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Hemorrhagic septicemia in livestock: economic impacts on milk and meat production, epidemiology, diagnostic advances, and control strategies

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ABSTRACT

Hemorrhagic Septicemia (HS) is a rapidly progressing, highly fatal bacterial disease that significantly constrains livestock productivity, particularly in cattle and buffalo across tropical and subtropical regions. The disease is caused by the Gram-negative bacterium *Pasteurella multocida*, predominantly associated with capsular serotypes B:2 and E:2. HS outbreaks are characterized by acute septicemia, high mortality, and substantial economic losses in dairy and meat production systems. In endemic regions of South and Southeast Asia and Africa, HS significantly reduces milk yield, impairs growth performance, increases mortality of productive animals, and elevates costs associated with treatment and vaccination. This review synthesizes current knowledge on the epidemiology, transmission dynamics, and global distribution of HS, with particular emphasis on its economic impact on livestock production systems. Advances in diagnostic technologies, including polymerase chain reaction (PCR), molecular typing, and genome-based approaches, have improved disease detection and epidemiological surveillance. Concurrently, vaccine development has progressed from conventional bacterins to recombinant, sub-unit, and DNA-based platforms, offering improved prospects for long-term disease control. Despite these advancements, persistent challenges—including incomplete vaccination coverage, environmental stressors, and the emergence of antimicrobial-resistant *P. multocida* strains—continue to limit effective disease management. Addressing these constraints requires integrated prevention strategies combining vaccination, biosecurity, and coordinated surveillance. Future research should focus on molecular epidemiology, vaccine innovation, and antimicrobial stewardship to reduce the global burden of HS and support sustainable livestock production in endemic regions. These efforts are essential for improving livestock productivity and ensuring sustainable milk and meat production systems in endemic regions.

Keywords: Hemorrhagic septicemia, *Pasteurella multocida*, Livestock production, Epidemiology, Economic impact, Molecular diagnostics, Antimicrobial resistance, Vaccination

1. Introduction

Hemorrhagic septicemia (HS) is an acute and highly fatal bacterial disease that remains a major constraint to livestock production and productivity in tropical and subtropical regions (Peng *et al.*, 2019) [1]. Clinically, the disease presents with a sudden onset of high fever, respiratory distress, subcutaneous edema, and, in many cases, peracute death, often resulting in severe herd-level losses during outbreaks (Wilson *et al.*, 2013; Zamri-Saad *et al.*, 2016; Peng *et al.*, 2019) [2, 3, 1]. Among bovine diseases, HS is considered one of the most economically significant, particularly affecting cattle and buffalo populations in developing countries (Lestari *et al.*, 2025; Cuevas *et al.*, 2020; Abusalab *et al.*, 2003) [5, 6, 4]. The causative agent, *Pasteurella multocida*, is a Gram-negative bacterium belonging to the family Pasteurellaceae, with capsular serotypes B:2 and E:2 primarily responsible for HS outbreaks in Asia and Africa, respectively (Harper *et al.*, 2006; Ferreira *et al.*, 2015; Almoheer *et al.*, 2022) [8, 9, 7]. Beyond HS, *P. multocida* is also associated with other forms of pasteurellosis and is recognized as an opportunistic zoonotic pathogen (Ferreira *et al.*, 2015; Abera *et al.*, 2022) [9, 10].

Infection typically occurs through inhalation or ingestion of contaminated secretions, feed, or water, followed by rapid systemic dissemination leading to septicemia. Carrier animals harboring the pathogen in the upper respiratory tract play a critical role in maintaining infection cycles and facilitating transmission within herds (Cuevas *et al.*, 2020) [6]. Disease outcome depends on complex interactions among host immunity, environmental stressors, and pathogen virulence (Lin *et al.*, 2022) [11].

Cattle and water buffalo are the primary hosts, with buffalo often exhibiting higher mortality rates, posing significant challenges in regions where they are central to milk production and agricultural systems (Wilson

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et al., 2013; Peng *et al.*, 2019) [2, 1]. Although HS predominantly affects bovines, sporadic infections have been reported in other domestic and wild species, reflecting the pathogen's broad host range (Farooq *et al.*, 2011; Piorunek *et al.*, 2023; Shome *et al.*, 2024) [12, 13, 14].

HS is widely distributed across tropical and subtropical regions, where climatic conditions such as high humidity, heavy rainfall, and temperature fluctuations favor disease outbreaks (Lestari *et al.*, 2025; Benkirane and De Alwis, 2002; Moustafa *et al.*, 2013) [5, 15]. Stress factors—including transportation, overcrowding, poor nutrition, and suboptimal management—further increase susceptibility and transmission risk (Abdulkadir *et al.*, 2024) [16].

The economic burden of HS extends beyond mortality, encompassing reduced milk yield, impaired growth performance, loss of draft power, and increased costs associated with treatment and vaccination. These impacts are particularly severe in low- and middle-income countries, where livestock are integral to livelihoods and food security (Shome *et al.*, 2019; Michael *et al.*, 2021) [18, 17].

Despite the availability of vaccines, HS persists in endemic regions due to incomplete vaccination coverage, limited surveillance infrastructure, environmental stressors, and the emergence of antimicrobial-resistant *P. multocida* strains. This review is particularly relevant to tropical livestock production systems, where HS remains endemic and continues to significantly impact milk and meat production systems. The review also highlights advances in diagnostics, vaccination strategies, and integrated control approaches.

1.1 Literature search strategy

A comprehensive literature search was conducted using electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar. Relevant articles published between 2000 and 2025 were retrieved using keywords such as “Hemorrhagic septicemia (HS),” “*Pasteurella multocida*,” “livestock diseases,” “economic impact,” “milk production,” “meat production,” “diagnostics,” “vaccines,” and “antimicrobial resistance”. Studies were selected based on relevance, scientific quality, and applicability to livestock production systems. Both original research articles and review papers were included, while non-peer-reviewed sources and duplicate studies were excluded. The collected literature was critically evaluated, compared, and synthesized to provide a comprehensive and unbiased overview of the topic. Where applicable, preference was given to peer-reviewed

and high-impact studies to ensure reliability of the synthesized information.

2. Etiology and pathogenesis

2.1 Causative agent

Hemorrhagic septicemia (HS) is caused by *Pasteurella multocida*, a Gram-negative, non-motile, facultatively anaerobic coccobacillus belonging to the family Pasteurellaceae. Microscopically, the organism exhibits characteristic bipolar (“safety-pin”) staining morphology and forms smooth, non-hemolytic colonies on enriched media such as blood agar (Sajid *et al.*, 2024; Cuevas *et al.*, 2020; Harper *et al.*, 2006) [19, 6, 8].

P. multocida comprises four recognized subspecies—*multocida*, *septica*, *gallicida*, and *tigris*—and belongs to the broader family that includes genera such as *Pasteurella*, *Actinobacillus*, and *Haemophilus*, many of which colonize mucosal surfaces of vertebrate hosts (Wilson *et al.*, 2013; Harper *et al.*, 2017; Duan *et al.*, 2024) [2, 21, 20].

The organism possesses a single circular chromosome (~2.25 Mb) with multiple coding regions and ribosomal operons, along with plasmids that may encode virulence factors and antimicrobial resistance traits (Bosch *et al.*, 2004 [22]; May *et al.*, 2001 [23]; Peng *et al.*, 2019 [1]; Mahboob *et al.*, 2023 [24]; El-Demerdash *et al.*, 2023 [25]; Zhao *et al.*, 2021) [26]. These plasmids can also serve as useful epidemiological markers for strain differentiation (Vu-Khac *et al.*, 2020) [27].

Based on capsular antigen composition, *P. multocida* is classified into serogroups A, B, D, E, and F, of which serotypes B:2 and E:2 are primarily responsible for HS outbreaks in Asia and Africa, respectively (Benkirane and De Alwis, 2002 [15]; Ferreira *et al.*, 2015 [9]; Shivachandra *et al.*, 2011 [28]; Richardson *et al.*, 2023) [29]. Although the bacterium infects a wide range of hosts, cattle and buffalo are the principal species affected by HS, with isolation from outbreak cases confirming its etiological role (Bhardwaj *et al.*, 2025; Jatoti *et al.*, 2024) [30, 31].

Pathogenicity is largely mediated by structural components such as the capsular polysaccharide and other virulence determinants that facilitate colonization, immune evasion, and systemic dissemination (Sajid *et al.*, 2024; Harper *et al.*, 2006) [19, 8]. While serotypes B:2 and E:2 consistently reproduce disease experimentally, other type B strains have been associated with sporadic outbreaks with variable pathogenicity (Mostaan *et al.*, 2020; Orynbayev *et al.*, 2019) [32, 33].

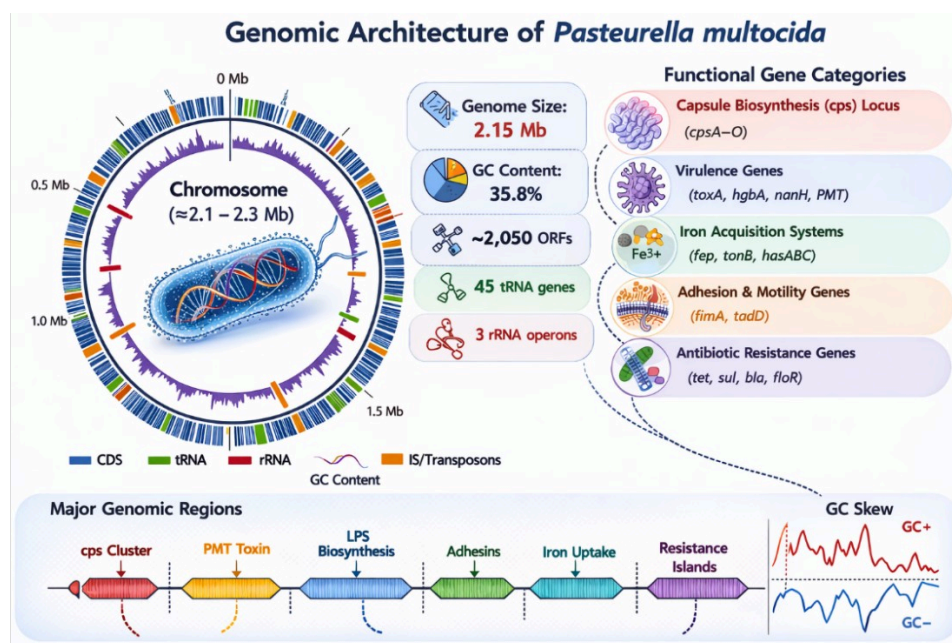


Figure 1. Genomic architecture of *P. multocida*

2.2 Virulence factors

The virulence of *P. multocida* is mediated by a combination of structural and molecular determinants that enhance survival, colonization, and systemic spread within the host (Abed *et al.*, 2020) [34]. The polysaccharide capsule is a key virulence factor that protects the bacterium from phagocytosis and complement-mediated killing, thereby facilitating immune evasion (Richardson *et al.*, 2023) [29].

Lipopolysaccharide (LPS), a major component of the outer membrane, plays a central role in pathogenesis by inducing strong inflammatory responses, vascular damage, and endotoxemia, ultimately contributing to septicemia (Harper *et al.*, 2012; Harper *et al.*, 2017) [21, 35].

In addition, multiple virulence-associated genes involved in capsule biosynthesis, adhesion, and iron acquisition contribute to pathogenicity and host adaptation (Verma *et al.*, 2013; Ferreira *et al.*, 2012; Tawab *et al.*, 2020; Zhao *et al.*, 2021) [37, 36, 38, 26]. Outer membrane proteins (OMPs) and adhesins facilitate bacterial attachment to epithelial surfaces, promoting colonization and invasion of host tissues (Harper *et al.*, 2006; Shivachandra *et al.*, 2011) [8, 28].

Iron acquisition systems further enhance bacterial survival by enabling efficient utilization of host iron sources, which are essential for growth during infection. Variations in capsular structure and associated virulence factors, particularly among serotypes B and E, contribute to differences in pathogenicity and epidemiological patterns (Richardson *et al.*, 2023; Desem *et al.*, 2023) [29, 39]. Collectively, these mechanisms enable *P. multocida* to evade host defenses, proliferate

rapidly, and initiate systemic infection (Peng *et al.*, 2016; Doyle-Baker *et al.*, 2020) [41, 40].

2.3 Pathogenesis

The pathogenesis of HS is characterized by rapid progression from localized infection to fatal septicemia. Infection typically occurs through inhalation or ingestion of contaminated materials, allowing *P. multocida* to enter via respiratory or mucosal surfaces (Abera *et al.*, 2022) [10].

Following entry, the bacteria colonize the upper respiratory tract, where they may persist transiently. Under predisposing conditions such as stress, poor nutrition, or immunosuppression, the organism multiplies rapidly and invades the bloodstream, leading to systemic dissemination (Harper *et al.*, 2017; Cuevas *et al.*, 2020) [21, 6].

The release of endotoxins and other virulence factors triggers severe inflammatory responses, vascular damage, and increased permeability (Kubatzky, 2022) [42]. These processes result in widespread edema, hemorrhages, and congestion of internal organs, particularly in the lungs, lymph nodes, and subcutaneous tissues of the throat and brisket region—hallmark features of HS (Shome *et al.*, 2019) [18].

Disease progression is typically rapid, and animals may die within a short period, sometimes before the onset of clear clinical signs. When clinical disease is evident, pathological findings include severe edema, petechial hemorrhages, and generalized organ congestion, reflecting systemic infection (Moustafa *et al.*, 2013; Doyle-Baker *et al.*, 2020; Sajid *et al.*, 2024) [40, 19]. Endotoxin release plays a central role in mediating systemic inflammation and vascular injury during disease progression (Kubatzky, 2022; Horadagoda *et al.*, 2001) [42, 43].

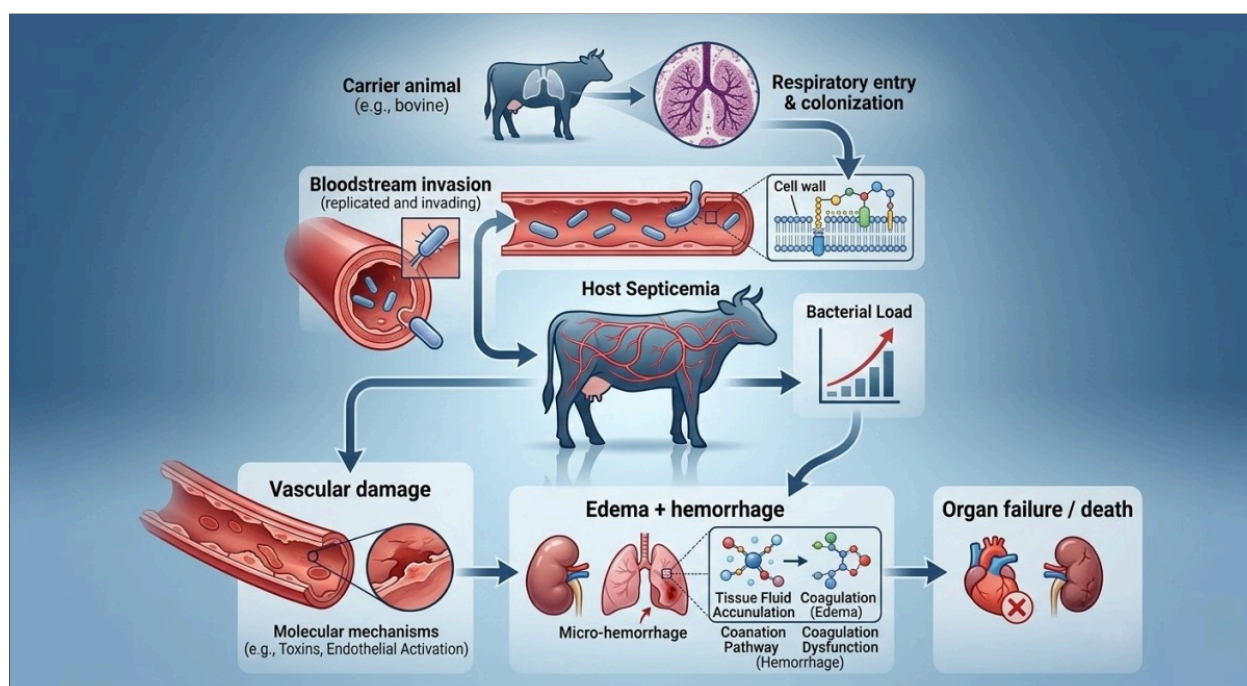


Figure 2. Pathogenesis of hemorrhagic septicemia caused by *P. multocida*

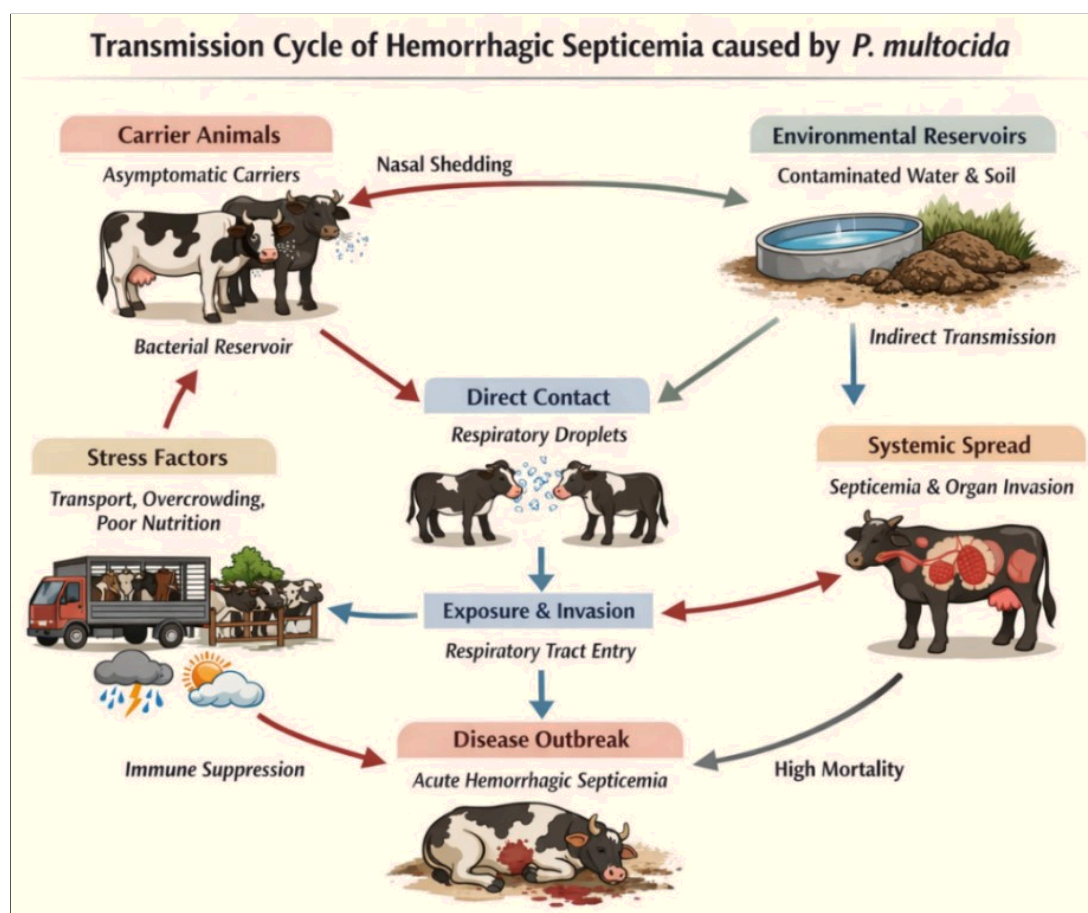


Figure 3. Transmission cycle of hemorrhagic septicemia caused by *P. multocida*

3. Transmission

Transmission of Hemorrhagic Septicemia (HS) primarily occurs through direct contact with infected or carrier animals shedding *Pasteurella multocida* via respiratory secretions. The pathogen is commonly transmitted through nasal discharge, saliva, and other bodily fluids, enabling rapid spread in densely populated herds (Cuevas *et al.*, 2020) [6]. Indirect transmission may also occur through contaminated feed, water, and fomites, particularly under inadequate biosecurity conditions. The organism can persist for short periods in moist environments, enhancing indirect transmission, particularly under conditions of high humidity and rainfall. Outbreaks are frequently associated with high humidity, heavy rainfall, and sudden temperature fluctuations, which enhance pathogen persistence (Abdulkadir *et al.*, 2024) [16]. Host stress is a major predisposing factor influencing disease occurrence. Conditions such as transportation, overcrowding, poor nutrition, and concurrent infections compromise immune defenses, increasing susceptibility and facilitating transmission (Farooq *et al.*, 2011) [12]. Consequently, HS outbreaks are often linked to periods of environmental or management stress. Proper carcass disposal and improved husbandry practices are essential to reduce transmission risk (Chanda *et al.*, 2024) [44].

4. Clinical signs, lesions, and differential diagnosis

4.1 Clinical signs

HS is characterized by a rapid and often fatal clinical course. Early signs include high fever (41–42 °C), depression, anorexia, lacrimation, salivation, and nasal discharge, reflecting initial respiratory involvement (Cuevas *et al.*, 2020) [6]. As the disease progresses, severe respiratory distress develops, including labored breathing and open-mouth respiration. A hallmark feature is subcutaneous edema in the throat, dewlap, and brisket regions, resulting from vascular leakage and inflammatory responses (Shome *et al.*, 2019) [18]. Advanced stages are characterized by weakness, recumbency, and cyanotic mu-

cous membranes due to circulatory and respiratory failure. Disease progression is typically rapid, with death occurring within 24–48 hours, and in some cases, sudden death without prior clinical signs—particularly in buffalo (Farooq *et al.*, 2011; Moustafa *et al.*, 2013) [12]. Experimental studies further confirm the septicemic nature of infection (Doyle-Baker *et al.*, 2020) [40].

4.2 Gross and microscopic lesions

Post-mortem findings are consistent with acute septicemia. Prominent gross lesions include extensive subcutaneous edema in the throat and brisket regions, often with gelatinous, serosanguinous fluid accumulation (Cuevas *et al.*, 2020) [6]. Widespread hemorrhages and vascular congestion are observed in multiple organs. The lungs are typically congested and edematous, with frothy exudate in the airways. Lymph nodes are enlarged, and petechial or ecchymotic hemorrhages may occur on serosal surfaces and internal organs (Shome *et al.*, 2019) [18]. Microscopically, lesions include endothelial damage, vascular leakage, and inflammatory cell infiltration. Pulmonary congestion and edema are prominent, reflecting systemic dissemination of the pathogen (Moustafa *et al.*, 2013; Doyle-Baker *et al.*, 2020) [40].

4.3 Differential diagnosis

Clinical presentation of HS may resemble several infectious diseases, making differential diagnosis essential. Conditions such as Foot-and-Mouth Disease, Anthrax, Blackquarter, and Rinderpest (historically) share features like fever and sudden death. However, these diseases can be distinguished based on characteristic features: vesicular lesions in Foot-and-Mouth Disease, unclotted blood and splenomegaly in Anthrax, emphysematous muscle swelling in Blackquarter, and gastrointestinal erosions in Rinderpest. Definitive diagnosis requires laboratory confirmation, including isolation of *P. multocida* on culture media and molecular detection methods. The organism shows characteristic bipolar staining in tissue smears, aiding preliminary identification (Panna *et al.*, 2015; Varshney *et al.*, 2020; Fegan *et al.*, 2024; OIE, 2008) [46, 47, 45, 48].

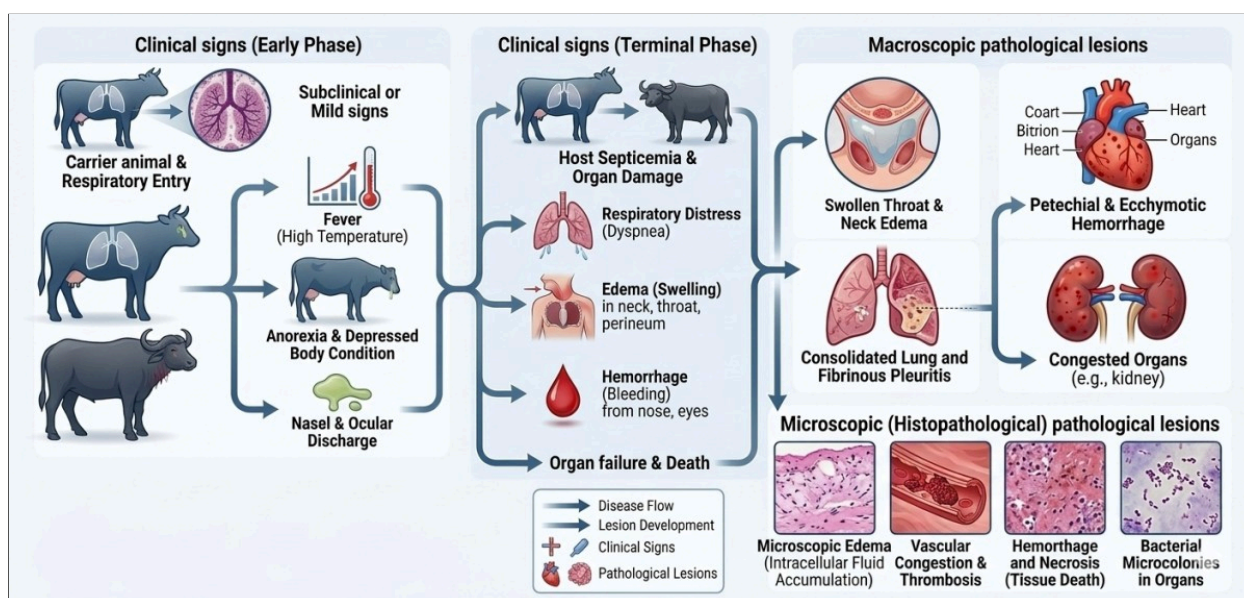


Figure 4. Clinical signs and pathological lesions of hemorrhagic septicemia in cattle and buffalo

5. Epidemiology

Hemorrhagic septicemia (HS) remains a major endemic disease of bovine populations in tropical and subtropical regions, where climatic conditions, host susceptibility, and management practices interact to drive disease dynamics (Lestari *et al.*, 2025) [5]. Despite vaccination efforts, periodic outbreaks continue due to incomplete coverage, environmental stressors, and persistence of carrier animals (Cuevas *et al.*, 2020; Shome *et al.*, 2019) [6, 18].

Epidemiologically, HS is characterized by rapid outbreaks with high mortality, often approaching 100% in susceptible populations. The disease is particularly significant in developing countries, where livestock play a critical role in livelihoods and food security. Multiple studies have identified environmental and management-related risk

factors contributing to outbreak occurrence (Moustafa *et al.*, 2017; Jauregui *et al.*, 2023) [50, 49].

5.1 Global distribution

HS is widely distributed across South and Southeast Asia and parts of Africa, where environmental conditions favor pathogen survival. Endemic countries include India, Pakistan, Bangladesh, Thailand, Indonesia, Malaysia, and others in Asia, as well as several African nations (Verma *et al.*, 1998; Benkirane *et al.*, 2002; Khan *et al.*, 2006; Habib *et al.*, 2019) [51, 15, 52, 53]. Serotype distribution shows clear geographic patterns, with B:2 predominating in Asia and E:2 in Africa (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6]. Outbreaks are frequently seasonal, often peaking during the rainy season due to favorable environmental conditions (Verma *et al.*, 2004; Chanda *et al.*, 2024) [54, 44].



Figure 5. Global distribution and endemic regions of hemorrhagic septicemia in livestock populations

Table 1. Global distribution of hemorrhagic septicemia in livestock

Region	Countries affected	Host species	Predominant serotypes of <i>P. multocida</i>	Outbreak frequency / prevalence	Case fatality rate (%)	References
South Asia	India, Pakistan, Bangladesh, Sri Lanka	Cattle, Buffalo	B:2	Endemic with seasonal outbreaks, particularly during monsoon periods	50–80%	Verma and Jaiswal (1998); Shome <i>et al.</i> (2019)
Southeast Asia	Thailand, Philippines, Indonesia, Malaysia, Cambodia, Laos, Myanmar	Cattle, Buffalo	B:2	Sporadic to frequent outbreaks reported in humid tropical regions	40–70%	Benkirane and De Alwis (2002); Shome <i>et al.</i> (2019)
East Asia	China, Vietnam	Cattle, Buffalo	B:2	Occasional outbreaks with regional endemicity in certain provinces	40–60%	Cuevas <i>et al.</i> (2020)
Africa	Sudan, Egypt, Ethiopia, other Sub-Saharan countries	Cattle, Buffalo	E:2	Endemic occurrence with periodic outbreaks, often associated with climatic stress	50–80%	Moustafa <i>et al.</i> (2013); Benkirane and De Alwis (2002)

5.2 Carrier animals and wildlife reservoirs

Carrier animals are central to HS epidemiology. Clinically healthy cattle and buffalo may harbor *P. multocida* in the upper respiratory tract, acting as reservoirs during inter-epidemic periods (Shivachandra *et al.*, 2011; Cuevas *et al.*, 2020) [28, 6]. Wildlife species also contribute to disease persistence, particularly in ecosystems with shared grazing. These reservoirs complicate control efforts by enabling spillover transmission to domestic livestock (Kutzer *et al.*, 2021; Sting *et al.*, 2020) [55, 56].

5.3 Incidence and prevalence

HS remains a leading cause of mortality in bovine populations in endemic regions. Case fatality rates commonly range from 50% to nearly 100% in untreated animals, particularly in buffalo (Farooq *et al.*, 2011; Shome *et al.*, 2019) [12, 18]. Disease burden is highest in smallholder systems with limited access to veterinary services and vaccination programs. Numerous studies report widespread occurrence across Asia and Africa, reflecting persistent endemicity (Jatoi *et al.*, 2024; Bisht *et al.*, 2006; Khan *et al.*, 2006; Habib *et al.*, 2019) [31, 57, 52, 53].

5.4 Environmental and seasonal factors

Environmental conditions strongly influence HS outbreaks. High humidity, heavy rainfall, and temperature fluctuations promote bacterial survival and transmission, particularly during monsoon seasons

(Abdulkadir *et al.*, 2024) [16]. These conditions often coincide with increased stress due to overcrowding and poor management, enhancing susceptibility and triggering outbreaks (Cuevas *et al.*, 2020) [6].

5.5 Host susceptibility and risk factors

Host susceptibility to Hemorrhagic septicemia (HS) varies according to species, age, and immune status. Buffalo are generally more susceptible than cattle and often experience higher mortality rates during outbreaks (Farooq *et al.*, 2011; Shome *et al.*, 2019) [12, 18]. Additional risk factors include age- and breed-related variability, poor nutritional status and compromised immunity, as well as overcrowding and increased livestock movement. Collectively, these factors enhance transmission dynamics and contribute to increased outbreak severity (Khan, 2012) [58].

5.6 Epidemiological drivers

HS outbreaks result from the interaction of multiple drivers, including carrier animals, environmental conditions, susceptible hosts, and gaps in vaccination coverage. These interacting factors enable persistence of *P. multocida* and periodic outbreak occurrence in endemic regions. Understanding these drivers is essential for designing effective surveillance, vaccination, and biosecurity strategies aimed at reducing disease burden and improving livestock productivity.

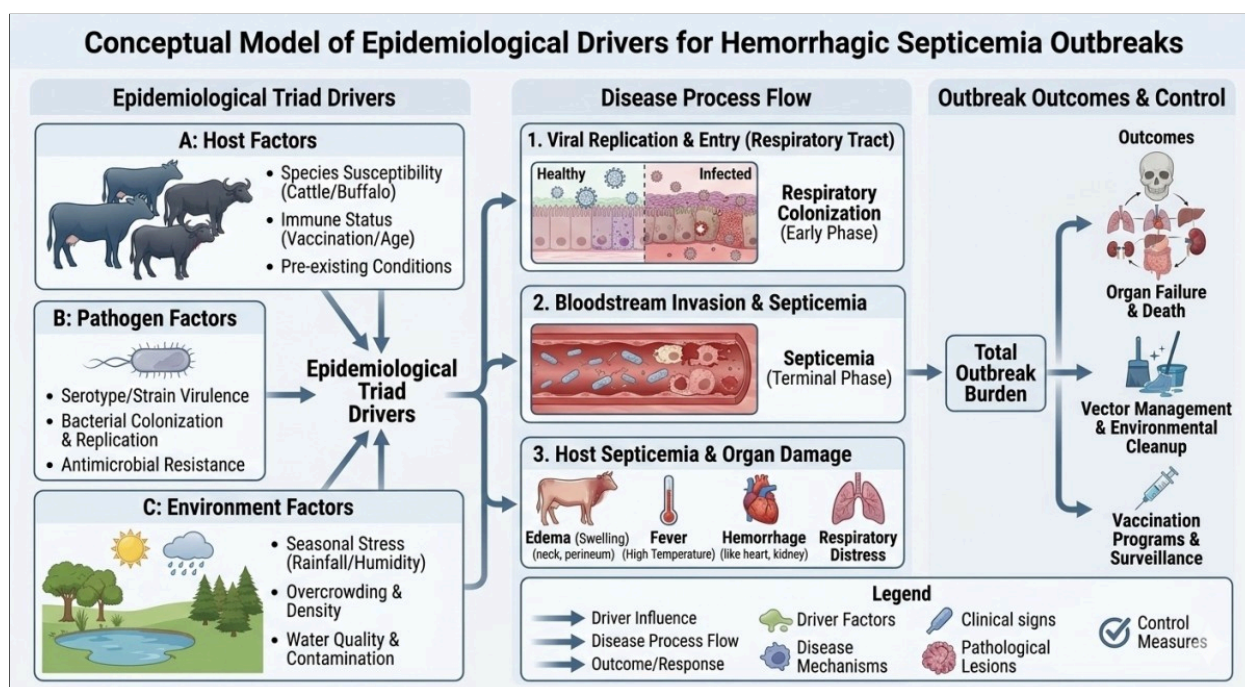


Figure 6. Epidemiological drivers of hemorrhagic septicemia outbreaks

6. Economic impact on livestock production systems

Hemorrhagic Septicemia (HS) is one of the most economically significant bacterial diseases affecting bovine livestock in tropical and sub-tropical regions. The disease causes substantial financial losses through high mortality, reduced productivity, and increased costs associated with treatment, vaccination, and disease control programs (Bardhan *et al.*, 2020; Kawasaki *et al.*, 2015) [59, 60]. These impacts are particularly severe in developing countries, where cattle and buffalo play a critical role in milk production, meat supply, and draft power.

Beyond farm-level losses, HS affects regional livestock economies by disrupting production systems, reducing farmer income, and increasing the financial burden of disease management. In endemic regions, recurrent outbreaks impose sustained economic pressure on livestock sectors and rural livelihoods (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6].

6.1 Direct economic losses

Direct economic losses primarily arise from high mortality and reduced productivity. HS outbreaks are characterized by rapid disease progression, with mortality rates ranging from 50% to nearly 100% in untreated animals, particularly in buffalo (Farooq *et al.*, 2011) [12]. Surviving animals often exhibit reduced feed intake, impaired growth, decreased reproductive performance, and diminished draft capacity, leading to long-term production inefficiencies. Major components of direct losses include mortality of cattle and buffalo, reduced milk production, decreased growth and weight gain, and loss of draft power (Kawasaki *et al.*, 2015) [60].

2. Indirect economic losses

Indirect economic losses result from expenditures associated with disease prevention and control, including antimicrobial treatment, veterinary services, vaccination programs, and surveillance activities (Benkirane *et al.*, 2002) [15]. Additional impacts arise from movement restrictions during outbreaks, which disrupt livestock trade and limit market access. These constraints often delay sales and reduce animal value, further compounding financial losses. Indirect losses include treatment and veterinary costs, vaccination campaigns, surveillance and outbreak response, and trade and movement restrictions (Kawasaki *et al.*, 2015) [60].

6.3 Impact on milk production

Milk production losses represent one of the most significant economic consequences of HS, particularly in dairy-dependent regions (Cuevas *et al.*, 2020) [6]. Infection leads to rapid decline in milk yield due to fever, anorexia, and systemic illness. Even after recovery, prolonged reductions in productivity are common (Shome *et al.*, 2019) [18]. Buffalo populations, which contribute substantially to milk production in South Asia, are particularly vulnerable, amplifying the economic impact at both farm and regional levels.

Table 2. Estimated impact of hemorrhagic septicemia on milk production

Parameter	Estimated effect	Reference
Reduction in daily milk yield	20–40% decline during infection	Shome <i>et al.</i> , 2019
Duration of production loss	2–4 weeks post-infection	Cuevas <i>et al.</i> , 2020
Herd-level milk production decline	Up to 30% reduction during outbreaks	Farooq <i>et al.</i> , 2011

6.4 Impact on meat production

HS adversely affects meat production through mortality and reduced growth performance. Death of animals during outbreaks represents immediate loss to the meat supply chain. Surviving animals often exhibit reduced growth rates and delayed market readiness due to metabolic stress and decreased feed efficiency (Cuevas *et al.*, 2020) [6]. These effects lead to reduced carcass weight and lower economic returns, particularly in smallholder production systems.

Table 3. Estimated impact of hemorrhagic septicemia on meat production

Parameter	Estimated impact	Reference
Mortality rate during outbreaks	50–100% in untreated animals	Farooq <i>et al.</i> , 2011
Reduction in growth rate	10–25% lower weight gain	Cuevas <i>et al.</i> , 2020
Loss of marketable animals	Significant during outbreaks	Shome <i>et al.</i> , 2019

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6.5 Regional economic impact

The economic burden of HS varies across regions but is consistently significant in endemic areas.

India: Experiences substantial losses due to its large bovine population, with HS contributing significantly to mortality and reduced milk production (Shome *et al.*, 2019; Shivachandra *et al.*, 2011; Mondal and Yamage, 2014; De Alwis, 1992) [18, 28, 61].

Pakistan: HS is a major livestock disease, with frequent outbreaks during monsoon seasons causing high mortality in buffalo populations central to the dairy sector (Farooq *et al.*, 2011) [12].

Southeast Asia: Countries such as Thailand, Indonesia, and Malaysia face recurring outbreaks that disproportionately affect smallholder farmers and rural economies (Benkirane *et al.*, 2002) [15].

6.6 Global economic burden

Globally, HS imposes a substantial economic burden due to combined effects of mortality, reduced productivity, and control costs. Although precise global estimates remain limited, the disease is widely recognized as a major constraint to livestock production in tropical regions (Lestari *et al.*, 2025) [5]. The cumulative economic impact is particularly severe in countries with large cattle and buffalo populations,

where outbreaks simultaneously affect milk production, meat supply, and draft power. Persistent endemicity, coupled with recurring outbreaks, results in continuous financial losses and undermines livestock sector sustainability (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6]. However, the absence of standardized global economic assessment models limits accurate estimation of the true burden of HS.

Table 4. Major categories of economic losses associated with hemorrhagic septicemia

Category	Type of loss	Examples
Direct losses	Mortality	Death of cattle and buffalo
	Reduced milk yield	Decline in dairy production
	Reduced growth	Lower body weight gain
Indirect losses	Treatment costs	Veterinary care and antibiotics
	Vaccination programs	Preventive immunization campaigns
	Trade restrictions	Movement and market limitations

Table 5. Table 5 Globally estimated annual economic losses due to hemorrhagic septicemia in major endemic countries

Country / Region	Major affected species	Estimated annual economic loss (USD)	Major contributing factors	References
India	Cattle, buffalo	\$40–70 million	High bovine population, seasonal outbreaks, milk production losses	Shome <i>et al.</i> , 2019; Cuevas <i>et al.</i> , 2020
Pakistan	Buffalo, cattle	\$15–25 million	High buffalo mortality, reduced dairy productivity	Farooq <i>et al.</i> , 2011
Bangladesh	Cattle, buffalo	\$8–12 million	Frequent outbreaks, limited vaccination coverage	Benkirane and De Alwis, 2002
Thailand	Cattle, buffalo	\$5–10 million	Periodic outbreaks, reduced growth and meat production	Benkirane and De Alwis, 2002
Indonesia	Cattle, buffalo	\$6–12 million	Regional outbreaks affecting dairy and meat production	Cuevas <i>et al.</i> , 2020
Malaysia	Cattle, buffalo	\$3–6 million	Control costs and vaccination campaigns	Benkirane and De Alwis, 2002
African countries (Sudan, Ethiopia, Egypt)	Cattle	\$10–20 million	High mortality, limited veterinary infrastructure	Moustafa <i>et al.</i> , 2013

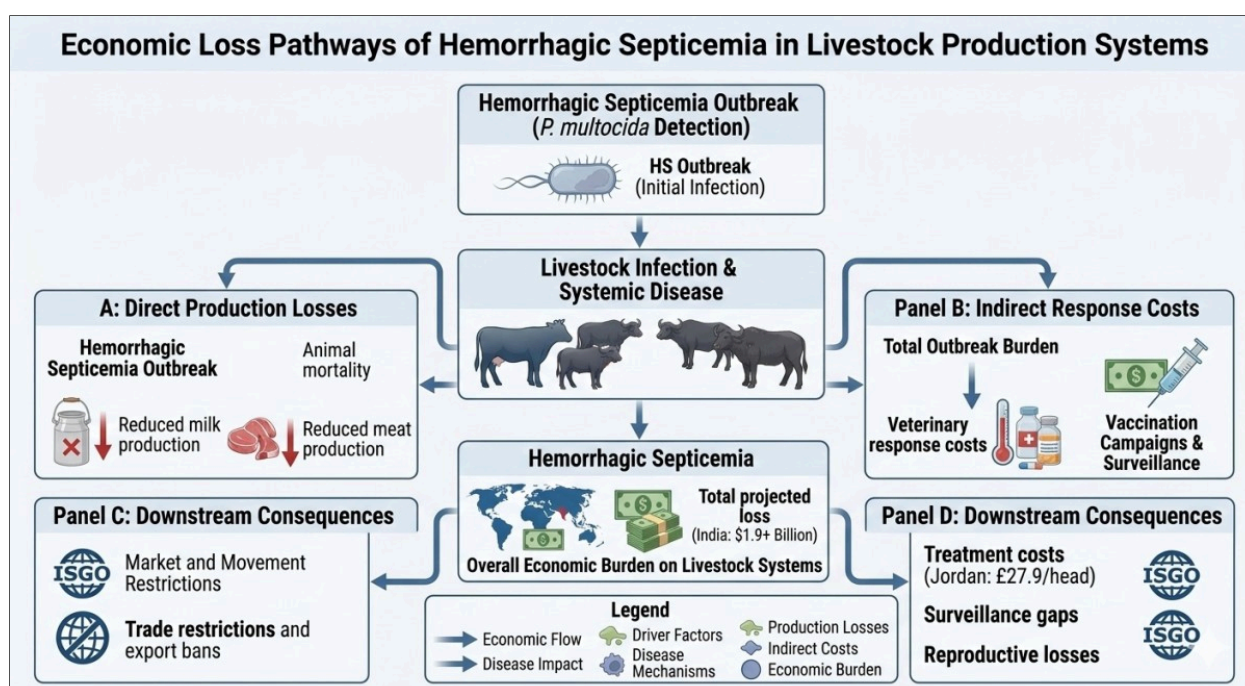


Figure 7. Economic loss pathways of hemorrhagic septicemia in livestock production systems

7. Diagnostic advances

Accurate and rapid diagnosis of Hemorrhagic Septicemia (HS) is critical for effective disease control and outbreak management. Early detection of *Pasteurella multocida* enables timely therapeutic and preventive interventions (Maddock *et al.*, 2025). Traditionally, diagnosis relied on clinical signs, post-mortem findings, and bacteriological isolation; however, advances in molecular and genomic technologies have significantly enhanced diagnostic sensitivity, specificity, and speed (Calderon Bernal *et al.*, 2022; Cuevas *et al.*, 2020; Harper *et al.*, 2006) [62, 6, 8]. Modern diagnostic strategies integrate conventional bacteriology with molecular and genomic tools to improve disease confirmation and epidemiological surveillance. In addition, molecular characterization studies have revealed considerable diversity in virulence factors and antimicrobial resistance profiles among *P. multocida* isolates (Ali *et al.*, 2025; Sunartatie *et al.*, 2024; Bhardwaj *et al.*, 2025) [63, 64, 30].

7.1 Conventional diagnostic methods

Microscopy Microscopic examination of stained smears from blood or tissues provides rapid preliminary diagnosis. *P. multocida* exhibits a characteristic bipolar (“safety-pin”) appearance when stained with Giemsa or methylene blue. However, due to limited sensitivity and specificity, microscopy is suitable only for presumptive diagnosis (Harper *et al.*, 2006) [8].

Bacterial Culture Isolation on media such as blood agar remains a standard confirmatory method. The organism forms smooth, grayish colonies, followed by biochemical and serotyping identification (OIE, 2008) [48]. Despite its reliability, culture is time-consuming and dependent on viable organisms, with reduced success following antibiotic treatment (Cuevas *et al.*, 2020) [6].

2. Molecular diagnostics

Molecular diagnostic techniques have significantly improved the detection of *P. multocida* by offering rapid and highly sensitive identification. PCR-based assays targeting species-specific genes enable detection even at low bacterial loads (Cuevas *et al.*, 2020) [6]. Advanced applications include detection of virulence-associated genes, capsular typing for strain differentiation, and epidemiological tracing of outbreaks. Multiplex PCR enhances diagnostic efficiency by enabling simultaneous detection of multiple targets. In addition, techniques such as REP-PCR support strain differentiation (Townsend *et al.*, 1997) [65], while loop-mediated isothermal amplification (LAMP) provides rapid field-level detection (Bhimani *et al.*, 2015) [66]. Standard diag-

nistic protocols recommended by OIE (2008) [48] integrate bacteriological and PCR-based methods.

7.3 Serological methods

Serological assays, including ELISA and indirect hemagglutination tests, are widely used for herd-level surveillance and epidemiological studies (Shome *et al.*, 2019; Singhla *et al.*, 2020) [18, 67]. These methods are valuable for assessing exposure and immune response but cannot reliably distinguish between vaccinated and naturally infected animals. Therefore, they are typically used alongside bacteriological and molecular diagnostics.

4. Emerging genomic tools

Genomic technologies have substantially advanced understanding of HS epidemiology and pathogen biology. Whole-genome sequencing (WGS) and comparative genomics enable detailed characterization of genetic diversity, virulence determinants, and evolutionary relationships among *P. multocida* strains (Narcana *et al.*, 2020; Hanchanachai *et al.*, 2021) [69, 68]. These approaches facilitate identification of novel diagnostic markers, support rational vaccine design, and enable high-resolution outbreak tracing. Recent studies have further highlighted genetic variability, antimicrobial resistance determinants, and host–pathogen interactions (Desem *et al.*, 2023 [39]; Abed *et al.*, 2020 [34]; Tabatabaei and Abdolahi, 2023 [70]; Jia *et al.*, 2023 [71]; Piorunek *et al.*, 2023) [13]. Although currently applied mainly in research settings, genomic tools are expected to become increasingly integrated into routine diagnostics as sequencing technologies become more accessible (Cao *et al.*, 2024; Cuevas *et al.*, 2020) [72, 6].

7.5 Integrated diagnostic approaches

Given the limitations of individual methods, integrated diagnostic approaches are essential for accurate HS detection. Clinical and pathological findings provide initial indications, while laboratory confirmation is achieved through culture, molecular assays, and genomic tools (Desem *et al.*, 2023) [39]. Such integration enhances diagnostic accuracy, supports epidemiological surveillance, and enables rapid outbreak response—particularly in endemic regions where early detection is critical for minimizing economic losses (Harper *et al.*, 2006; Cuevas *et al.*, 2020) [8, 6].

Recent advances, including quantitative PCR, genomic epidemiology, and detection of co-infections, have further strengthened diagnostic capabilities and improved understanding of disease dynamics (Pan *et al.*, 2025; Amirgazin *et al.*, 2025; Zhao *et al.*, 2026; Rahman *et al.*, 2024) [74, 73, 75, 76].

Table 6. Comparison of diagnostic approaches used for hemorrhagic septicemia detection

Diagnostic method	Principle	Advantages	Limitations	Typical use	Key references
Microscopy	Identification of bipolar-staining bacteria in stained smears	Rapid, inexpensive, simple	Low specificity and sensitivity	Preliminary diagnosis during outbreaks	Harper <i>et al.</i> , 2006
Bacterial culture	Isolation of pathogen on blood agar or selective media	Gold standard confirmation; allows antimicrobial testing	Time-consuming; requires viable samples	Laboratory confirmation	Cuevas <i>et al.</i> , 2020
PCR (Polymerase Chain Reaction)	Amplification of pathogen-specific DNA sequences	High sensitivity and specificity; rapid detection	Requires specialized equipment and trained personnel	Early detection and molecular epidemiology	Cuevas <i>et al.</i> , 2020
Virulence gene detection	Identification of genes associated with pathogenicity	Useful for strain characterization and epidemiological studies	Higher technical requirements	Research and advanced diagnostics	Harper <i>et al.</i> , 2006; Abed <i>et al.</i> , 2020
Serological tests (ELISA, IHA)	Detection of antibodies against pathogen antigens	Suitable for herd-level surveillance and monitoring immunity	Cannot always differentiate infection from vaccination	Epidemiological studies and surveillance	Shome <i>et al.</i> , 2019

8. Treatment dynamics and antimicrobial resistance

Effective treatment of HS depends on early diagnosis and timely therapeutic intervention. Antimicrobial therapy remains the primary treatment strategy, often combined with supportive care such as *anti-inflammatory* drugs and fluid therapy to improve survival (Farooq *et al.*, 2011; Shome *et al.*, 2019) [12, 18]. However, increasing antimicrobial resistance (AMR) in *P. multocida* isolates has emerged as a major challenge, potentially compromising treatment efficacy (Ali *et al.*, 2025; Sunartatie *et al.*, 2024; Tabatabaei *et al.*, 2023; Cuevas *et al.*, 2020) [63, 64, 70, 6].

8.1 Antibiotic therapy

Commonly used antibiotics include penicillin, tetracyclines, sulfonamides, cephalosporins, and fluoroquinolones (Kerek *et al.*, 2024) [77]. Penicillin and tetracycline derivatives have historically been effective against HS (Farooq *et al.*, 2011) [12]. While many isolates remain susceptible, resistance to sulfonamides and cotrimoxazole has been reported (Petrocchi-Rilo *et al.*, 2018; Ferreira *et al.*, 2012) [78, 36]. Treatment success depends on early administration, as advanced cases often progress rapidly to septicemia, limiting therapeutic effectiveness.

2. Antimicrobial resistance patterns

Emerging resistance to commonly used antibiotics, including tetracyclines and β -lactams, has been reported in *P. multocida* isolates (Sunartatie *et al.*, 2024; Michael *et al.*, 2021; Shome *et al.*, 2019) [64, 17, 18]. Resistance development is frequently associated with indiscriminate antibiotic use, prophylactic administration, and incomplete treatment regimens. Therefore, antimicrobial susceptibility testing is essential for guiding appropriate therapeutic decisions and improving treatment outcomes (Pint r *et al.*, 2024; Michael *et al.*, 2021; Shome *et al.*, 2019) [79, 17, 18]. The lack of standardized global surveillance

frameworks for antimicrobial resistance in *P. multocida* remains a critical limitation in developing effective control strategies.

3. Multidrug resistance

The emergence of multidrug-resistant (MDR) *P. multocida* strains represents a growing challenge to effective disease management (Cuevas *et al.*, 2020; Abdulkadir *et al.*, 2024; Sabsabi *et al.*, 2021) [6, 16, 80]. This trend underscores the urgent need for antimicrobial stewardship programs, strengthened surveillance systems, and reduced reliance on antibiotics in livestock production. Recent evidence indicates increasing resistance trends across livestock populations, highlighting the importance of implementing responsible antimicrobial use strategies (Sunartatie *et al.*, 2024; Ali *et al.*, 2025; Bitew *et al.*, 2025) [64, 63, 81].

9. Implications of antimicrobial resistance for disease control

The rise of antimicrobial resistance significantly complicates HS management by reducing the effectiveness of commonly used antibiotics (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6]. The presence of multidrug-resistant strains further limits therapeutic options and increases the risk of treatment failure. Modern diagnostic tools play a crucial role in addressing this challenge by enabling detection of resistance-associated genes and supporting targeted therapy (Michael *et al.*, 2021) [17]. Importantly, the growing limitations of antimicrobial therapy highlight the need for preventive strategies. Vaccination, improved herd management, and strengthened biosecurity measures are essential for reducing disease incidence and minimizing reliance on antibiotics. Thus, sustainable HS control increasingly depends on integrated approaches combining diagnostics, vaccination, and responsible antimicrobial use (Michael *et al.*, 2021) [17].

Table 7. Antibiotics used for hemorrhagic septicemia treatment and reported resistance patterns

Antibiotic	Drug class	Typical use	Reported resistance trends	References
Penicillin	β -lactam	Traditional treatment for HS	Generally effective but emerging resistance reported	Farooq <i>et al.</i> , 2011
Tetracycline / Oxytetracycline	Tetracycline class	Widely used in livestock	Moderate resistance reported in some isolates	Shome <i>et al.</i> , 2019
Enrofloxacin	Fluoroquinolone	Treatment of severe infections	Resistance relatively low but increasing in some regions	Cuevas <i>et al.</i> , 2020
Ceftiofur	Third-generation cephalosporin	Advanced therapy for Gram-negative infections	Limited resistance reported	Cuevas <i>et al.</i> , 2020
Florfenicol	Phenicol antibiotic	Respiratory and systemic infections	Effective but resistance emerging in some isolates	Shome <i>et al.</i> , 2019

10. Vaccination strategies

Among available control measures, vaccination continues to be the primary and most widely implemented strategy for the prevention and control of Hemorrhagic Septicemia (HS), particularly in endemic regions where *Pasteurella multocida* serotypes B:2 and E:2 are prevalent (Shivachandra *et al.*, 2011) [28]. Due to the rapid progression of the disease and limited effectiveness of treatment in advanced stages, prophylactic immunization is essential for reducing mortality and outbreak frequency. A range of vaccine formulations has been developed, including conventional bacterins, live attenuated vaccines, and emerging recombinant and DNA-based vaccines. These approaches differ in immunogenicity, duration of protection, safety, and field applicability. Despite advances in next-generation vaccines, conventional oil-adjuvant bacterin vaccines remain the most widely used due to their affordability and suitability for large-scale immunization programs (Hodgson *et al.*, 2005 [82]; Kumar *et al.*, 2015 [83]; Yuan *et al.*, 2024; He *et al.*, 2024 [84]; Tesfaye *et al.*, 2025 [85]; Xu *et al.*, 2025) [86].

Vaccination programs typically involve primary immunization at 4–6 months of age, followed by booster doses and annual revaccination to maintain herd immunity (Muenthaisong *et al.*, 2020) [87].

10.1 Conventional bacterin vaccines

Inactivated bacterin vaccines, including oil-adjuvant (OAV) and alum-precipitated (APV) formulations, have been widely used for decades in endemic regions (Afroz *et al.*, 2022) [88]. OAV formulations are generally preferred due to their ability to induce stronger and more sustained humoral immune responses (Rustagi *et al.*, 2023; Zamri-Saad *et al.*, 2021) [89]. However, bacterin vaccines are associated with limitations, including relatively short duration of immunity (particularly for APV), limited cross-protection among strains, and the need for repeated booster doses. Vaccine efficacy may also vary depending on strain diversity, storage conditions, and field practices. Despite these constraints, bacterin vaccines remain central to HS control programs due to their cost-effectiveness and field applicability (Jia *et al.*, 2023) [71].

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2. Live attenuated vaccines

Live attenuated vaccines are capable of inducing both humoral and cell-mediated immune responses, potentially providing stronger and longer-lasting protection compared with inactivated vaccines (Afroz *et al.*, 2022; He *et al.*, 2024) [88, 84]. However, their practical application remains limited due to concerns regarding reversion to virulence, biosafety risks, and regulatory constraints.

10.3 Subunit vaccines

Subunit vaccines utilize specific antigenic components such as outer membrane proteins and capsular antigens to induce targeted immune responses. These vaccines offer improved safety and specificity but often require potent adjuvants to achieve adequate immunogenicity (Facciola *et al.*, 2022; Mostaan *et al.*, 2020; Cuevas *et al.*, 2020) [90, 32, 6].

10.4 Recombinant vaccines

Recombinant vaccines expressing defined protective antigens, such as outer membrane proteins (e.g., Omp16), have demonstrated promising immunogenicity in experimental studies (Shivachandra *et al.*, 2017) [91]. These vaccines offer advantages including a high safety profile, targeted immune responses, and scalability. Nevertheless, their use remains largely experimental, and further field validation is required prior to widespread adoption.

10.5 DNA vaccines

DNA vaccines represent an emerging strategy capable of inducing both humoral and cellular immunity. These vaccines offer advantages such as stability and multivalent antigen expression but face challenges related to delivery efficiency, regulatory approval, and field applicability (Cuevas *et al.*, 2020) [6].

Table 8. Comparison of available vaccines for hemorrhagic septicemia in livestock

Vaccine type	Principle / Composition	Advantages	Limitations	Duration of immunity	References
Bacterin (inactivated vaccine)	Formalin-killed <i>P. multocida</i> cells, often formulated with oil adjuvants	Safe, widely used, relatively inexpensive, easy to produce	Short-term immunity; may require booster doses; limited cross-protection	4–6 months	Facciola <i>et al.</i> , 2022; Benkirane and De Alwis, 2002; Shome <i>et al.</i> , 2019
Bacterin (APV)	Inactivated whole cells with alum adjuvant	Safe, widely used	Short duration of immunity	3–4 months	Shivachandra <i>et al.</i> , 2011
Bacterin (OAV)	Inactivated cells with oil adjuvant	Strong antibody response	Injection site reactions possible	6–12 months	OIE, 2008
Live attenuated vaccine	Weakened strains of <i>P. multocida</i> capable of limited replication in the host	Induces strong immune response and longer protection compared with bacterins	Risk of reversion to virulence; stability concerns; strict storage conditions	Up to 1 year	Harper <i>et al.</i> , 2006
Subunit vaccine	Purified antigenic components such as outer membrane proteins (e.g., OmpH, PlpE)	Highly specific immune response; improved safety profile	Higher production cost; may require adjuvants	Variable, depends on formulation	Cuevas <i>et al.</i> , 2020; Mostaan <i>et al.</i> , 2020
Recombinant vaccine	Genetically engineered antigens expressed in bacterial or viral vectors	Targeted immune response; potential cross-protection	Still under development; limited field application	Under investigation	Harper <i>et al.</i> , 2006; Cuevas <i>et al.</i> , 2020
DNA vaccine	Plasmid DNA encoding specific antigenic proteins of <i>P. multocida</i>	Stimulates both humoral and cellular immunity; promising future approach	Experimental stage; regulatory and delivery challenges	Experimental	Cuevas <i>et al.</i> , 2020

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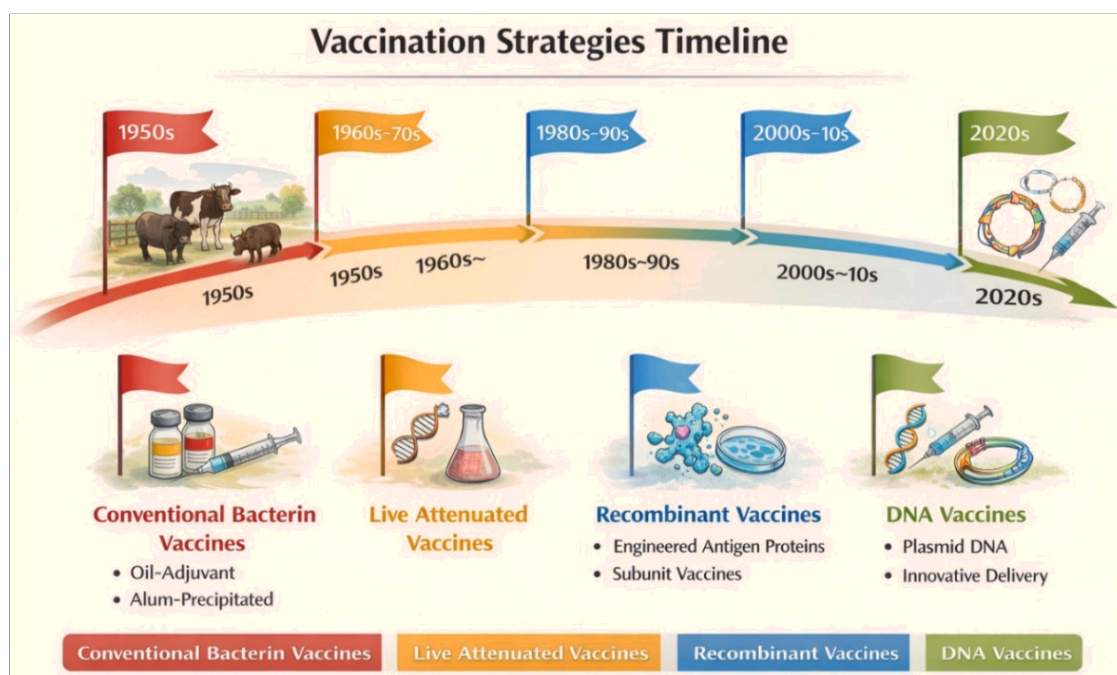


Figure 8. Vaccination strategies timeline

11. Prevention and control strategies

Effective control of Hemorrhagic septicemia (HS) requires an integrated approach combining vaccination, biosecurity, surveillance, and movement control. Preventive strategies are essential, as therapeutic interventions are often ineffective once septicemia develops (Shome *et al.*, 2019) [18]. Coordinated control programs demonstrate that the integration of vaccination with surveillance and movement restrictions significantly reduces outbreak frequency and overall disease impact, particularly in endemic regions (Farooq *et al.*, 2011; Shome *et al.*, 2019) [12, 18].

Vaccination programs

Routine vaccination remains the cornerstone of HS prevention in endemic regions. Immunization is typically implemented prior to the rainy season, when environmental conditions favor pathogen survival and transmission (Shivachandra *et al.*, 2011) [28]. Sustained herd immunity depends on achieving high vaccination coverage, maintaining cold-chain integrity, and ensuring timely administration of booster doses (Shome *et al.*, 2019) [18].

However, the field efficacy of currently available vaccines may vary due to strain diversity, improper storage, and inconsistent vaccination practices, highlighting the need for improved vaccine formulations and delivery systems.

2. Biosecurity measures

Biosecurity plays a critical role in preventing the introduction and transmission of HS within livestock populations. Key measures include maintaining hygienic housing conditions, ensuring access to clean and uncontaminated feed and water, and minimizing animal overcrowding. In addition, effective stress management—such as reducing transportation stress and mitigating adverse environmental conditions—is essential for maintaining immune competence and reducing susceptibility to infection (Farooq *et al.*, 2011; Shome *et al.*, 2019) [12, 18].

11.3 Quarantine and movement control

Quarantine of newly introduced animals and restriction of animal movement during outbreaks are essential for limiting disease spread. Monitoring livestock markets and implementing emergency vaccination in affected areas further enhance outbreak containment and reduce transmission risk (Farooq *et al.*, 2011) [12].

11.4 Surveillance and monitoring

Robust surveillance systems are critical for early detection and rapid response to HS outbreaks. The integration of molecular diagnostics and genomic tools enhances disease tracking, facilitates identification of virulent strains, and supports evidence-based control strategies (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6].

11.5 Climate change and HS epidemiology

Environmental factors such as humidity, rainfall, and temperature play a significant role in the occurrence and spread of HS. Climate change may alter these parameters, potentially expanding the geographical distribution of the disease and modifying outbreak patterns. Understanding these dynamics is essential for developing predictive models and adaptive control strategies (Shome *et al.*, 2019; Cuevas *et al.*, 2020) [18, 6].

11.6 One Health approach

The One Health framework integrates animal health, environmental monitoring, and disease surveillance to enhance HS control. Coordinated implementation of these components improves early detection, strengthens outbreak response, and supports sustainable disease management. Recent studies emphasize the importance of integrating molecular epidemiology, antimicrobial resistance monitoring, and ecological factors within a One Health framework to address the multifaceted challenges associated with HS (Richardson *et al.*, 2023 [29]; Tabatabaei and Abdolahi, 2023 [70]; Jia *et al.*, 2023 [71]; Desem *et al.*, 2023 [39]; Piorunek *et al.*, 2023) [13].

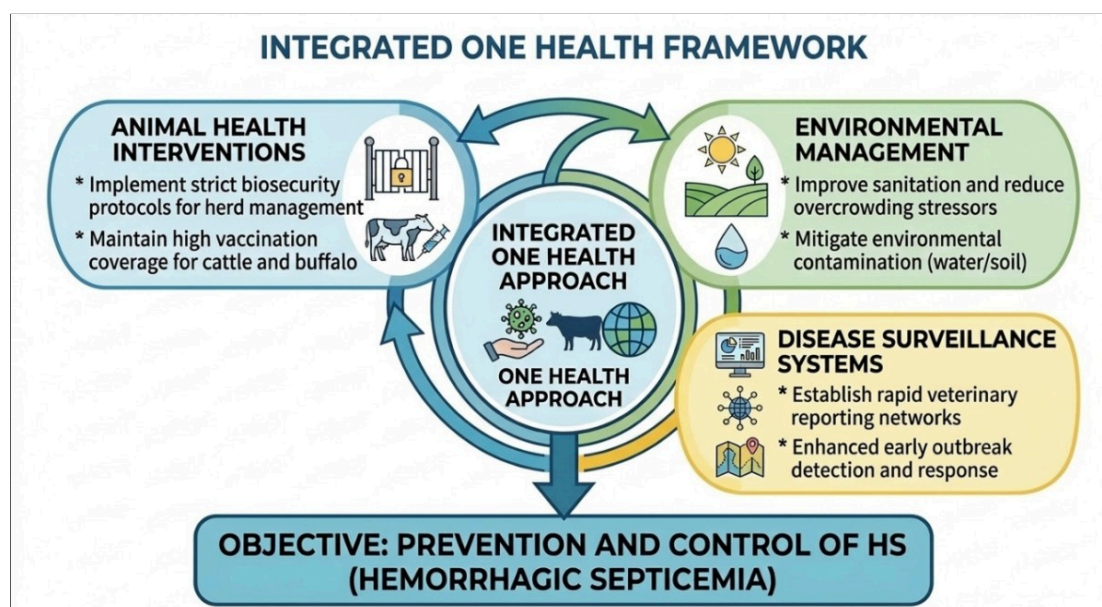


Figure 9. Integrated One Health–economic framework for hemorrhagic septicemia control

12. Future perspectives and research needs

Despite significant advances in HS research, several challenges remain, including pathogen diversity, limitations in vaccine efficacy, and the emergence of antimicrobial resistance. Addressing these challenges requires the integration of advanced molecular tools with conventional disease control strategies (Shivachandra *et al.*, 2011; Shome *et al.*, 2019; Narcana *et al.*, 2020; Hanchanachai *et al.*, 2021) [28, 18, 69, 68].

Future research should prioritize the development of next-generation vaccines that provide broad and long-lasting protection using recombinant and nucleic acid-based technologies. In addition, molecular epidemiological studies should be expanded to improve understanding of pathogen evolution, transmission dynamics, and regional strain diversity (Cuevas *et al.*, 2020; Verma *et al.*, 2013; Ferreira *et al.*, 2015; Zhao *et al.*, 2021; Tawab *et al.*, 2020) [6, 37, 9, 26, 38].

Strengthening surveillance systems through improved diagnostic infrastructure and the implementation of digital reporting platforms will enhance early outbreak detection and response (Shome *et al.*, 2019) [18]. Furthermore, antimicrobial stewardship programs promoting rational antibiotic use, in combination with vaccination strategies, are essential to reduce antimicrobial dependency and limit resistance development (Cuevas *et al.*, 2020) [6].

Recent advances in genomics and diagnostic technologies continue to improve understanding of pathogen evolution, virulence mechanisms, and disease emergence patterns (Rahman *et al.*, 2024; Pan *et al.*, 2025; Amirgazin *et al.*, 2025; Zhao *et al.*, 2026) [76, 74, 73, 75].

13. Discussion

Despite considerable progress in understanding and managing Hemorrhagic septicemia (HS), several challenges continue to limit effective disease control. Current vaccines, although effective under controlled conditions, often exhibit variable efficacy in field situations due to strain variation, improper storage, and inadequate vaccination coverage.

Advanced diagnostic techniques such as PCR and LAMP provide high sensitivity and specificity; however, their widespread application in rural and resource-limited settings remains constrained by cost, infrastructure, and technical expertise. This limitation highlights the need for affordable and field-deployable diagnostic tools. Furthermore, the emergence of antimicrobial resistance in *Pasteurella multocida* complicates treatment strategies. In addition, inconsistencies in surveillance systems and underreporting hinder accurate assessment of disease burden, particularly in developing countries. These limitations directly affect milk yield and meat production,

thereby influencing the economic sustainability of livestock production systems. Future research should prioritize the development of thermostable and broad-spectrum vaccines, improved molecular epidemiological tools, and cost-effective diagnostic platforms. Strengthening surveillance systems, enhancing farmer awareness, and implementing integrated disease management strategies will be critical for sustainable control of HS in endemic regions.

14. Conclusion

Hemorrhagic septicemia (HS) remains a major bacterial disease of cattle and buffalo in tropical and subtropical regions, characterized by rapid disease progression, high mortality, and substantial economic losses. Its impact extends beyond animal health, significantly affecting rural livelihoods, food security, and livestock-based economies. Despite the availability of vaccines and control measures, HS continues to persist in endemic regions due to limitations in vaccine efficacy, gaps in surveillance, and the emergence of antimicrobial resistance. Environmental and climatic factors further influence disease dynamics, increasing the complexity of control efforts. Effective long-term management of HS requires an integrated approach combining vaccination, improved diagnostics, strengthened surveillance systems, and responsible antimicrobial use. In this context, the adoption of a One Health framework—linking animal health, environmental factors, and epidemiological monitoring—will be essential for sustainable disease control. Future strategies should prioritize the development of next-generation vaccines, expansion of genomic surveillance, and implementation of evidence-based disease management practices. Addressing these gaps through coordinated global strategies and strengthened surveillance frameworks will be essential to reduce the burden of HS and support sustainable milk and meat production systems.

References

1. Peng Z, Wang X, Zhou R, Chen H, Wilson BA, Wu B. *Pasteurella multocida*: genotypes and genomics. *Microbiology and Molecular Biology Reviews*. 2019;83(4):10-1128.
2. Wilson BA, Ho M. *Pasteurella multocida*: from zoonosis to cellular microbiology. *Clinical Microbiology Reviews*. 2013;26(3):631-655.
3. Zamri-Saad M, Annas S. Vaccination against Hemorrhagic Septicemia of Bovines: A Review. *Pakistan Veterinary Journal*. 2016;36(1):1-5.
4. Abusalab SM, Ahmed AM, Hamid ME. Haemorrhagic septicaemia: a general review. *Sudan J Vet Res*. 2003;18:1-14.

5. Lestari TD, Khairullah AR, Damayanti R, Mulyati S, Rimayanti R, Hernawati T, Kusala MKJ. Hemorrhagic septicemia: A major threat to livestock health. *Open Veterinary Journal*. 2025;15(2):519.
6. Cuevas I, Carbonero A, Cano D, Pacheco IL, Marín JC, Borge C. First outbreak of bovine haemorrhagic septicaemia caused by *Pasteurella multocida* type B in Spain—Short communication. *Acta Veterinaria Hungarica*. 2020;68(1):8-11.
7. Almoheer R, Abd Wahid ME, Zakaria HA, Jonet MAB, Al-Shaibani MM, Al-Gheethi A, Addis SNK. Spatial, temporal, and demographic patterns in the prevalence of hemorrhagic septicemia in 41 countries in 2005–2019: A systematic analysis with special focus on the potential development of a new-generation vaccine. *Vaccines*. 2022;10(2):315.
8. Harper M, Boyce JD, Adler B. *Pasteurella multocida* pathogenesis: 125 years after Pasteur. *FEMS Microbiology Letters*. 2006;265(1):1–10.
9. Ferreira TS, Felizardo MR, de Gobbi DD, Moreno M, Moreno AM. Antimicrobial resistance and virulence gene profiles in *Pasteurella multocida* strains isolated from cats. *Brazilian Journal of Microbiology*. 2015;46(1):271–277.
10. Abera D, Mossie T. A review on pneumonic pasteurellosis in small ruminants. *Journal of Applied Animal Research*. 2022;51(1):1-10.
11. Lin H, Liu Z, Zhou Y, Lu W, Xu Q. Characterization of resistance and virulence of *Pasteurella multocida* isolated from pet cats in South China. *Antibiotics*. 2022;11(10):1387.
12. Farooq U, Saeed Z, Khan MA, Iftikhar A, Qamar MF. Sero-surveillance of hemorrhagic septicemia in buffaloes and cattle in Southern Punjab, Pakistan. *Pakistan Veterinary Journal*. 2011;31(3):253–255.
13. Piorunek M, Brajer-Luftmann B, Walkowiak J. *Pasteurella multocida* infection in humans. *Pathogens*. 2023;12(10):1210.
14. Shome R, Kanani A, Gurrappanaidu G, Subbanna NKG, Mohandoss N, Prajapati A, Habibur R. Sero-prevalence of hemorrhagic septicemia in cattle and buffalo population of Indian states *Karnataka* and *Gujarat*. *Veterinary Sciences*. 2024;11(8):386.
15. Benkirane A, De Alwis MCL. Haemorrhagic septicaemia, its significance, prevention and control in Asia. *VETERINARNI MEDICINA-PRAHA*. 2002;47(8):234-240.
16. Abdulkadir M, Nigussie T, Kebede IA. Isolation and identification of *Pasteurella multocida* and *Mannheimia haemolytica* from pneumonic small ruminants and their antibiotic susceptibility in Haramaya District, Eastern Ethiopia. *The Scientific World Journal*. 2024;2024(1):5605552.
17. Michael FS, Cairns CM, Fleming P, Vinogradov EV, Boyce JD, Harper M, Cox AD. The capsular polysaccharides of *Pasteurella multocida* serotypes B and E: Structural, genetic and serological comparisons. *Glycobiology*. 2021;31(3):307-314.
18. Shome R, Deka RP, Sahay S, Grace D, Lindahl JF. Seroprevalence of hemorrhagic septicemia in dairy cows in Assam, India. *Infection Ecology & Epidemiology*. 2019;9(1):1604064.
19. Sajid SM, Yousaf A, Irshad H, Ahsan A, Nawaz M, Waqas M, Shahzad MA. Hemorrhagic Septicemia: Etiology, Pathogenesis and Vaccine Strategies. *International Journal of Agriculture and Biology*. 2024;32:341–354.
20. Duan R, Lyu D, Qin S, Liang J, Gu W, Duan Q, Wang X. *Pasteurella multocida* strains of a novel capsular serotype and lethal to *Marmota himalayana* on Qinghai-Tibet plateau in China. *International Journal of Medical Microbiology*. 2024;314:151597.
21. Harper M, Boyce JD. The myriad properties of *Pasteurella multocida* lipopolysaccharide. *Toxins*. 2017;9(8):254.
22. Bosch M, Garrido ME, De Rozas AMP, Badiola I, Barbé J, Llagostera M. *Pasteurella multocida* contains multiple immunogenic haemin-and haemoglobin-binding proteins. *Veterinary Microbiology*. 2004;99(2):103-112.
23. May BJ, Zhang Q, Li LL, Paustian ML, Whittam TS, Kapur V. Complete genomic sequence of *Pasteurella multocida*, Pm70. *Proceedings of the National Academy of Sciences*. 2001;98(6):3460-3465.
24. Mahboob S, Ullah N, Farhan Ul Haque M, Rauf W, Iqbal M, Ali A, Rahman M. Genomic characterization and comparative genomic analysis of HS-associated *Pasteurella multocida* serotype B: 2 strains from Pakistan. *BMC Genomics*. 2023;24(1):546.
25. El-Demerdash AS, Mowafy RE, Fahmy HA, Matter AA, Samir M. Pathognomonic features of *Pasteurella multocida* isolates among various avian species in Sharkia Governorate, Egypt. *World Journal of Microbiology and Biotechnology*. 2023;39(12):335.
26. Zhao X, Shen H, Liang S, Zhu D, Wang M, Jia R, Cheng A. The lipopolysaccharide outer core transferase genes *pcgD* and *hptE* contribute differently to the virulence of *Pasteurella multocida* in ducks. *Veterinary Research*. 2021;52(1):37.
27. Vu-Khac H, Trinh TH, Nguyen TG, Nguyen XT, Nguyen TT. Prevalence of virulence factor, antibiotic resistance, and serotype genes of *Pasteurella multocida* strains isolated from pigs in Vietnam. *Veterinary World*. 2020;13(5):896.
28. Shivachandra SB, Viswas KN, Kumar AA. A review of hemorrhagic septicemia in cattle and buffalo. *Animal Health Research Reviews*. 2011;12(1):67-82.
29. Richardson NI, Ravenscroft N, Kuttel MM. Conformational comparisons of *Pasteurella multocida* types B and E and structurally related capsular polysaccharides. *Glycobiology*. 2023;33(9):745-754.
30. Bhardwaj M, Verma S, Barsain S, Dhar P, Chahota R. Isolation and detection of *Pasteurella multocida* from clinical hemorrhagic septicemia cases of buffaloes. *Indian Journal of Veterinary Pathology*. 2025;49(3):215-218.
31. Jatoi BA, Mirani AH, Bhutto AL, Laghari A, Magsi AS, Jatoi AS, Malak AA. Prevalence of hemorrhagic septicemia and use of *Moringa oleifera* and *Eucalyptus camaldulensis* extracts against buffalo *Pasteurella multocida* isolates. *Journal of Animal Health and Production*. 2024;12(3):380-386.
32. Mostaan S, Ghasemzadeh A, Sardari S, Shokrgozar MA, Brujeni GN, Abolhassani M, Karam MRA. *Pasteurella multocida* vaccine candidates: A systematic review. *Avicenna Journal of Medical Biotechnology*. 2020;12(3):140.
33. Orynbayev M, Sultankulova K, Sansyzbay A, Rystayeva R, Shorayeva K, Namet A, Kock R. Biological characterization of *Pasteurella multocida* present in the Saiga population. *BMC Microbiology*. 2019;19(1):37.
34. Abed AH, El-Seedy FR, Hassan HM, Nabih AM, Khalifa E, Salem SE, Menshawy AM. Serotyping, genotyping and virulence genes characterization of *Pasteurella multocida* and *Mannheimia haemolytica* isolates recovered from pneumonic cattle calves in North Upper Egypt. *Veterinary Sciences*. 2020;7(4):174.
35. Harper M, Boyce JD, Adler B. The key surface components of *Pasteurella multocida*: Capsule and lipopolysaccharide. In B. Adler (Ed.), *Pasteurella multocida*: Molecular biology, toxins and infection (Current Topics in Microbiology and Immunology, Vol. 361, pp. 39–51). Springer; 2012.
36. Ferreira TS, Felizardo MR, de Gobbi DD, Gomes CR, Filsner PHN, Moreno M, Paixão RJ de JP, Moreno AM. Virulence genes and antimicrobial resistance profiles of *Pasteurella multocida* strains isolated from rabbits in Brazil. *The Scientific World Journal*. 2012;2012:685028.

37. Verma S, Sharma M, Katoch S, Verma L, Kumar S, Dogra V, Singh G. Profiling of virulence-associated genes of *Pasteurella multocida* isolated from cattle. *Veterinary Research Communications*. 2013;37(1):83–89.
38. Tawab A, Akbar N, Hassan M, Habib F, Ali A, Rahman M, Iqbal M. Mass spectrometric analysis of lipid A obtained from the lipopolysaccharide of *Pasteurella multocida*. *RSC Advances*. 2020;10(51):30917–30933.
39. Desem MI, Handharyani E, Setiyono A, Safika S, Subekti DT, Ekawasti F. Morphology, biochemical, and molecular characterization of *Pasteurella multocida* causing hemorrhagic septicemia in Indonesia. *Veterinary Medicine International*. 2023;2023(1):7778707.
40. Doyle-Baker D, Ngeleka M, Janzen E, Briggs RE, Davies JL. Septicemic pasteurellosis causing peracute death and necrotizing myositis in a beef heifer calf (*Bos taurus*) in Alberta, Canada. *The Canadian Veterinary Journal*. 2020;61(12):1303–1305.
41. Peng Z, Liang W, Liu W, Wu B, Tang B, Tan C, Chen H. Genomic characterization of *Pasteurella multocida* HB01, a serotype A bovine isolate from China. *Gene*. 2016;581(1):85–93.
42. Kubatzky KF. *Pasteurella multocida* toxin—lessons learned from a mitogenic toxin. *Frontiers in Immunology*. 2022;13:1058905.
43. Horadagoda NU, Hodgson JC, Moon GM, Wijewardana TG, Eckersall PD. Role of endotoxin in the pathogenesis of haemorrhagic septicemia in the buffalo. *Microbial Pathogenesis*. 2001;30(3):171–178.
44. Chanda MM, Purse BV, Hemadri D, Patil SS, Yogisharadhya R, Prajapati A, Shivachandra SB. Spatial and temporal analysis of haemorrhagic septicemia outbreaks in India over three decades (1987–2016). *Scientific Reports*. 2024;14(1):6773.
45. Fegan JE, Waeckerlin RC, Tesfaw L, Islam EA, Deresse G, Dufera D, Gray-Owen SD. Developing a PmSLP3-based vaccine formulation that provides robust long-lasting protection against hemorrhagic septicemia—causing serogroup B and E strains of *Pasteurella multocida* in cattle. *Frontiers in Immunology*. 2024;15:1392681.
46. Panna SN, Nazir KNH, Rahman MB, Ahamed S, Saroare MG, Chakma S, Majumder UH. Isolation and molecular detection of *Pasteurella multocida* Type A from naturally infected chickens, and their histopathological evaluation in artificially infected chickens in Bangladesh. *Journal of Advanced Veterinary and Animal Research*. 2015;2(3):338–345.
47. Varshney R, Chaturvedi VK, Rawat M, Saminathan M, Singh V, Gupta PK. Development of novel iron-regulated *Pasteurella multocida* B: 2 bacterin and refinement of vaccine quality in terms of minimum variation in particle size and distribution vis-a-vis critical level of iron in media. *Microbial Pathogenesis*. 2020;147:104375.
48. OIE. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. Paris: World Organisation for Animal Health; 2008.
49. Jauregui R, Barcelo A, Bennett P, Foxwell J, Buckle K, O'Connell J, McCulley M. Complete genomes of *Pasteurella multocida* isolates from porcine, bovine, and cervine samples found in New Zealand farms. *Microbiology Resource Announcements*. 2023;12(12):e00884–23.
50. Moustafa AM, Ali SN, Bennett MD, Hyndman TH, Robertson ID, Edwards J. A case–control study of haemorrhagic septicemia in buffaloes and cattle in Karachi, Pakistan, in 2012. *Transboundary and Emerging Diseases*. 2017;64(2):520–527.
51. Verma R, Jaiswal TN. Haemorrhagic septicemia vaccines. *Vaccine*. 1998;16(11–12):1184–1192.
52. Khan A, Saddique U, Ahmad R, Khan H, Mohammad Y, Zubair M. Serosurveillance of hemorrhagic septicemia in cattle and buffaloes in district Malakand, NWFP. *J Agric Biol Sci*. 2006;1:11–14.
53. Habib F, Jabbar A, Shah Nawaz R, Memon A, Yousaf A, Bilal M, Sharif A. Prevalence of hemorrhagic septicemia in cattle and buffaloes in Tandojam, Sindh, Pakistan. *Online Journal of Animal and Feed Research*. 2019;9(5):187–190.
54. Verma SC, Mahajan NK, Malik G, Dahiya JP. An epidemiological study of bovine hemorrhagic septicemia in Haryana. *Indian Journal of Animal Research*. 2004;38(1):14–19.
55. Kutzer P, Szentiks CA, Bock S, Fritsch G, Magyar T, Schulze C, Ewers C. Re-emergence and spread of haemorrhagic septicemia in Germany: the wolf as a vector. *Microorganisms*. 2021;9(9):1999.
56. Sting R, Blazey B, Schwabe I, Schwalm A, Müller S, Sprague L, Rau J. Haemorrhagic septicemia (septicemic pasteurellosis) in cattle in Baden-Württemberg (Germany). *Berliner und Münchener Tierärztliche Wochenschrift*. 2020;133(7–8):289–296.
57. Bisht KS, Hassan L, Salim N, Zunita Z, Liew CS, Kamil WM, Saharee AA. Outbreak of haemorrhagic septicemia in Malaysia. *The Indian Veterinary Journal*. 2006;83(10):1110–1111.
58. Khan FM. Field epidemiology of an outbreak of hemorrhagic septicemia in dromedary population of greater Cholistan desert (Pakistan). *Pak. Vet. J*. 2012;32(1):31–4.
59. Bardhan D, Kumar S, Sekaran GA, Meraj M, Chilambarasan M, Singh RK, Kumar D. Economic losses due to hemorrhagic septicemia in India. *Indian Journal of Animal Sciences*. 2020;90(3):341–346.
60. Kawasaki M, Young JR, Suon S, Bush RD, Windsor PA. The Socioeconomic Impacts of Clinically Diagnosed Haemorrhagic Septicemia on Smallholder Large Ruminant Farmers in Cambodia. *Transboundary and Emerging Diseases*. 2015;62(5):535–548.
61. Mondal SP, Yamage M. A retrospective study on the epidemiology of anthrax, foot and mouth disease, haemorrhagic septicemia, peste des petits ruminants and rabies in Bangladesh, 2010–2012. *PLOS ONE*. 2014;9(8):e104435.
62. Calderon Bernal JM, Fernández A, Arnal JL, Sanz Tejero C, Fernández-Garayzábal JF, Vela AI, Cid D. Molecular epidemiology of *Pasteurella multocida* associated with bovine respiratory disease outbreaks. *Animals*. 2022;13(1):75.
63. Ali S, Tariq MHA, Yaqoob M, Haq MU, Zahra R. Molecular epidemiology and characterization of antibiotic resistance of *Pasteurella multocida* isolated from livestock population of Punjab, Pakistan. *International Journal of Veterinary Science and Medicine*. 2025;13(1):1–12.
64. Sunartatie T, Safika AH, Citra KR, Putra MA, Nugroho CMH, Mayasari NLPI, Indrawati A. Identification and Antibiotic resistance of *Pasteurella multocida* isolated from infected layer chickens in West Java, Indonesia. *Journal of World's Poultry Research*. 2024;14(3):282–290.
65. Townsend KM, Dawkins HJS, Papadimitriou JM. REP-PCR analysis of *Pasteurella multocida* isolates that cause haemorrhagic septicemia. *Research in Veterinary Science*. 1997;63(2):151–155.
66. Bhimani MP, Bhandari BB, Roy AR. Loop-mediated isothermal amplification assay (LAMP) based detection of *Pasteurella multocida* in cases of haemorrhagic septicemia and fowl cholera. *Vet Ital*. 2015;51(2):115–21.
67. Singhla T, Tankaw P, Sthitmatee N. Validation of a novel ELISA for the diagnosis of hemorrhagic septicemia in dairy cattle from Thailand using a Bayesian approach. *Veterinary Sciences*. 2020;7(4):163.
68. Hanchanachai N, Chumnanpuen P, E-kobon T. Interaction study of *Pasteurella multocida* with culturable aerobic bacteria isolated from porcine respiratory tracts using coculture in conditioned media. *BMC Microbiology*. 2021;21(1):19.

69. Narcana IK, Suardana IW, Besung INK. Molecular characteristic of *Pasteurella multocida* isolates from Sumba Island at East Nusa Tenggara Province, Indonesia. *Veterinary World*. 2020;13(1):104.
70. Tabatabaei M, Abdolahi F. Molecular evaluation of sheep and goats isolates of *Pasteurella multocida* and their antibiotic resistance. *Veterinary Research Forum*. 2023;14(9):481.
71. Jia J, Zhao M, Ma K, Zhang H, Gui L, Sun H, Zhao J. The Immunoprotection of OmpH gene deletion mutation of *Pasteurella multocida* on hemorrhagic sepsis in Qinghai Yak. *Veterinary Sciences*. 2023;10(3):221.
72. Cao X, Gu L, Gao Z, Fan W, Zhang Q, Sheng J, Sun Y. Pathogenicity and genomic characteristics analysis of *Pasteurella multocida* serotype a isolated from argali hybrid sheep. *Microorganisms*. 2024;12(6):1072.
73. Amirgazin A, Yessembekova G, Akhmetova A, Karibayev T, Mukanov K, Shevtsova E, Abdrakhmanov S. Genomic Insights into *Pasteurella multocida* Serotype B: 2 from Hemorrhagic Septicemia Outbreaks in Wildlife and Livestock in Kazakhstan. *Pathogens*. 2025;14(12):1273.
74. Pan Y, Yu Q, Wang Q, Li Q, Tian W, Xin L, Wu J. Establishment and epidemiological investigation of a dual fluorescent qPCR assay for *Pasteurella multocida* and *Salmonella* in yaks in the Tibetan Autonomous Prefecture of Garzê, China. *Frontiers in Cellular and Infection Microbiology*. 2025;15:1599817.
75. Zhao Y, Wang P, Yang Y, Xu Y, Wang J. Whole-Genome Sequencing and Pathogenic Characterization of a *Pasteurella multocida* Serotype A Isolate from a Case of Respiratory Disease in Tan Sheep. *Microorganisms*. 2026;14(1):154.
76. Rahman MA, Zereen F, Hossain MG, Al-Amin M, Alam J, Shimada M, Saha S. The first report of concurrent infection of hemorrhagic septicemia with foot and mouth disease in cattle in Bangladesh. *Comparative Immunology, Microbiology and Infectious Diseases*. 2024;115:102272.
77. Kerek Á, Szabó Á, Jerzsele Á. Antimicrobial susceptibility profiles of *Pasteurella multocida* isolates from clinical cases of waterfowl in Hungary between 2022 and 2023. *Veterinary Sciences*. 2024;11(5):194.
78. Petrocchi-Rilo M, Gutiérrez-Martín CB, Méndez-Hernández JI, Rodríguez-Ferri EF, Martínez-Martínez S. Antimicrobial resistance of *Pasteurella multocida* isolates recovered from swine pneumonia in Spain throughout 2017 and 2018. *Veterinary and Animal Science*. 2018;7:100044.
79. Pintér K, Pollák BD, Gantelet H, Magyar T. Antimicrobial susceptibility of *Pasteurella multocida* strains isolated from different host species. *Acta Veterinaria Hungarica*. 2024;72(2):71-79.
80. Sabsabi MA, Zakaria Z, Abu J, Faiz NM. Molecular characterisation and antibiotic sensitivity profile of *Pasteurella multocida* isolated from poultry farms in Malaysia. *Austral Journal of Veterinary Sciences*. 2021;53(2):121-126.
81. Bitew Z, Abayneh Tefera T, Deneke Y, Mariam T, Yihunie FB. Molecular serotyping and antimicrobial susceptibility profiles of *Pasteurella multocida* isolated from cases of hemorrhagic septicemia in cattle from selected districts of Keffa and Bench Sheko zones, South West Ethiopia. *BMC Microbiology*. 2025;25(1):224.
82. Hodgson JC, Finucane A, Dagleish MP, Ataei S, Parton R, Coote JG. Efficacy of vaccination of calves against hemorrhagic septicemia with a live *aroA* derivative of *Pasteurella multocida* B: 2 by two different routes of administration. *Infection and Immunity*. 2005;73(3):1475-1481.
83. Kumar S, Chaturvedi VK, Kumar B, Kumar P, Somarajan SR, Mishra AK, Sharma B. Effect of alum co-adjuvant of oil adjuvant vaccine on emulsion stability and immune responses against haemorrhagic septicemia in mice. *Iranian Journal of Microbiology*. 2015;7(2):79.
84. He F, Xiong P, Zhang H, Yang L, Qiu Y, Li P, Peng Y. Attenuated vaccine PmCQ2Δ4555–4580 effectively protects mice against *Pasteurella multocida* infection. *BMC Veterinary Research*. 2024;20(1):94.
85. Tesfaye AB, Werid GM, Tao Z, You L, Han R, Zhu J, Chu Y. Advances in *Pasteurella multocida* vaccine development: From conventional to next-generation strategies. *Vaccines*. 2025;13(10):1034.
86. Xu Q, Guan L, Xue Y, Zhao Z. Natural Attenuated Vaccines for *Pasteurella multocida*. *Biology*. 2025;14(11):1466.
87. Muenthaisong A, Nambooppha B, Rittipornlertrak A, Tankaew P, Varinrak T, Muangthai K, Sthitmatee N. An intranasal vaccination with a recombinant outer membrane protein H against haemorrhagic septicemia in swamp buffaloes. *Veterinary Medicine International*. 2020;2020(1):3548973.
88. Afroz H, Sattar S, Aziz A, Zamir B, Khan MUZ, Hussain S, Ahmad HMW. Comparative Assessment of Serum Biochemical Markers from Cattle and Buffaloes Vaccinated with Three Different Oil Adjuvant Vaccines of Hemorrhagic Septicemia from Punjab, Pakistan. *NUST Journal of Natural Sciences*. 2022;7(2):21–28.
89. Rustagi N, Kakker NK, Sheoran S, Santosh PC, Dahiya S, Gupta AK. Kinetics of humoral response to *Pasteurella multocida* in buffaloes against combined foot-and-mouth+ haemorrhagic septicemia vaccine. *Indian Journal of Animal Sciences*. 2023;93(9):853-857.
90. Facciola A, Visalli G, Laganà A, Di Pietro A. An overview of vaccine adjuvants: current evidence and future perspectives. *Vaccines*. 2022;10(5):819.
91. Shivachandra SB, Kumar A, Mohanty NN, Yogisharadhya R. Immunogenicity of recombinant Omp16 protein of *Pasteurella multocida* B: 2 in mouse model. *Indian Journal of Animal Sciences*. 2017;87(1):29-34.
92. Farooq U, Hussain M, Irshad H, Badar N, Munir R, Ali Q. Status of hemorrhagic septicemia based on epidemiology in Pakistan. *Pakistan Veterinary Journal*. 2007;27(2):67–72.
93. Moustafa AM, Bennett MD, Edwards J, Azim K, Mesaik MA, Choudhary MI, Anjum R. Molecular typing of haemorrhagic septicemia-associated *Pasteurella multocida* isolates from Pakistan and Thailand using multilocus sequence typing and pulsed-field gel electrophoresis. *Research in Veterinary Science*. 2013;95(3):986-990.
94. Yuan J, Li J, Du S, Wen Y, Wang Y, Lang YF, Cao SJ. Revealing the lethal effects of *Pasteurella multocida* toxin on multiple organ systems. *Frontiers in Microbiology*. 2024;15:1459124.

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