especially in India:

1. **Few local isolates.** Most Indian data are serological. Isolation and genomic characterisation of circulating strains from cattle, buffaloes, sheep and goats are needed to define the true serovar and species distribution.
2. **Economic impact.** Systematic estimates of losses from abortion, infertility and milk drop are lacking, which weakens the case for investment in control.
3. **Diagnostic access.** MAT is available in only a few reference laboratories. Validated, low-cost point-of-care tests, including LAMP and recombinant antigen-based rapid tests, are needed for field veterinarians.
4. **Genital leptospirosis.** The role of genital carriage in repeat breeding and embryonic loss under Indian conditions requires investigation using PCR on genital samples.
5. **Vaccines.** Locally relevant multivalent vaccines that prevent renal and genital colonisation, and studies of their cost-effectiveness, are a priority. Recombinant and subunit vaccines based on conserved outer membrane proteins merit evaluation.
6. **Integrated surveillance.** Linked animal, human, rodent and environmental surveillance, combined with climate data, is needed to understand transmission and predict outbreaks.
7. **Small ruminants and buffaloes.** These species are under-studied relative to cattle despite their large populations and close contact with water and people.

12. CONCLUSION

Leptospirosis is an endemic, under-recognised cause of reproductive failure, milk loss and mortality in ruminants, and ruminants are an important source of human infection. Host-adapted Hardjo strains maintain silent carriage in cattle and sheep, while incidental serovars from rodents, pigs and dogs cause severe acute disease, particularly in tropical, flood-prone smallholder systems such as those in India. Accurate diagnosis requires combining MAT-based herd serology with PCR or LAMP on urine, genital and foetal samples. Effective control rests on identifying and treating carriers, vaccinating with locally relevant serovars, improving water, drainage and rodent management, and protecting people who work with livestock. Isolation of local strains, development of affordable diagnostics and vaccines, and integrated One Health surveillance are the most important steps toward reducing the burden of leptospirosis in ruminants and in the communities that depend on them.

ACKNOWLEDGEMENT

The author thanks the Associate Dean, KNP College of Veterinary Science, Shirwal, and Maharashtra Animal and Fishery Sciences University, Nagpur, for providing facilities and support.

CONFLICT OF INTEREST

The author declares no conflict of interest.

FUNDING

This review received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

REFERENCES

1. *Adler, B., & de la Peña Moctezuma, A. (2010). Leptospira and leptospirosis. Veterinary Microbiology, 140(3–4), 287–296. https://doi.org/10.1016/j.vetmic.2009.03.012*
2. *Ahmed, A., Engelberts, M. F. M., Boer, K. R., Ahmed, N., & Hartskeerl, R. A. (2009). Development and validation of a real-time PCR for detection of pathogenic Leptospira species in clinical materials. PLoS ONE, 4(9), e7093.*
3. *Alt, D. P., Zuerner, R. L., & Bolin, C. A. (2001). Evaluation of antibiotics for treatment of cattle infected with Leptospira borgpetersenii serovar hardjo. Journal of the American Veterinary Medical Association, 219(5), 636–639.*
4. *Balamurugan, V., Alamuri, A., Bharathkumar, K., Patil, S. S., Govindaraj, G. N., Nagalingam, M., Krishnamoorthy, P., Rahman, H., & Shome, B. R. (2018). Prevalence of Leptospira serogroup-specific antibodies in cattle associated with reproductive problems in endemic states of India. Tropical Animal Health and Production, 50(5), 1131–1138.*
5. *Barragan, V., Nieto, N., Keim, P., & Pearson, T. (2017). Meta-analysis to estimate the load of Leptospira excreted in urine: Beyond rats as important sources of transmission in low-income rural communities. BMC Research Notes, 10, 71.*
6. *Bharti, A. R., Nally, J. E., Ricaldi, J. N., Matthias, M. A., Diaz, M. M., Lovett, M. A., Levett, P. N., Gilman, R. H., Willig, M. R., Gotuzzo, E., & Vinetz, J. M. (2003). Leptospirosis: A zoonotic disease of global importance. The Lancet Infectious Diseases, 3(12), 757–771.*
7. *Bolin, C. A., & Alt, D. P. (2001). Use of a monovalent leptospiral vaccine to prevent renal colonization and urinary shedding in cattle exposed to Leptospira borgpetersenii serovar hardjo. American Journal of Veterinary Research, 62(7), 995–1000.*
8. *Cilia, G., Bertelloni, F., & Fratini, F. (2020). Leptospira infections in domestic and wild animals. Pathogens, 9(7), 573.*
9. *Costa, F., Hagan, J. E., Calcagno, J., Kane, M., Torgerson, P., Martinez-Silveira, M. S., Stein, C., Abela-Ridder, B., & Ko, A. I. (2015). Global morbidity and mortality of leptospirosis: A*

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 11| November 2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

systematic review. PLoS Neglected Tropical Diseases, 9(9), e0003898.

10. *Ellis, W. A. (1994). Leptospirosis as a cause of reproductive failure. Veterinary Clinics of North America: Food Animal Practice, 10(3), 463–478.*
11. *Ellis, W. A. (2015). Animal leptospirosis. In B. Adler (Ed.), Leptospira and Leptospirosis. Current Topics in Microbiology and Immunology (Vol. 387, pp. 99–137). Springer.*
12. *Goris, M. G. A., & Hartskeerl, R. A. (2014). Leptospirosis serodiagnosis by the microscopic agglutination test. Current Protocols in Microbiology, 32, Unit 12E.5.*
13. *Grooms, D. L. (2006). Reproductive losses caused by bovine viral diarrhea virus and leptospirosis. Theriogenology, 66(3), 624–628.*
14. *Haake, D. A., & Levett, P. N. (2015). Leptospirosis in humans. In B. Adler (Ed.), Leptospira and Leptospirosis. Current Topics in Microbiology and Immunology (Vol. 387, pp. 65–97). Springer.*
15. *Hamond, C., Martins, G., Loureiro, A. P., Pestana, C., Lawson-Ferreira, R., Medeiros, M. A., & Lilenbaum, W. (2014). Urinary PCR as an increasingly useful tool for an accurate diagnosis of leptospirosis in livestock. Veterinary Research Communications, 38(1), 81–85.*
16. *Karpagam, K. B., & Ganesh, B. (2020). Leptospirosis: A neglected tropical zoonotic infection of public health importance—an updated review. European Journal of Clinical Microbiology & Infectious Diseases, 39(5), 835–846.*
17. *Ko, A. I., Goarant, C., & Picardeau, M. (2009). Leptospira: The dawn of the molecular genetics era for an emerging zoonotic pathogen. Nature Reviews Microbiology, 7(10), 736–747.*
18. *Lau, C. L., Smythe, L. D., Craig, S. B., & Weinstein, P. (2010). Climate change, flooding, urbanisation and leptospirosis: Fuelling the fire? Transactions of the Royal Society of Tropical Medicine and Hygiene, 104(10), 631–638.*
19. *Levett, P. N. (2001). Leptospirosis. Clinical Microbiology Reviews, 14(2), 296–326.*
20. *Lilenbaum, W., & Martins, G. (2014). Leptospirosis in cattle: A challenging scenario for the understanding of the epidemiology. Transboundary and Emerging Diseases, 61(Suppl. 1), 63–68.*
21. *Loureiro, A. P., & Lilenbaum, W. (2020). Genital bovine leptospirosis: A new look for an old disease. Theriogenology, 141, 41–47.*
22. *Martins, G., & Lilenbaum, W. (2014). Leptospirosis in sheep and goats under tropical conditions. Tropical Animal Health and Production, 46(1), 11–17.*
23. *Martins, G., & Lilenbaum, W. (2017). Control of bovine leptospirosis: Aspects for consideration in a tropical environment. Research in Veterinary Science, 112, 156–160.*
24. *Musso, D., & La Scola, B. (2013). Laboratory diagnosis of leptospirosis: A challenge. Journal of Microbiology, Immunology and Infection, 46(4), 245–252.*
25. *Mwachui, M. A., Crump, L., Hartskeerl, R., Zinsstag, J., & Hattendorf, J. (2015). Environmental and behavioural determinants of leptospirosis transmission: A systematic review. PLoS Neglected Tropical Diseases, 9(9), e0003843.*
26. *Picardeau, M. (2017). Virulence of the zoonotic agent of leptospirosis: Still terra incognita? Nature Reviews Microbiology, 15(5), 297–307.*
27. *Sonthayanon, P., Chierakul, W., Wuthiekanun, V., Thaipadungpanit, J., Kalambaheti, T., Boonsilp, S., Amornchai, P., Smythe, L. D., Limmathurotsakul, D., Day, N. P., & Peacock, S. J. (2011). Accuracy of loop-mediated isothermal amplification for diagnosis of human leptospirosis in Thailand. American Journal of Tropical Medicine and Hygiene, 84(4), 614–620.*
28. *Stoddard, R. A., Gee, J. E., Wilkins, P. P., McCaustland, K., & Hoffmaster, A. R. (2009). Detection of pathogenic Leptospira spp. through TaqMan polymerase chain reaction targeting the LipL32 gene. Diagnostic Microbiology and Infectious Disease, 64(3), 247–255.*
29. *Sykes, J. E., Reagan, K. L., Nally, J. E., Galloway, R. L., & Haake, D. A. (2022). Role of diagnostics in epidemiology, management, surveillance, and control of leptospirosis. Pathogens, 11(4), 395.*
30. *Torgerson, P. R., Hagan, J. E., Costa, F., Calcagno, J., Kane, M., Martinez-Silveira, M. S., Goris, M. G. A., Stein, C., Ko, A. I., & Abela-Ridder, B. (2015). Global burden of leptospirosis: Estimated in terms of disability adjusted life years. PLoS Neglected Tropical Diseases, 9(10), e0004122.*
31. *Vincent, A. T., Schiettekatte, O., Goarant, C., Neela, V. K., Bernet, E., Thibeaux, R., Ismail, N., Mohd Khalid, M. K. N., Amran, F., Masuzawa, T., Nakao, R., Amara Korba, A., Bourhy, P., Veyrier, F. J., & Picardeau, M. (2019). Revisiting the taxonomy and evolution of pathogenicity of the genus Leptospira through the prism of genomics. PLoS Neglected Tropical Diseases, 13(5), e0007270.*
32. *World Organisation for Animal Health (WOAH). (2021). Leptospirosis (Chapter 3.1.12). In Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. WOAH.*
33. *Zarantonelli, L., Suanes, A., Meny, P., Buroni, F., Nieves, C., Salaberry, X., Briano, C., Ashfield, N., Da Silva Silveira, C., Dutra, F., Easton, C., Fraga, M., Giannitti, F., Hamond, C., Macías-Rioseco, M., Menéndez, C., Mortola, A., Picardeau, M., Quintero, J., … Buschiazzo, A. (2018). Isolation of pathogenic Leptospira strains from naturally infected cattle in Uruguay reveals high serovar diversity, and uncovers a relevant risk for human leptospirosis. PLoS Neglected Tropical Diseases, 12(9), e0006694.*