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Bridging Gaps Between Humans and Animals: Managing Leptospirosis the One Health Way

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Abstract:

Leptospirosis is a globally significant zoonotic disease, driven by intricate interactions among humans, animals, and the environment. Caused by pathogenic *Leptospira* species, the disease exhibits a broad spectrum of clinical manifestations in humans and animals, ranging from subclinical infections to severe multi-organ involvement or reproductive losses. Transmission primarily occurs via urine from maintenance hosts including rodents, livestock, and companion animals contaminating soil and water, with human exposure heightened through occupational, recreational, and environmental contact. Environmental and climatic factors, such as flooding, poor sanitation, and warm, waterlogged conditions, amplify transmission risk, while socioeconomic determinants contribute to sustained outbreaks. Diagnosis remains challenging due to nonspecific symptoms, necessitating laboratory confirmation through molecular, serological, or culture-based methods. Effective prevention and control demand a One Health approach integrating coordinated animal vaccination, environmental management, occupational safety, public awareness, and interdisciplinary surveillance. Sustainable leptospirosis management relies on cross-sector collaboration among veterinary, medical, and environmental stakeholders, emphasizing ecosystem-level interventions to reduce disease burden and mitigate outbreaks. This integrated perspective underscores that leptospirosis is not solely a medical concern but a multifaceted ecological and public health challenge.

Keywords: Environmental Risk, Epidemiology, Leptospira, Leptospirosis, One Health, Surveillance, Zoonosis

Introduction:

Leptospirosis is a zoonotic disease of significant global importance, occurring in urban settings of

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both industrialised and developing nations as well as in diverse rural environments (Vinetz, 2001). The disease is attributed to pathogenic species of the genus *Leptospira* which is thin, motile, spiral-shaped bacteria capable of penetrating host tissues and persisting in moist environments and is widely distributed, contributing substantially to morbidity and mortality in both humans and animals (Bharti *et al.,* 2003). The family *Leptospiraceae* comprises two genera, *Leptospira* and *Leptonema*, with *Leptospira interrogans* being the most frequently implicated species in clinical infections. Transmission occurs through both direct and indirect exposure, primarily via urine-shedding by infected animals that contaminates soil, water, and surrounding environments (Antony, 1996). Human infection typically arises during routine occupational or recreational activities involving contact with contaminated materials, placing farmers, veterinarians, livestock handlers, residents of waterlogged areas, and adventure travellers particularly those engaging in water-based sports at heightened risk (Lau *et al.,* 2010). Globally, leptospirosis accounts for more than one million severe cases and nearly 60,000 deaths annually, with the highest burden concentrated in developing regions characterised by monsoon-associated flooding, inadequate sanitation, and high rodent density (Thayaparan *et al.,* 2013). The intricate interactions among humans, animals, and the environment position leptospirosis as a prime example of a One Health challenge, underscoring the need for coordinated medical, veterinary, and environmental interventions (Morgan *et al.,* 2002; Haake *et al.,* 2002). Strengthening public awareness and improving understanding of transmission pathways are critical to mitigating the growing threat posed by this widespread zoonosis (Costa *et al.,* 2015).

Microbiological and Pathogenic Features of *Leptospira*:

Leptospirosis is caused by pathogenic bacteria of the genus *Leptospira*, widely recognized for their zoonotic potential and global distribution, and these leptospires are spirochaetes belonging to the order *Spirochaetales* and the family *Leptospiraceae*, which comprises the genera *Leptospira* and *Leptonema*, representing an early-diverging group of bacteria in evolutionary history (Levett, 2004). Leptospira are obligate aerobes with an optimum growth temperature ranging from 28^{o} C to 30^{o} C (Paster *et al.,* 1991). Historically, the genus was classified into only two species, *Leptospira interrogans* (pathogenic) and *Leptospira biflexa* (saprophytic) but advancements in molecular phylogeny have expanded this framework to more than 64 recognized species, now grouped into pathogenic (P1), intermediate (P2), and saprophytic (S) clusters (Adler and de la Peña Moctezuma, 2010; Vincent *et al.,* 2019). Pathogenic *Leptospira* comprise over 300 serovars and exhibit a remarkable ability to survive in moist environments (Levett, 2004). Reservoir animals often remain asymptomatic carriers and shed the organisms through urine for prolonged periods, contaminating water bodies, soil, and the environment, which subsequently become sources of infection for incidental hosts such as humans, dogs, and livestock (Levett, 2004). Leptospira species are thin, tightly coiled spirochetes, typically 6–20 µm long and 0.1 µm in diameter, with hook-

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shaped ends giving them the characteristic question-mark appearance (Bharti *et al.,* 2003). They possess two periplasmic flagella inserted at each end, enabling rapid spiral motility that facilitates tissue penetration and dissemination throughout the host's body. Their cell envelope resembles that of gram-negative bacteria but contains unique features; the LPS of Leptospira has lower endotoxic activity compared to enterobacterial LPS, yet it plays a critical role in immune recognition and serovar classification (Haake and Levett, 2015). The outer membrane of pathogenic Leptospira contains several virulence-associated proteins, including LipL32, the most abundant outer membrane protein present only in pathogenic strains, and LigA/LigB proteins, which contribute to adhesion, invasion, and immune evasion. These molecules are crucial for binding extracellular matrix components such as fibronectin, collagen, and laminin, promoting colonization of host tissues (Cullen *et al.,* 2002). Pathogenic strains grow well at 37°C, consistent with mammalian hosts. In the environment, the organisms survive for weeks to months in moist, neutral pH conditions and are particularly abundant after heavy rainfall, floods, or in stagnant water, which explains seasonal outbreaks in tropical and subtropical countries (Ko *et al.,* 2009). Their impressive environmental persistence and wide host range demonstrate why leptospirosis is deeply interconnected with animal reservoirs, ecological conditions, and human exposures making it a quintessential One Health disease.

Animal Hosts and Transmission Dynamics:

Leptospirosis persists globally through a wide spectrum of maintenance hosts that carry pathogenic *Leptospira* spp. Asymptomatically while continuously shedding the organisms in urine. These hosts support chronic renal colonization, enabling long-term environmental contamination. Rodents, particularly *Rattus norvegicus*, represent the most significant reservoirs worldwide and maintain *L. interrogans* serovar Icterohaemorrhagiae, a major cause of severe human disease in urban regions (Ko *et al.,* 2009). Among domestic animals, cattle, pigs, dogs, and small ruminants sustain the serovars Hardjo, Pomona, Canicola, and Tarassovi, respectively, reinforcing endemicity in rural and mixed livestock systems (Ellis, 2015). Numerous wildlife species such as raccoons, mongooses, bats, and opossums further broaden the ecological range of *Leptospira*, facilitating its persistence across forested, agricultural, and peri-urban environments (Costa *et al.,* 2015; Thilageshwaran *et al.,* 2024). Humans function as incidental, dead-end hosts, acquiring infection primarily through contact with urine from infected animals or contaminated water, soil, or vegetation, and human-to-human transmission is extremely rare. Exposure commonly occurs during agricultural work, livestock handling, recreational water activities, sewage operations, and following flooding events, which enhance bacterial survival in moist environments (Bharti *et al.,* 2003). In rural settings, farmers, veterinarians, dairy workers, and slaughterhouse personnel are at elevated risk, whereas in urban areas, rodent proliferation, inadequate sanitation, and stagnant wastewater increase exposure, especially in densely populated settlements (Costa *et al.,* 2015; Thilageshwaran *et al.,* 2024).

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Sustained transmission depends heavily on environmental and climatic conditions, particularly warm temperatures, high rainfall, and waterlogging, which prolong bacterial survival. One Health stressors including rapid urbanization, increased animal–human interface, poor waste management, and climate-change-associated flooding further perpetuate the transmission cycle. This interconnected animal–environment-human dynamic underscores the need for integrated surveillance, environmental sanitation, rodent control, and coordinated veterinary–public health efforts to reduce zoonotic transmission.

Ecological Drivers and Exposure Risks:

Environmental conditions strongly influence the survival and transmission of *Leptospira*. The pathogen can persist for extended periods in moist, warm, and waterlogged environments, making high rainfall, flooding, and inadequate drainage major drivers of outbreaks (Bharti *et al.,* 2003). During monsoon seasons, stagnant water and overflowed sewage systems increase human exposure, while flooding enhances rodent movement and environmental contamination. Urban areas face added risks due to inadequate sanitation, open drains, accumulated waste, and high rodent density, all of which collectively sustain a continuous environmental reservoir of infection (Costa *et al.,* 2015). In rural settings, wet agricultural fields, irrigation channels, and livestock sheds provide ideal microhabitats for leptospiral survival. Occupational activities such as rice farming, animal handling, cleaning livestock sheds, and working barefoot in muddy fields further heighten contact with contaminated soil or water, while recreational exposure through swimming or wading in natural water bodies also contributes. Climate change, through increasing extreme rainfall and flooding events, amplifies environmental contamination and expands geographic risk zones (Lau *et al.,* 2010). These environmental and climatic factors interact with socioeconomic conditions including overcrowded settlements, poor waste management, and close animal–human proximity to create a persistent cycle of environmental contamination and human exposure as illustrated in Fig. 1.

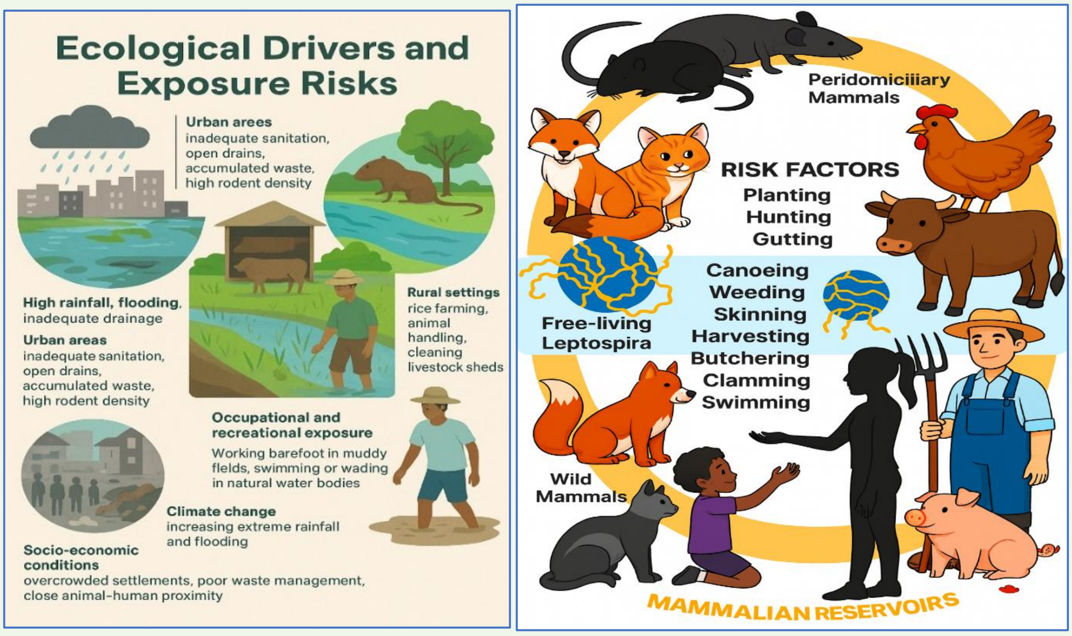

Fig. 1. Schematic representation of leptospirosis transmission pathways and key ecological and behavioral risk factors.

Spectrum of Clinical Signs Across Host Species:

Clinical manifestations of leptospirosis vary widely in both humans and animals, ranging from subclinical infection to severe multisystem disease. In humans, the illness typically presents as an acute

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febrile syndrome characterized by fever, myalgia, conjunctival suffusion, and headache. A subset of patients progresses to Weil's disease marked by jaundice, renal failure, and hemorrhagic tendencies or to severe pulmonary hemorrhage syndrome, which carries a high mortality rate (Bharti *et al.,* 2003; Ko *et al.,* 2009). The clinical course is generally biphasic, with an initial septicemic phase followed by an immune phase in which organ dysfunction becomes evident.

In animals, infection is often asymptomatic in maintenance hosts but may cause reproductive losses and systemic disease in incidental hosts. Cattle infected with serovar Hardjo commonly exhibit infertility, abortion, stillbirth, and reduced milk production, whereas pigs may develop fever, jaundice, and reproductive failure (Ellis, 2015). Dogs infected with serovar Canicola or emerging serovars frequently present with acute kidney injury, hepatic dysfunction, vomiting, and lethargy, and may also develop pulmonary complications similar to those observed in humans (Sykes *et al.,* 2011). The clinical spectrum across species reflects serovar–host adaptation, host immune responses, and pathogen load.

Diagnostic Approaches and Surveillance Strategies:

Diagnosis of leptospirosis requires integrating clinical suspicion, epidemiological exposure, and laboratory confirmation, as early symptoms are nonspecific and overlap with other febrile illnesses. Laboratory diagnosis relies on direct detection of the organism during the acute phase and serological testing during the immune phase. Molecular assays such as PCR enable early, sensitive detection of leptospiral DNA in blood, urine, or tissue samples and are now considered the preferred method for early diagnosis (Picardeau, 2013). The Microscopic Agglutination Test (MAT) remains the international reference standard for serological confirmation, although it requires paired sera, live antigen panels, and specialized laboratory facilities. Rapid tests including ELISA-IgM and lateral flow assays are widely used for screening but demonstrate variable specificity depending on endemicity and circulating serovars (Levett, 2001). Surveillance systems are essential for monitoring transmission trends, detecting outbreaks, and guiding public health interventions. Effective surveillance integrates human case reporting, animal infection monitoring, and environmental assessments, aligning with One Health frameworks. In many endemic regions, leptospirosis remains underreported due to limited diagnostic capacity, misclassification with other acute febrile illnesses, and the absence of routine veterinary testing (Costa *et al.,* 2015). Enhanced surveillance strategies include serovar mapping, rodent population monitoring, livestock screening, and environmental sampling of water bodies to identify high-risk zones. Incorporating climate and rainfall data into surveillance models has strengthened early warning systems, enabled prediction of seasonal outbreaks and facilitating targeted control measures (Lau *et al.,* 2010).

One Health–Based Prevention and Management:

Effective prevention and control of leptospirosis require a coordinated One Health strategy that integrates human, animal, and environmental interventions. In animals, control measures focus on

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livestock and dog vaccination, improved farm biosecurity, and limiting access to contaminated water. Vaccination against key serovars such as Hardjo in cattle and Pomona in pigs reduces renal colonization and environmental shedding, thereby lowering zoonotic transmission (Ellis, 2015). Rodent control through sanitation, waste management, and targeted population reduction is essential, particularly in urban settings. Environmental interventions include improved drainage, flood management, safe water access, and minimizing human exposure to stagnant or contaminated water. Public health strategies emphasize early outbreak detection, chemoprophylaxis for high-risk groups during floods or occupational exposure, and community education on protective behaviours (WHO, 2003). At the human–animal–environment interface, integrated surveillance, coordinated outbreak response, and cross-sectoral data sharing strengthen control efforts. Monitoring serovar circulation in livestock and wildlife, identifying rodent hotspots, and using rainfall-linked early warning systems have improved targeted interventions, especially in flood-prone tropical regions (Costa *et al.,* 2015; Lau *et al.,* 2010). Collectively, these strategies underscore that sustainable leptospirosis control depends on unified action across veterinary, public health, and environmental sectors.

Analysis and Implications:

Leptospirosis remains a globally significant zoonotic disease, with transmission driven by complex interactions among humans, animals, and the environment. Small mammals serve as primary reservoirs, while large herbivores act as additional important sources of infection (Haake and Levett, 2015). Maintenance hosts including rodents, livestock, and companion animals shed pathogenic *Leptospira* in urine, contaminating soil and water, which constitute the main sources of human infection (Haake and Levett, 2015). Environmental factors such as flooding, poor sanitation, and occupational exposure further amplify transmission risk, highlighting the critical role of ecological and socioeconomic determinants in disease dynamics. Clinically, leptospirosis exhibits a broad spectrum, ranging from subclinical or mild febrile illness to severe multi-organ involvement in humans, and reproductive losses or renal disease in animals. Early diagnosis is challenging due to nonspecific symptoms, necessitating laboratory confirmation via serology, PCR, or culture (Levett, 2004). Surveillance programs integrating human and animal health data are essential for timely outbreak detection and risk mapping. Prevention and control require a One Health approach, combining environmental management, animal vaccination, public awareness, and occupational safety measures. Effective collaboration among veterinary, medical, and environmental sectors can reduce disease burden, mitigate outbreaks, and inform policy interventions (Costa *et al.,* 2015). This integrative perspective underscores that controlling leptospirosis is not solely a medical challenge but an ecosystem-level imperative.

Conclusions and Future Directions:

Leptospirosis is a complex zoonotic disease with significant public health and veterinary implications, sustained by persistent animal reservoirs and environmental contamination. Its clinical heterogeneity in humans and animals, along with diagnostic challenges, highlights the need for vigilant surveillance and timely intervention. Effective prevention and control require an integrated One Health approach, incorporating coordinated efforts in animal vaccination, environmental management, occupational safety, and community education. Strengthened interdisciplinary collaboration among medical, veterinary, and environmental sectors is essential to mitigate transmission, reduce morbidity, and guide evidence-based policy development. Ultimately, addressing leptospirosis demands a comprehensive strategy that extends beyond individual health domains and accounts for ecosystem-level determinants.

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