



Original Review Paper

Bovine Mastitis: A Review of the Current State of Knowledge

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Key Words Abstract

bovine mastitis; dairy cows; costly diseases in dairy farming worldwide. It causes significant economic losses through reduced udder health; milk yield, discarded milk, treatment expenses, cow culling, and it poses welfare and health prevention; challenges to dairy animals. Mastitis can be clinical, with observable udder inflammation and economic abnormal milk, or subclinical, with no visible signs but elevated somatic cell counts and diminished impact; milk production. The current state of knowledge on bovine mastitis, encompassing its etiology antimicrobial (primarily bacterial pathogens such as *Staphylococcus aureus* and *Streptococcus* spp.), resistance. pathogenesis of intramammary infection, risk factors influencing its occurrence (e.g. poor milking hygiene, environmental conditions), epidemiological trends in both global and Indian contexts. Methods for diagnosis and detection – from traditional culture and somatic cell count to newer molecular and on-farm rapid tests – are discussed, as are strategies for prevention and control. Management practices like proper milking routines, teat disinfection, and dry cow therapy remain cornerstones of mastitis control, while antibiotic therapy is the mainstay of treatment despite growing concerns over antimicrobial resistance. The highlights emerging alternatives such as vaccination efforts, genetic selection for mastitis resistance, novel non-antibiotic therapies (e.g. plant-based compounds, nanoparticles, phage therapy) aimed at improving udder health in the post-antibiotic era. The substantial economic impact of mastitis (billions of dollars loss) its implications for animal health, public health, continued research and improved management are critical. This comprehensive overview provides insight to current challenges and future prospects for controlling bovine mastitis in dairy herds globally.

Introduction

Bovine mastitis – the inflammation of the udder tissue – is a pervasive and enduring problem in dairy farming, with well-documented detrimental effects on animal welfare and farm profitability (Ruegg, 2017). The condition has a complex etiology and multifactorial nature, making it difficult to eliminate completely (Ashraf & Imran, 2020). Mastitis may result from physical injury or chemical irritation, but most often it is caused by microbial infection of the mammary gland. It has long been recognized as one of the most common diseases of dairy cattle globally, with incidence and prevalence remaining high in many regions despite decades of control efforts (Seegers *et al.*, 2003). Importantly, mastitis occurs in two broad forms: **clinical mastitis**, where cows exhibit visible symptoms (swollen, red or painful udder; abnormal milk with clots or pus; fever or depression in severe cases), and

subclinical mastitis, which shows no outward signs but is characterized by an elevated somatic cell count (SCC) in milk and decreased milk production (Cheng & Han, 2020). Subclinical cases are far more frequent than clinical ones and often go undetected without specific testing, yet they account for a large proportion of the total economic losses due to mastitis (Cheng & Han, 2020).

The economic impact of bovine mastitis is profound at both the farm and industry levels. Infected cows produce less milk and of poorer quality, leading to reduced revenue for farmers. Milk from mastitic quarters may need to be discarded or may incur penalties for high SCC or antibiotic residues (Halasa *et al.*, 2007). Treatment and veterinary costs add to the financial burden, and cows with chronic or recurrent mastitis often have to be culled prematurely, representing a loss of valuable animals. It is estimated

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that mastitis costs the global dairy sector on the order of **\$20–30 billion USD per year** (Paramasivam & Gopal, 2023), with average losses of about \$110–200 per cow annually when both clinical and subclinical cases are considered (Cheng & Han, 2020; Hogeveen et al., 2011). In India – the world’s largest milk producer, contributing roughly 24% of global milk supply – mastitis is recognized as a major constraint to dairy productivity. Prevalence of subclinical mastitis in Indian dairy herds is very high, and the annual economic losses due to mastitis in India have been estimated at around \$2 billion (Bardhan, 2013; Paramasivam & Gopal, 2023). Beyond economics, mastitis also has public health implications: the infection can introduce bacterial pathogens and antibiotic residues into milk. If milk from mastitic cows is consumed raw or improperly pasteurized, it could pose zoonotic risks (Ashraf & Imran, 2020). Moreover, the heavy use of antibiotics to treat mastitis contributes to the emergence of antimicrobial-resistant bacteria, raising concerns from a One Health perspective (Paramasivam & Gopal, 2023). For all these reasons, controlling mastitis is critical for sustainable dairy farming.

This paper provides a comprehensive review of the current state of knowledge on bovine mastitis. It covers the causative agents and pathogenic mechanisms, identifies key risk factors and epidemiological trends, and discusses the available methods for diagnosis along with their advancements. Strategies for prevention and control are examined, including both traditional approaches (such as hygiene and antibiotics) and newer or alternative approaches (such as vaccines, genetic resistance breeding, and phytotherapy). The **global perspective** is complemented by specific notes on the Indian context where relevant, reflecting the importance of mastitis in India’s dairy industry alongside its worldwide significance. By synthesizing recent research findings and prevailing practices, we aim to highlight progress made and gaps that remain, informing future efforts to mitigate bovine mastitis and improve dairy cow health and productivity.

Etiology and Pathogenesis

Causative Agents of Mastitis

Mastitis is predominantly caused by **infectious microorganisms**, chiefly bacteria. Over 150 different bacterial species have been identified as causative agents of bovine mastitis, though a much smaller subset comprises the majority of cases (Paramasivam & Gopal, 2023; Seegers et al., 2003). The mastitis-causing bacteria are often classified into two groups based on their source and mode of transmission: **contagious pathogens** and **environmental pathogens**. Contagious mastitis pathogens are those that typically live on or in the cow’s udder and teat skin and spread from cow to cow, usually during milking. The classic

contagious organisms are *Staphylococcus aureus* and *Streptococcus agalactiae*, and to some extent *Mycoplasma bovis* and *Corynebacterium bovis*. These bacteria tend to cause persistent intramammary infections and can pass between cows via contaminated milking equipment, teat cups, or milker’s hands and towels (Cheng & Han, 2020). Environmental mastitis pathogens, on the other hand, are organisms that normally inhabit the cow’s surroundings (such as bedding, soil, manure, or water) and opportunistically infect the udder, usually between milking sessions. Major environmental culprits include coliform bacteria like *Escherichia coli* and *Klebsiella spp.*, as well as environmental streptococci such as *Streptococcus uberis* and *Streptococcus dysgalactiae* (Cheng & Han, 2020; Ajose et al., 2022). Coagulase-negative staphylococci (CNS), a group of minor staph species often found on skin, can also cause mostly subclinical mastitis and are considered opportunistic environmental pathogens.

Other types of microorganisms can cause mastitis less frequently. **Mycoplasmas** (especially *Mycoplasma bovis*) are an important cause of contagious mastitis in some herds; they can spread rapidly and lead to severe outbreaks that are unresponsive to typical antibiotic therapy due to the mycoplasma’s lack of a cell wall (Morales-Ubaldo et al., 2023). *M. bovis* mastitis is often accompanied by other systemic signs (like respiratory or joint issues) and once established in a herd, can be difficult to eradicate without aggressive measures (Cheng & Han, 2020). **Fungal** mastitis also occurs, usually as sporadic cases. Yeasts such as *Candida spp.* and filamentous fungi like *Aspergillus fumigatus* or *Trichosporon spp.* can infect the udder, often after prolonged antibiotic treatments which disrupt the normal teat canal flora (Ashraf & Imran, 2020). Fungal mastitis cases are typically subacute or chronic and may not respond to conventional antibiotics, sometimes requiring antifungal therapy or spontaneous culling of infected quarters. **Viral** causes of mastitis are relatively rare in bovines, but a few instances have been documented. For example, bovine herpesvirus-4 (BHV-4) has been associated with a condition called mammary pustular dermatitis and mastitis in cows, and retroviruses such as bovine leukemia virus or small ruminant lentiviruses (in goats) can occasionally localize in the mammary gland and contribute to inflammation (Ashraf & Imran, 2020). Overall, however, bacteria remain by far the principal cause of mastitis, with *Staphylococcus aureus* often cited as the single most commonly isolated pathogen from mastitic milk worldwide (Paramasivam & Gopal, 2023).

Table 1 below summarizes some of the major mastitis pathogens, their classification, and notable characteristics:

Table 1. Major Pathogens Causing Bovine Mastitis, Their Categories, and Characteristics

Pathogen	Category (Primary Source)	Notable Characteristics and Impact on Mastitis
<i>Staphylococcus aureus</i>	Contagious (cow-to-cow)	Gram-positive cocci; colonizes teat skin and udder. Causes chronic subclinical mastitis with intermittent clinical flare-ups. Often leads to high somatic cell counts and milk quality issues. Can form biofilms and abscesses in udder tissue, evading immune response. Infections are

		difficult to cure and commonly recur. Many strains show antimicrobial resistance (e.g., penicillin resistance is high), making treatment challenging (Hoque et al., 2018; Morales-Ubaldo <i>et al.</i> , 2023).
<i>Streptococcus agalactiae</i>	Contagious (cow-to-cow)	Gram-positive cocci; obligate udder parasite (found almost exclusively in infected udders). Spreads during milking. Causes subclinical mastitis with very high bacterial counts in milk and elevated SCC. Typically affects multiple quarters. Can be effectively controlled or eradicated at the herd level by aggressive antibiotic therapy and milking hygiene, as it is highly sensitive to penicillin (Cheng & Han, 2020).
<i>Escherichia coli</i>	Environmental (manure, bedding)	Gram-negative rod (coliform); ubiquitous in feces and the farm environment. Causes acute clinical mastitis, often of peracute or toxic form. Infected quarters become swollen and produce watery or serum-like milk; severe cases lead to fever, depression, or even endotoxemia in the cow. Many cases are short in duration because the cow's immune system can eliminate the infection quickly, but they can cause sudden milk drop and require supportive care (Cheng & Han, 2020). Antibiotic treatment may be of limited benefit in severe endotoxemic coliform mastitis, and prevention via cleanliness is key.
<i>Klebsiella</i> spp. (e.g. <i>K. pneumoniae</i>)	Environmental (soil, bedding especially wood shavings)	Gram-negative rod (coliform); thrives in organic bedding materials like sawdust. Tends to cause acute, often gangrenous mastitis with a high risk of quarter loss or cow mortality. <i>Klebsiella</i> mastitis presentations are similar to <i>E. coli</i> but can be more persistent. Prevention by avoiding sawdust bedding or ensuring it is dry and clean is important. Some strains have acquired resistance to multiple antibiotics, complicating treatment (Morales-Ubaldo <i>et al.</i> , 2023).
<i>Streptococcus uberis</i>	Environmental (bedding, pasture)	Gram-positive cocci; common in straw bedding, muck, and on pasture. Causes subclinical or mild clinical mastitis, frequently in the dry period or early lactation. Often associated with high SCC and chronic infections if not detected. Responds variably to antibiotics; ensuring good bedding hygiene and dry cow therapy helps control <i>S. uberis</i> (Ajose <i>et al.</i> , 2022).
<i>Mycoplasma bovis</i>	Contagious (respiratory transmission & milk)	Cell-wall-less bacterium; can cause severe mastitis outbreaks. Infected cows shed mycoplasma in milk, and it spreads via milking procedure or close contact. <i>M. bovis</i> mastitis typically results in multiple quarters being affected with a drastic drop in milk yield (milk may become thick or sandy). There is often no fever. Because <i>M. bovis</i> is inherently resistant to beta-lactam antibiotics (no cell wall) and many other drugs, these mastitis cases do not respond to standard therapy. Control hinges on identifying and segregating or culling infected cows to protect the herd (Morales-Ubaldo <i>et al.</i> , 2023).
Minor pathogens (various)	Opportunistic (skin flora, etc.)	A variety of other microbes can occasionally cause mastitis. For example, coagulase-negative staphylococci (like <i>Staph. epidermidis</i> , <i>Staph. simulans</i> , etc.) are normal teat skin flora that cause mild subclinical mastitis, often self-limiting but contributing to elevated SCC. <i>Corynebacterium bovis</i> is another minor pathogen colonizing teat ends; it causes mild inflammation and is often an indicator of teat hygiene issues. Fungal organisms (yeasts, <i>Nocardia</i> , <i>Prototheca</i> algae) are sporadic causes typically linked to contaminated treatment or environment. These minor pathogens usually produce less severe symptoms, but their presence can interfere with udder health monitoring programs (Ashraf & Imran, 2020).

Pathogenesis of Intramammary Infection

The development of mastitis begins with pathogens gaining entry into the mammary gland, almost always via the teat canal (streak canal). The teat canal is normally a tight barrier sealed by a keratin plug and sphincter, which helps prevent organisms from entering. During milking (or nursing), however, the teat sphincter relaxes and the canal opens, which can allow bacteria present on the teat skin or in the milking machine liners to penetrate into the teat cistern. Small

teat lesions or sores, as well as residual milk leakage after milking, can also facilitate bacterial entry. Once bacteria breach the teat canal defenses, they can colonize the milk in the teat and gland cisterns and begin to multiply in the nutrient-rich environment of milk.

The cow's immune system responds quickly to intramammary infection. The presence of bacteria and their toxins (such as endotoxins from Gram-negative bacteria or exotoxins from *S. aureus*) triggers an

inflammatory reaction. Chemical signals (cytokines, chemokines) are released by udder epithelial cells and resident immune cells, which cause blood vessels in the udder to dilate and recruit large numbers of white blood cells into the mammary tissue and milk. Neutrophils are the first major immune cells to flood into the gland; they migrate through the blood-udder barrier and enter the alveoli to phagocytose (ingest and kill) the invading bacteria. This influx of somatic cells (mostly neutrophils, along with macrophages and some lymphocytes) is what raises the **somatic cell count** in milk during mastitis from normal levels (usually <200,000 cells/mL) to often several million cells per milliliter. Neutrophils are essential for eliminating the infection, but in the process of attacking bacteria they also release enzymes and free radicals that can cause collateral damage to milk-producing tissue. Thus, a strong immune response can reduce milk secretion and alter milk composition (e.g., elevated sodium and chloride, giving salty taste, and decreased lactose) during the infection (Sharun *et al.*, 2021).

The severity of mastitis is influenced by both pathogen factors and host factors. **Pathogen virulence factors** play a significant role in how the disease manifests. For instance, *Staph. aureus* produces a suite of virulence factors: it can form protective biofilms inside the gland, secrete toxins (like alpha-toxin) that damage tissues and leukocytes, and evade immune detection through protein A and other mechanisms. These features help *S. aureus* establish chronic infections that smolder as subclinical mastitis with periodic clinical episodes (Hoque *et al.*, 2018). *E. coli*, in contrast, has potent endotoxins (lipopolysaccharide) on its cell wall that often provoke an intense immune response – sometimes so intense that it leads to systemic shock symptoms in the cow (fever, rapid pulse, dehydration). However, *E. coli* does not tend to persist long in the udder; the cow either clears the infection or, in severe cases, succumbs to the toxemia. *Strep. agalactiae* is a pathogen of relatively lower intrinsic virulence, but it multiplies extremely well in milk and can infect many quarters, leading to high bacterial counts and SCC until treated. *Mycoplasma bovis* can adhere to and damage udder tissue and often causes caseous necrosis (pus-filled nodules) internally, which is why it often results in a non-responsive mastitis that necessitates culling. From the **host perspective**, the outcome of infection depends on the cow's immune competency and other factors. Early lactation cows (soon after calving) and cows around the time of drying off are at higher risk for mastitis; during these periods, significant physiological and hormonal changes can temporarily dampen the udder's immune defenses (Cheng & Han, 2020). Older cows tend to be more susceptible to mastitis than first-lactation heifers because they may have accumulated teat-end damage or harbor chronic infections, and their teat canals may be more open or scarred (Cheng & Han, 2020). Once infection is established, if the immune system cannot eliminate all pathogens, a chronic infection may ensue. In chronic mastitis, the persistent inflammation can lead to fibrosis and scarring of milk-secreting tissue and blockage of ducts. This is why chronic mastitis (often due to *Staph. aureus* or *Strep.*

agalactiae) results in permanently reduced milk production in affected quarters. In summary, mastitis pathogenesis is a race between bacterial multiplication and virulence versus the cow's immune clearance; the clinical signs observed (from mild changes in milk to severe illness) reflect the intensity of this battle.

Risk Factors and Epidemiology

Risk Factors for Bovine Mastitis

The occurrence of mastitis in a dairy herd is influenced by a multitude of risk factors that can be broadly categorized into **pathogen factors, host (cow) factors, and environmental/management factors** (Cheng & Han, 2020). Understanding these risk factors is crucial for designing effective prevention strategies.

Pathogen Factors: Some mastitis pathogens inherently pose a higher risk due to their contagious nature or tenacity in the environment. For example, herds that have *Staphylococcus aureus* or *Mycoplasma bovis* present in even a few cows are at risk of wider transmission, especially if milking hygiene is suboptimal. Contagious pathogens can spread every milking if infected cows are not milked last or if equipment sanitization is inadequate. Pathogens capable of forming biofilms (like *S. aureus*) or surviving inside host cells can persist and cause recurrent infections despite therapy, increasing overall mastitis incidence (Hoque *et al.*, 2018). On the other hand, a farm environment heavily contaminated with environmental bacteria (like mud or manure laden with *E. coli* or *Strep. uberis*) raises the risk of new environmental mastitis cases, particularly if cows have exposure to dirty bedding or standing water.

Host (Cow) Factors: Individual cow susceptibility varies. Age and parity are important risk determinants – older cows (with higher parity) have been shown to experience more mastitis (Cheng & Han, 2020). This is partly because older cows' teat ends often have thicker, sometimes cracked skin and looser sphincters, making bacterial penetration easier, and they may have lingering infections from previous lactations. Additionally, older cows' immune functions can be less efficient in the face of repeated challenges. The **stage of lactation** is critical: mastitis is most likely to occur in the early lactation (first month after calving) and immediately after the dry-off period. Early lactation cows are immunocompromised due to the stresses of calving and the onset of heavy milk production; their nutritional demands and hormonal changes can reduce immune responsiveness (Cheng & Han, 2020). During the dry period, especially right after drying off and right before calving, the udder is vulnerable – if any bacteria enter the teat end when the cow is not being milked (hence bacteria are not regularly flushed out), they can establish infections that manifest as mastitis in the next lactation. Practices like providing *internal teat sealants* and antibiotic dry cow therapy address this risk. Cow anatomy and conformation can play a role too: cows with pendulous udders or teat injuries have higher risk, as do those with teat-end hyperkeratosis (callus formation) which can crack and harbor bacteria. Any condition that weakens the cow's immune system (such as negative energy balance, mineral/vitamin deficiencies, or other diseases like diabetes or ketosis)

can increase mastitis susceptibility. For instance, deficiency in vitamin E or selenium is known to impair neutrophil function and has been linked to higher mastitis rates; supplementation of these around calving has been shown to help reduce mastitis incidence (Cheng & Han, 2020).

Environmental and Management Factors: Mastitis is very much a management-related disease. **Milking hygiene** is paramount – poor milking practices greatly elevate risk. For example, not properly cleaning and drying teats before milking, or using common cloths on multiple cows, can transfer pathogens directly into the teat canal. Likewise, failing to disinfect teats after milking (post-milking teat dipping) leaves the teat canal susceptible to environmental bacteria while the sphincter is still relaxed for about an hour post-milking. It has been demonstrated that consistent post-milking teat dipping with an effective disinfectant dramatically reduces new infections by contagious organisms. **Milking machine function** is another key factor: if vacuum levels are too high or fluctuate, or if liners are worn out, the teat ends can be damaged or experience “liner slip” (suction loss with an influx of air). Liner slips can propel droplets of milk (potentially containing bacteria) up into the teat canal – a mechanism known as “impact mastitis.” Regular maintenance of milking machines and proper milking vacuum settings help avoid such issues. **Housing and bedding conditions** strongly influence environmental mastitis risk. Cows kept in damp, dirty bedding (whether in freestalls or loose housing) are far more likely to develop environmental mastitis. High **stocking density**, poor stall design, or inadequate bedding can lead to cows lying in manure or on wet surfaces, exposing teats to bacteria (Cheng & Han, 2020). Studies have shown that herds with cleaner, drier bedding (e.g., sand bedding or frequently changed straw) have lower rates of environmental mastitis (Cheng & Han, 2020). **Weather and climate** also play a role: hot and humid conditions can promote bacterial growth in the environment and on teat skin, partly explaining why mastitis (especially environmental mastitis) often peaks in warm, wet seasons. In contrast, in very cold climates, cows may spend more time in confined indoor spaces (with increased contact with bedding contaminants), and teat skin may chap, also raising risk. **Herd management practices** like how cows are grouped and milked can matter – for example, always milking known high-SCC or infected cows last helps prevent spreading their infection to others via the machine. Lack of routine herd health monitoring (e.g., not conducting regular milk somatic cell count checks or California Mastitis Tests) means subclinical cases go unnoticed and can become reservoirs of infection. Finally, **human factors** – the diligence and training of farm personnel – are fundamental. Farms with well-trained milkers who follow consistent udder preparation and milking protocols tend to have significantly lower mastitis incidence than farms where protocols are not standardized or followed. In summary, any factor that compromises udder defense (like injury or poor immunity) or increases bacterial exposure to the teat (like poor hygiene or dirty environment) can be

considered a risk factor for mastitis.

Epidemiology: Prevalence, Incidence, and Patterns

Bovine mastitis occurs worldwide and is often cited as the costliest disease of dairy cattle globally. The **prevalence** of mastitis can be defined at different levels – cow-level prevalence (the proportion of cows in a herd that have mastitis at a given time) or quarter-level prevalence (proportion of quarters infected), and it encompasses both clinical and subclinical cases. Numerous studies and surveys have attempted to quantify mastitis prevalence. A recent comprehensive meta-analysis reported that, on average, about **42% of dairy cows globally have subclinical mastitis** (as indicated by elevated SCC or positive tests) and approximately **15% of cows have clinical mastitis** during a given period (Paramasivam & Gopal, 2023). These numbers underline that subclinical infections are far more common than overt clinical cases. In many modern, well-managed herds in Europe or North America, the herd-level prevalence of subclinical mastitis (SCC >200,000 cells/mL in at least one quarter) might range from 20–40% of cows at any time, whereas clinical mastitis cases might be 2–10% of cows at a time. In contrast, in developing dairy sectors or smallholder systems (and even in some large farms) where preventive practices are less rigorous, prevalence can be higher. For example, studies in parts of Asia and Africa often find **50% or more cows with subclinical mastitis**. In India, a large systematic review estimated that around **45% of cows have subclinical mastitis and 18% have clinical mastitis** on average (Paramasivam & Gopal, 2023). Some local surveys in Indian states have revealed even more alarming figures – for instance, one study in the Kashmir region found over 80% of tested cows positive for subclinical mastitis (Bhat *et al.*, 2021, as cited in local literature), highlighting the endemic nature of the issue.

It's important to note that **prevalence varies widely by region and management level**. The burden of mastitis tends to be higher in countries with tropical climates and predominantly smallholder dairy farms (due to challenges in maintaining hygiene and routine veterinary care), as well as in farms that are undergoing intensification without adequate adoption of mastitis control measures. Conversely, regions with long-standing mastitis control programs (like Scandinavian countries) have comparatively lower prevalence.

Another epidemiological measure, the **incidence rate** of clinical mastitis, is often expressed as number of cases per 100 cows per year. A typical target in many dairy herd health programs is to keep clinical mastitis incidence below about 25 cases per 100 cow-years. Data from large-scale studies indicate incidence rates in well-managed U.S. and European herds around **20–30 cases per 100 cows annually** (Fortune Business Insights, 2020; Hogeveen *et al.*, 2011). This means roughly one in four cows might have a clinical case in a year. However, incidence can be much higher if control breaks down – in some herds it exceeds 100% (more than one case per cow per year on average). Seasonal peaks are common: environmental mastitis (coliforms, environmental streps) often surges in warm,

humid summer months or rainy seasons, while some contagious mastitis might spike after the stress of winter calving or housing periods. In India and other South Asian countries, mastitis incidence tends to be highest in the monsoon season due to moisture and mud, and also around the peri-parturient period.

The **distribution of mastitis pathogens** also shows epidemiological patterns. Contagious pathogens like *S. agalactiae* have become rare in herds that have instituted antibiotic dry cow therapy and testing programs – for instance, many herds in North America and Europe have eliminated *Strep. agalactiae* entirely. *S. aureus* remains ubiquitous worldwide but its prevalence in bulk milk or in cows varies: some herds have very low levels (few chronic carriers) while others

have a high proportion of cows infected. Environmental pathogens are more uniformly present across herds because all farms have some level of environmental bacteria. However, farm management influences which environmental bacteria dominate – e.g., herds using organic bedding might have more *Klebsiella* issues, grazing herds might see more *Strep. uberis* from pasture, etc.

To illustrate regional differences, **Figure 1** shows reported prevalence rates of *Staphylococcus aureus* intramammary infection in mastitic cows from selected countries across different continents. These data, drawn from various studies, reflect how *S. aureus* infection rates can vary widely by location:

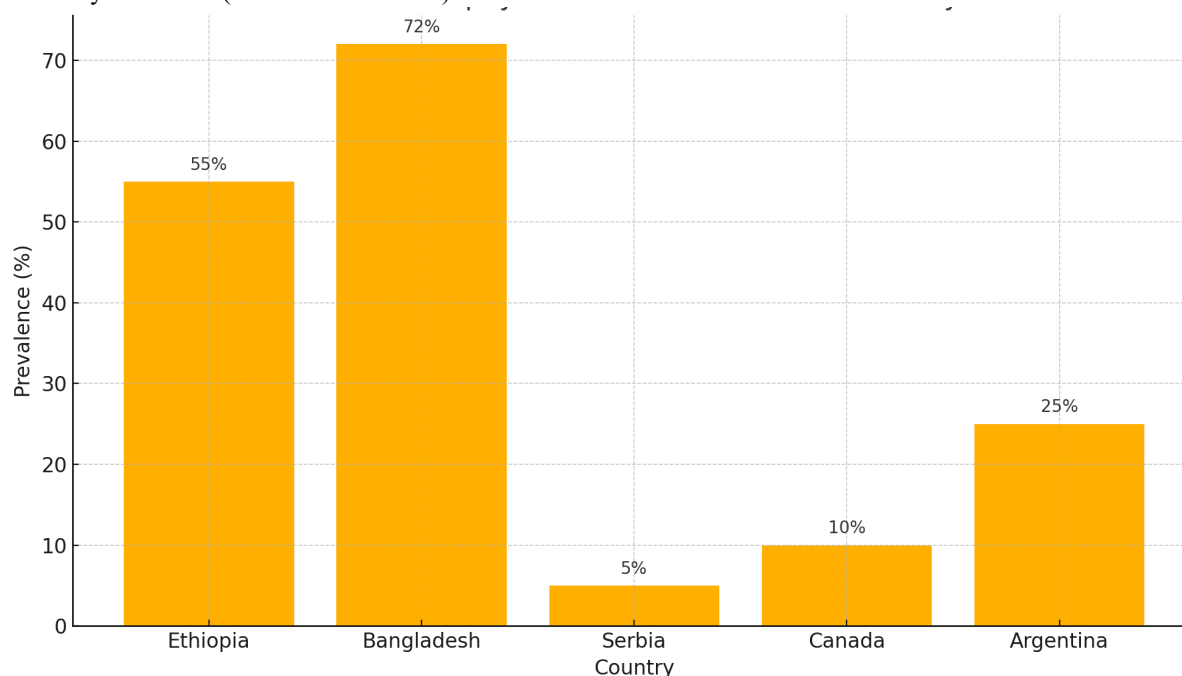


Figure 1. Prevalence of Staphylococcus aureus mastitis in dairy cows (percentage of cows or quarters infected) in selected countries. Data are approximate, based on reports from Ethiopia, Bangladesh, Serbia, Canada, and Argentina, representing Africa, Asia, Europe, North America, and South America respectively. Higher values indicate a larger proportion of mastitis cases caused by S. aureus. (Data synthesized from Morales-Ubaldo et al., 2023 and cited regional studies.)

As shown in Figure 1, studies in Ethiopia have found around 50–60% of mastitis cases involving *S. aureus*, while in Bangladesh the prevalence of *S. aureus* was extremely high (over 70% of mastitic cows in one study were positive for *S. aureus*) (Hoque et al., 2018). In contrast, a survey in Serbia (Europe) reported only about 5% prevalence of *S. aureus* mastitis, reflecting effective control in some European herds. North American data (e.g., Canada) often show relatively moderate levels (~10% of cows). South American results like in Argentina have reported *S. aureus* in roughly 20–30% of cases (Morales-Ubaldo et al., 2023). These differences stem from many factors: farm management quality, use of control practices, as well as possibly the dominant dairy breeds and production systems.

Another epidemiological aspect is **transmission dynamics**. In a herd context, contagious mastitis cases (especially *S. aureus* or mycoplasma) tend to create a

chronic reservoir of infection. If new infections outpace the removal or cure of existing infections, prevalence will climb. This can result in “problem herds” where mastitis becomes endemic. With good management, the transmission chain can be broken – for example, segregating or culling chronic *S. aureus* cows and rigorously post-dipping can dramatically lower new infection rates. Environmental mastitis does not transmit cow-to-cow, but its incidence is tied to daily exposure. Therefore, improvements in environmental cleanliness can yield immediate reductions in new cases.

Certain **management practices** influence epidemiological patterns: Herds that practice blanket dry cow antibiotic therapy (treating every quarter of every cow at dry-off) historically saw big drops in contagious mastitis prevalence and a reduction in new infections during the dry period (Halasa et al., 2007). Where such practices are not followed (like in small

farms or organic farms that avoid antibiotics), one might see higher carryover of infections from one lactation to the next. Likewise, farms with comprehensive mastitis monitoring (regular individual cow SCC testing, milk culturing of clinical cases) can identify issues early and intervene, thus keeping overall prevalence lower compared to farms without such monitoring.

In India, the epidemiology of mastitis is characterized by a very high prevalence of subclinical mastitis across both smallholder and commercial farms. Studies in various states of India commonly report 30–50% of animals affected subclinically (with higher rates in high-producing crossbred cows compared to indigenous breeds) (Bardhan, 2013). Clinical mastitis incidence in Indian dairy units has been noted to range widely but often falls between 20–40 cases per 100 cows-year, which is on the higher side by global standards. Contributory factors include the tropical climate (facilitating bacterial growth year-round), scarcity of trained personnel for milking in villages, and sometimes economic constraints leading to less use of dry cow therapy or teat dips. Notably, surveys in Central India found that mastitis accounted for approximately 49% of the total economic losses from dairy disease, underlining its significance (AJose *et al.*, 2022).

In summary, mastitis remains a ubiquitous problem with variable prevalence and incidence influenced by geography, farm management, and pathogen profiles. Global trends over recent decades have shown some improvement – for example, *Strep. agalactiae* has been dramatically reduced in many developed dairy industries – but other challenges such as environmental mastitis and *S. aureus* (including MRSA strains) persist. The epidemiological data reinforce that effective control measures can markedly alter herd-level mastitis rates, which is why continued emphasis on proven prevention strategies is warranted.

Diagnosis and Detection

Early and accurate diagnosis of mastitis is essential for effective management, as it enables timely treatment of clinical cases and identification of subclinical infections that might otherwise go unnoticed. The methods for diagnosing mastitis range from simple cow-side tests to advanced laboratory assays. Broadly, diagnostic approaches include **clinical examination of the udder and milk, somatic cell count measurements, bacteriological culture, and newer molecular or biochemical tests.**

Clinical Diagnosis: Farmers and veterinarians typically detect **clinical mastitis** through observation and physical exam. Signs in the cow's milk – such as clots, flakes, or a watery or blood-tinged appearance – are classic indicators. The affected quarter may be swollen, firm, warm to the touch, and painful (cow may kick or show discomfort on palpation). In moderate to severe cases, the cow may show systemic signs like fever, loss of appetite, or lethargy. During routine milking, milkers often use strip cups (fore-stripping a few streams of milk from each teat) to check for visible abnormalities in the milk as a quick screening for clinical mastitis. Any quarter showing visible changes

is marked as a clinical case for further evaluation. Clinical mastitis severity is sometimes graded: mild (abnormal milk only), moderate (abnormal milk with udder inflammation), or severe (systemic illness in cow). This helps decide treatment urgency.

Subclinical Mastitis Detection: Because subclinical mastitis does not produce visible changes in milk or udder, other tests are needed to detect it. The most widely used indicator is the **somatic cell count (SCC)** in milk. SCC is essentially a count of white blood cells (and some shed epithelial cells) present in the milk; an elevated SCC is a proxy for inflammation in the udder. In general, an individual quarter SCC above ~200,000 cells/mL is suggestive of mastitis (even if milk appears normal), and values in the millions are common in infected quarters (Sharun *et al.*, 2021). Many dairies regularly test bulk tank SCC (which is the composite of milk from all cows) as a measure of overall udder health – regulatory limits for bulk milk SCC are often around 400,000 cells/mL in the EU and 750,000 in the US, but quality-conscious farms aim for much lower (e.g., <200,000). To identify which cows are contributing high cells, **cow-level SCC tests** are performed, usually through monthly or quarterly milk recording programs where a sample from each cow is analyzed in a lab (often by electronic cell counters or via a portable device). Subclinically infected cows can then be flagged for management (such as treatment or segregation).

On-farm, a convenient test for subclinical mastitis is the **California Mastitis Test (CMT)**. The CMT is a cow-side test where a reagent is mixed with a squirt of milk from each quarter in a paddle; the reagent causes a gel-like reaction in proportion to the cell count. A thick gel indicates high SCC. Farmers use the CMT because it's inexpensive and easy – it provides a semi-quantitative indication of which quarters likely have infections. It is very useful for screening fresh cows after calving or periodically checking subclinical status of the herd. The CMT correlates reasonably well with lab SCC counts and can detect subclinical cases that should be further investigated (Stanek *et al.*, 2024). According to recent evaluations, the CMT has a sensitivity and specificity in the range of ~85–90% for detecting high cell count quarters, making it a reliable field tool (Paramasivam & Gopal, 2023).

Bacteriological Culture: To confirm the diagnosis of mastitis and identify the causative agent, **bacteriological culture of milk** is the gold standard. Milk samples from suspected mastitic quarters (whether clinical or subclinical high SCC) are aseptically collected and submitted to a laboratory, where they are plated on culture media to grow any bacteria present. Culture can tell not only if a significant pathogen is present, but also which organism it is (based on colony characteristics, Gram stain, biochemical tests, etc.). This is critical for distinguishing contagious from environmental mastitis and for making treatment decisions (certain bacteria, like *Mycoplasma* or *Nocardia*, won't respond to typical treatments, so knowing the pathogen guides proper action). In practice, many veterinarians or herd managers will culture milk from clinical cases either in

a professional lab or using on-farm culture systems. On-farm culture kits have been developed that allow farmers to grow common mastitis pathogens on special media plates within 24 hours. These can differentiate, for example, Gram-negative vs. Gram-positive growth (some systems use bi-plates or tri-plates with different media). The farmer can then decide treatment: for instance, if the culture shows Gram-negative bacteria (like *E. coli*), the farmer might avoid unwarranted antibiotic treatment and instead provide supportive care, whereas Gram-positives would be treated with appropriate antibiotics. This **selective treatment approach based on culture** has been shown to reduce antibiotic usage without harming outcomes (Ruegg, 2017). Traditional lab cultures also enable **antibiotic sensitivity testing**, where the isolated bacteria are tested against various antibiotics to see which are effective. This is particularly important in herds with recurring issues, to detect any antimicrobial resistance (e.g., some *Staph. aureus* may be penicillin-resistant, which would necessitate using a different drug).

However, one limitation of culture is that it can be time-consuming (taking 1–3 days for results), and some cases yield no growth (especially if the infection was cleared or if it's due to organisms like *Mycoplasma* that need special media, or if antibiotics were used before sampling). In recent years, **molecular diagnostic techniques** have emerged to complement or even replace culture in some settings.

Molecular and Rapid Diagnostics: Modern diagnostics leverage DNA-based methods to detect mastitis pathogens quickly and sensitively. **Polymerase chain reaction (PCR)** assays can identify DNA of common mastitis-causing bacteria directly from milk, even if the bacteria are present in low numbers or are not viable (Sharun *et al.*, 2021). Commercial PCR panels (such as multiplex PCR kits) are available that can simultaneously test for a dozen or more mastitis pathogens from a single milk sample. These PCR tests can return results within hours, identifying whether *S. aureus*, *Strep. agalactiae*, *Mycoplasma*, *E. coli*, etc., are present. They are highly useful for detecting problematic pathogens like *Mycoplasma bovis* which are hard to grow. One challenge is that PCR might detect DNA from dead bacteria or mild contamination, so results need interpretation in context (a positive PCR for a minor contaminant might not mean true infection). Nonetheless, PCR has been a game-changer for **bulk tank milk monitoring** – dairies can screen bulk milk periodically by PCR for traces of *Strep. agalactiae* or *Mycoplasma*, which if found trigger further investigation to find and manage the infected cows before an outbreak spreads (Morales-Ubaldo *et al.*, 2023).

Another advanced method is **matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF)** mass spectrometry, which some veterinary labs use to identify bacteria rapidly after a short culture, by analyzing the protein spectral “fingerprint” of the colony. This can significantly cut down identification time compared to traditional biochemical ID.

In addition to identifying the causative agents,

researchers and labs have been working on tests that detect **markers of mastitis** in milk or blood. For instance, mastitis causes an increase in certain enzymes in milk, like *N-acetyl-β-D-glucosaminidase (NAGase)* and *lactate dehydrogenase (LDH)*, which are released from damaged udder cells. These enzyme activities can be measured and have been proposed as indicators of subclinical mastitis. Stanek *et al.* (2024) note that tests based on NAGase or LDH activity are among the newer diagnostic methods under study, offering a way to sense tissue damage in early infection. Similarly, **acute phase proteins (APPs)** in blood or milk, such as haptoglobin, serum amyloid A (SAA), fibrinogen, and others, rise during inflammation. Elevated levels of these APPs in milk or serum can serve as additional biomarkers for mastitis (Stanek *et al.*, 2024). Some on-farm tests for haptoglobin or SAA are being explored to help detect subclinical mastitis or monitor herd inflammation status.

Technology is also enabling **automated mastitis detection**. Many modern milking systems and robotic milkers incorporate in-line sensors that measure things like milk electrical conductivity, color, and flow per quarter at each milking. During mastitis, the salt content of milk increases (due to leakage of sodium and chloride from blood) which raises electrical conductivity. Automated systems use sudden spikes in conductivity (or deviations from the cow's baseline) as a signal that a quarter may be developing mastitis. They can then alert the farmer or separate that milk. Some systems also use infrared thermography to detect heat in the udder or imaging to detect swelling asymmetry. These automated approaches can catch cases between routine checks, although they can generate false positives due to normal variations (Ashraf & Imran, 2020). Ongoing improvements, such as combining multiple sensor data (conductivity + milk color + cow activity etc.), are increasing the accuracy of on-line detection.

A notable advancement in subclinical mastitis monitoring is the introduction of **differential somatic cell count (DSCC)**. Traditional SCC does not distinguish the types of cells present, but DSCC refers to the proportion of neutrophils vs. lymphocytes/macrophages in the cell count. In mastitic milk, neutrophils dominate (often >80% of cells), whereas in normal milk macrophages and lymphocytes prevail. Newer cell counters can provide DSCC values, which might improve the ability to detect an ongoing infection or differentiate types of mastitis. Stanek *et al.* (2024) highlight DSCC as a more precise parameter to describe udder health compared to total SCC alone.

In summary, **diagnosis of mastitis** today is a combination of cow-side evaluations and advanced tests: farmers and vets identify clinical cases by inspection and simple tests like CMT, while veterinary labs or on-farm equipment provide SCC counts and identify causative organisms via culture or PCR. Early detection of subclinical cases (through SCC or sensors) is crucial, as treating or drying off those quarters can prevent progression and reduce spread. The trend is toward faster and more cow-side diagnostics – for example, developing a handheld device that could

detect specific mastitis pathogens in milk within minutes (Paramasivam & Gopal, 2023). While we are not fully there yet, progress in biosensors and molecular kits is continually improving mastitis detection, allowing farmers to make quicker decisions to protect udder health.

Prevention and Control

Preventing mastitis and controlling its spread in a herd require an integrated approach focusing on hygiene, management, and targeted therapeutic or prophylactic measures. The cornerstone principles of mastitis control were established decades ago in the form of the “Five-Point Plan” (which emphasized teat disinfection, dry cow therapy, prompt treatment of clinical cases, culling chronic cases, and milking machine maintenance) – and these principles remain highly relevant (Ruegg, 2017). Over time, additional strategies and technologies have been added to the toolbox. Here we discuss the major components of mastitis prevention and control, along with newer developments and their limitations.

Milking Hygiene and Routine

Proper milking practices are arguably the most important factor in preventing mastitis, especially the contagious type. **Pre-milking hygiene** involves cleaning the teats before attaching milking units. This typically means using a sanitizing teat dip or wash, then drying teats with individual paper towels or clean cloth towels. Effective pre-milking prep removes dirt and bacteria from the teat skin, reducing the chance of those organisms getting into the teat canal during milking. It also stimulates milk let-down. Farms that implement consistent teat preparation have significantly lower bacterial counts entering the bulk tank and lower new infection rates (Cheng & Han, 2020). **Post-milking teat disinfection** is crucial as well – immediately after milking, each teat is dipped (or sprayed) in an antiseptic solution (common ingredients include iodine, chlorhexidine, or lactic acid with emollients). This kills bacteria on the skin that could otherwise enter the open teat canal in the minutes after milking. Post-dipping is highly effective against contagious pathogens like *S. agalactiae* and *S. aureus*; it can reduce new infections by these organisms by over 50% when done consistently (Cheng & Han, 2020). It also helps with some environmental bacteria that get on teats right after milking.

The **milking order** can be strategically managed to control contagion: generally, milk the first-lactation heifers and uninfected cows first, and known high-SCC or mastitic cows last. This way, if there is any bacteria transfer via equipment, it goes from healthier to already-infected animals, not vice versa. Milking equipment should be **cleansed and sanitized** between milkings (and liners flushed between groups if possible) to avoid carrying pathogens over. Many farms have adopted **single-use cloths** or paper towels for teat drying to avoid cross-contamination (using the same cloth on multiple cows can spread bacteria).

Proper **functioning of the milking machine** must be maintained. Regular testing of vacuum stability, pulsation system, and replacing rubber parts (liners, hoses) on schedule helps prevent machine-induced teat

damage or “backflow” of milk that can carry pathogens. For example, high liner slips or cluster fall-offs that suck air can propel tiny droplets of milk upwards into the teat ends (an effect called “impact”), potentially injecting bacteria from the liner or from milk of another quarter. Modern machines often have features to minimize these effects, but only if well-maintained. Training milkers to **attach and detach units carefully** (e.g., shutting off vacuum before removal) also protects teat health.

In smallholder settings where hand-milking is practiced, hygiene is equally important: washers should clean the udder and hands should be clean (ideally dipped in sanitizer between cows). It’s recommended not to pool the milk from different quarters in the same bucket until each quarter’s foremilk has been examined for clots (to catch a mastitis quarter early and avoid spreading that milk).

Environmental Management

Because so many mastitis cases stem from the cow’s environment, keeping that environment clean and comfortable has huge benefits. **Bedding management** is a focal point. For housed cows, bedding (whether straw, sawdust, sand, or mattresses) should be kept dry and routinely changed or refreshed. Bacteria multiply rapidly in moist organic bedding. Using clean, dry bedding and replenishing it frequently reduces bacterial load that contacts teats (Cheng & Han, 2020). In high-risk areas (like maternity pens or calf nursing areas for nurse cows), extra attention to hygiene is needed since teat ends can be more exposed or calves could introduce oral bacteria to teat ends. **Manure management** in barns – regular scraping of alleyways, maintaining manure-free lying areas – lowers contamination. In pasture-based systems, providing a clean, dry lounging area (e.g., a well-drained pasture or providing access to a barn in extreme weather) can prevent environmental mastitis surges that happen when cows lie in muddy paddocks. Overcrowding is problematic because it increases the dirtiness of bedding and floors and also stresses cows, possibly lowering immunity. Thus, maintaining appropriate stocking density and giving cows ample space to lie down (without competition) contributes to mastitis control (Cheng & Han, 2020). Good **ventilation** in barns is sometimes overlooked but is important: proper airflow helps keep bedding dry and reduces the humidity that fosters bacterial growth.

The **dry cow period** is a critical time to manage environment, as new infections often begin when cows are dry. Dry cows should be kept on clean pasture or in well-bedded pens, and not commingled with lactating cows (to minimize exposure to contagious mastitis cows). If on pasture, providing shelter during wet conditions and controlling mud is beneficial.

Dry Cow Therapy and Teat Sealants

The practice of **dry cow therapy (DCT)** involves treating cows at the end of lactation (dry-off) by infusing a long-acting antimicrobial into each quarter. The goals are twofold: to cure any existing subclinical infections before the next lactation, and to remain in the teat during the early dry period to prevent new infections. Blanket dry cow therapy (treating every

quarter of every cow) has been a standard in many countries and proved very effective at reducing mastitis in herds (Halasa *et al.*, 2007). Common antibiotics for DCT include penicillin/streptomycin, cloxacillin, cephalosporins, etc., formulated in slow-release bases. Dry cow therapy can eliminate a high percentage of *Strep. agalactiae* and *Staph. aureus* infections present at dry-off and dramatically lower the prevalence of these contagious agents (Cheng & Han, 2020). With increasing concern about antibiotic use, some herds have shifted to **selective dry cow therapy** (treating only cows or quarters that show infection or high SCC, and leaving low-SCC cows untreated). When doing selective DCT, it's critical to accurately identify uninfected cows, usually via SCC records and possibly cultures before dry-off.

In addition to antibiotics, **internal teat sealants** have become popular. These are inert pastes (e.g., bismuth subnitrate based) that are infused into the teat canal at dry-off and form a physical barrier, mimicking the natural keratin plug. Teat sealants help prevent bacteria from entering the teat during the dry period. They are particularly useful for cows that might not form a good keratin plug or when environmental challenge is high. Studies have shown that using an internal teat sealant (especially in combination with DCT or on its own for uninfected cows) significantly reduces new dry period infections and subsequent mastitis in early lactation (Cheng & Han, 2020). At calving, the sealant comes out with the first strips of milk. Adoption of teat sealants in many herds has contributed to lowered incidence of environmental mastitis postpartum.

Prompt Treatment of Clinical Cases

Treating clinical mastitis quickly is important both to reduce the duration and severity of the disease for the cow and to limit the spread of infection (in case of contagious pathogens). The standard approach to treating a typical case of clinical mastitis (particularly those caused by Gram-positive bacteria like streps or staph) is to administer **intramammary antibiotic therapy**. A variety of intramammary infusion products are available (penicillins, cephalosporins, cloxacillin, erythromycin, etc.), and selection often depends on likely pathogen and local resistance patterns. For example, *Strep. agalactiae* is very responsive to penicillin, while *Staph. aureus* might require a longer course or a penicillinase-resistant antibiotic. Many mild cases of environmental mastitis (like *E. coli*) will self-cure, so some farms use a protocol to only use antibiotics if a Gram-positive is identified (via on-farm culture) or if the case doesn't rapidly improve. This selective treatment approach can reduce unnecessary antibiotic use and is increasingly promoted in light of antimicrobial stewardship (Ruegg, 2017).

For more severe mastitis cases, especially those with systemic involvement (fever, dehydration), **systemic treatments** are indicated. This typically includes systemic antibiotics (e.g., oxytetracycline, potentiated sulphonamides, or in some cases intravenous cephalosporins) and importantly, **supportive therapy** such as anti-inflammatory drugs (NSAIDs like flunixin meglumine) and fluid therapy. Anti-inflammatories help reduce the inflammation, pain, and fever, and

there's evidence they can improve outcomes and return to production by mitigating tissue damage (Hogeveen *et al.*, 2011). In cases of toxic mastitis (often from coliforms), aggressive support with IV fluids, electrolytes, and even calcium (to combat udder-derived hypocalcemia) can be life-saving. Frequent milking or stripping out of the quarter is recommended to remove toxins and bacteria-laden milk. Some protocols include using oxytocin injections to help milk out the quarter thoroughly.

After treatment, **milk from treated cows** must be discarded until the drug withdrawal period is over to ensure no antibiotic residues enter the food supply. Ensuring proper adherence to withdrawal times is a key part of mastitis control from a public health perspective. Many farms use individual cow charts or computer systems to track treated cows and auto-divert their milk.

Culling and Biosecurity

Despite best efforts, a subset of cows may become chronic mastitis cases. Cows that have repeated clinical episodes or consistently high SCC due to the same infection (especially with *Staph. aureus* or *Mycoplasma*) are often **culled (removed from the herd)** as part of control. Culling these chronically infected cows can dramatically reduce the herd's infection reservoir and lower exposure to other cows (Cheng & Han, 2020). It's a cost-benefit decision: if a cow is not responding to treatment and is a source of contagion, her removal protects herd mates and economic losses from her ongoing infection. Segregation is another approach – keeping high-SCC cows in a separate group and milking them last, or even having separate milking units for them – though not all farms have the capacity to do this long-term.

Biosecurity measures are critical to prevent introducing **new mastitis pathogens**. Purchasing cows or integrating animals from other herds can bring in problems like *Mycoplasma bovis* or other virulent bacteria. It is advisable to **screen new additions** (culture milk or PCR for key pathogens) and perhaps quarantine them before mixing with the herd. Also, visitors or personnel should maintain cleanliness (disinfect boots, etc.) to not inadvertently carry bacteria between farms. In some regions, *Strep. agalactiae* re-emergence has been linked to bought-in cattle from herds that still had it.

Vaccination

Vaccines against mastitis have been pursued for decades, but with limited success so far. A few commercial mastitis vaccines exist – notably, vaccines against *Escherichia coli* (J5 core-antigen vaccines) and *Staphylococcus aureus* bacterins or toxoid-based vaccines. The **J5 coliform vaccines** are commonly used in dairy herds; they do not prevent *E. coli* mastitis infection per se, but studies show they can reduce the severity of clinical signs when *E. coli* or other coliform mastitis occurs, essentially by priming the cow's immune system to better handle the endotoxin (Rainard *et al.*, 2022). Typically, cows are vaccinated in late gestation and again mid-lactation. These vaccines have become part of mastitis control in many large herds to curb the impact of coliform mastitis in early lactation.

For *Staph. aureus*, vaccines have been more challenging. *S. aureus* has multiple immune evasion strategies and antigenic diversity. Some **experimental and commercial staph vaccines** (often containing inactivated whole cells and possibly antigens like clumping factor or toxoids) have shown partial protection – for example, reducing the number of new infections or reducing the severity of existing infections – but none provide strong, lasting immunity that prevents infection entirely (Rainard *et al.*, 2022). Results from field trials are mixed: in some, vaccinated heifers had lower incidence of chronic *S. aureus* mastitis; in others, outcomes were not significantly improved. As a result, *S. aureus* vaccination has not been universally adopted, though in some regions farmers do use available products in hopes of some benefit.

Research is ongoing into improved mastitis vaccines targeting not just *S. aureus* but also *Strep. uberis* and other pathogens. Novel approaches, including targeting specific virulence factors, using surface protein cocktails, or even developing live-attenuated vaccines, are being explored (Rainard *et al.*, 2022). A recent perspective is that an effective mastitis vaccine may need to invoke a strong cell-mediated immunity in addition to antibodies, because the usual antibody response to natural infection doesn't protect well (Rainard *et al.*, 2022). So far, no vaccine provides sterilizing immunity or complete prevention of mastitis – at best, they are tools to reduce severity or number of cases. Nonetheless, even that reduction can be valuable in high-risk herds, and thus vaccination is often used as a supporting control measure, not a standalone solution.

Selective Breeding and Genetic Resistance

There is genetic variation among cattle in susceptibility to mastitis. Heritability of mastitis is moderate (often cited around 0.1 to 0.2 for SCC, meaning 10-20% of the variation is genetic), which means it's possible to make genetic progress by selecting for mastitis resistance. Most breeding programs for dairy cattle include **somatic cell score (a logarithmic transform of SCC)** as an indirect selection trait, since lower SCC tends to correlate with lower mastitis incidence. Over the years, bulls with progeny that have lower SCC and fewer mastitis cases are given better rankings for udder health. This has slowly been helping – for instance, Scandinavian countries have long selected for mastitis resistance and indeed their Nordic breeds (like Swedish Red) are known for lower mastitis rates compared to some other breeds under similar conditions.

More recently, genomic tools have identified specific **quantitative trait loci (QTLs) and single nucleotide polymorphisms (SNPs)** associated with mastitis resistance (Stanek *et al.*, 2024). For example, certain immune receptor genes or genes related to teat morphology can influence how well a cow resists infection. By incorporating these genomic markers into selection indices, breeders aim to accelerate gains in mastitis resistance. It's worth noting, however, that strong selection for milk yield in the past inadvertently increased mastitis susceptibility (high-producing cows tend to be more prone to mastitis), so now breeding programs try to balance production with health traits.

Selecting for cows that maintain lower SCC and have fewer clinical cases (data gathered through national databases or milk recording) is gradually making a more mastitis-resilient dairy cow. This is a long-term strategy – improvement is incremental over generations, but it is a sustainable form of prevention.

Alternative and Supportive Therapies

Given concerns about antibiotic overuse and resistance, there is active research and field experimentation with **alternative therapies** for mastitis. Some of these include:

- **Herbal and Plant-Based Treatments:** Various plant extracts and essential oils have antimicrobial or anti-inflammatory properties. For example, neem, turmeric, aloe vera, and green tea extracts have been tested as intramammary infusions or as supportive therapy for mastitis (Ajose *et al.*, 2022). A few herbal formulations have shown moderate success in reducing SCC or helping mild mastitis cases, but generally their efficacy is not on par with antibiotics for bacterial clearance in moderate to severe cases. They might, however, play a role in organic farming systems or as prophylactics (e.g., post-milking teat sprays containing essential oils that have antiseptic qualities). As an example, an ethno-veterinary approach in India using a poultice of turmeric and other herbs was noted anecdotally to reduce swelling and SCC (Ajose *et al.*, 2022).
- **Probiotics and Competitive Exclusion:** The idea here is to use beneficial bacteria to outcompete pathogenic ones in the teat canal or udder. Infusion of non-pathogenic lactic acid bacteria into the teat has been experimented with, aiming to establish a protective flora. Some preliminary studies found that certain ***Lactobacillus* strains** could reduce new infections, but this is not yet a mainstream practice.
- **Bacteriophage Therapy:** Bacteriophages are viruses that infect bacteria. Phage therapy for mastitis involves applying phages specific to common mastitis pathogens, such that they lyse the bacteria in the udder. This approach is promising especially for drug-resistant infections. Researchers have isolated phages that target *Staph. aureus*, and in vitro they can kill staph effectively. Some animal trials have shown reduction in bacterial counts with phage infusion (Ajose *et al.*, 2022). Challenges include ensuring the phages reach the infection site in sufficient numbers and remain active in the milk environment. Regulatory approval and consistency are still hurdles, but phages could become a valuable tool in the future, especially as an alternative to antibiotics for chronic staph mastitis.
- **Immune Modulation and Immunotherapy:** Instead of directly attacking the bacteria, another approach is to boost the cow's own

immune response to fight infection. Administering immunostimulants (like lysozyme or cytokine analogs) has been tried. For instance, substances like *Pegbovigrastim* (a bovine granulocyte colony-stimulating factor, trade name Imrestor) have been approved, which when given around calving can increase neutrophil counts and potentially reduce the incidence of early lactation mastitis by improving the cow's immune readiness. Additionally, some studies have looked at TLR (toll-like receptor) agonists to trigger local immune responses in the udder pre-infection (Stanek *et al.*, 2024). Another area is **stem cell therapy**, where bovine mammary stem cells or immune cells might be used to enhance healing of damaged tissue – this is still experimental.

- **Nanotechnology-Based Treatments:** Researchers are developing nano-formulations of antibiotics or novel antimicrobial substances that can better penetrate biofilms or release slowly in the udder. For example, nano-encapsulated antibiotics could potentially improve drug delivery to stubborn infections. Silver nanoparticles have antimicrobial properties and have been studied as a treatment for mastitis, with some success in reducing bacterial counts in milk in trials (Morales-Ubaldo *et al.*, 2023). However, safety and residue aspects must be considered for food products.

While these alternatives are promising, it's important to note that none yet replaces conventional methods in terms of proven efficacy for most mastitis cases. They are usually complementary. For example, a farmer might use a peppermint oil liniment to help reduce udder swelling (supportive care) but still rely on antibiotics to clear a *Strep* infection. Or use a post-milking teat dip containing essential oils to reduce chemical usage.

In the **post-antibiotic era mindset**, the goal is to reduce reliance on antibiotics due to resistance concerns (El-Sayed & Kamel, 2021). Therefore, much emphasis is put on *preventive measures* (hygiene, management, vaccination, genetics) to avoid infection in the first place. When infections do occur, one might increasingly choose targeted therapy (treat only the ones likely to benefit from antibiotics, based on culture results) and explore adjunct therapies to improve outcomes without excess drugs. In some high-value animals with chronic mastitis unresponsive to treatment, **surgical intervention** (such as teat canal surgery or even quarter mastectomy) can be a last resort to save the animal's productive life by removing the source of chronic infection, though this is rare and usually done only for pets or very valuable breeding stock given the cost and effort involved.

Monitoring and Ongoing Control

Mastitis control is not a one-time task but an

ongoing process. Regular monitoring of herd udder health through **bulk tank SCC** and individual cow SCC (via DHIA records or on-farm tests) is essential. Many farms set monthly targets and follow trends; if bulk SCC starts to creep up or the number of high-SCC cows increases, it's a red flag to investigate. **Milk culture surveillance** is also useful: for example, periodically culturing a random set of cows or all clinical cases to see what bugs are present and if any new problematic ones (like *Mycoplasma* or drug-resistant strains) have emerged. Some well-managed herds also perform **teat end scoring** (grading teat end condition) to ensure milking practices are not harming teat integrity – poor teat scores correlate with higher mastitis risk.

Education and training of farm staff is a continuous part of prevention. All milkers should understand the why and how of each hygiene step; new employees must be trained in the farm's protocols. Engaging with veterinarians and milk quality advisors for routine herd audits can help catch lapses or areas for improvement. Often, a **mastitis control plan** is documented, covering all these aspects (milking, environment, treatment protocols, culling policy, etc.), and reviewed annually.

Economic Impact

Mastitis inflicts a heavy economic toll on the dairy industry. The costs associated with mastitis are both **direct** and **indirect**, and they occur at both the farm level and the industry/national level. Understanding these costs is important for farmers and stakeholders to justify investments in mastitis control and to identify where interventions may yield the greatest economic benefit.

Direct Costs: These include the immediately observable expenses and losses from mastitis episodes. The primary direct cost is **lost milk production**. When a cow has mastitis, especially clinical mastitis, her milk yield typically drops – sometimes dramatically during the infection and often with incomplete recovery even after resolution (as damaged secretory tissue reduces future capacity). For subclinical mastitis, the loss is more insidious but cumulatively significant; a cow with a high somatic cell count produces less milk than she would if uninfected. Research indicates that each unit increase in SCC (on a log scale) corresponds to a measurable decline in milk yield. Mastitis can reduce a cow's lactation milk production by **5–25% or more** depending on severity and duration (Halasa *et al.*, 2007). Some estimates suggest a single clinical mastitis case can cause a loss of 200–500 kg of milk in the lactation, factoring in both immediate discard and residual loss in yield.

Another direct cost is **discarded milk**. Milk from clinical mastitis (with abnormal appearance) and milk from cows under treatment (with antibiotics) must be discarded and cannot be sold. In many cases, milk is discarded for several days per case (during treatment and the withdrawal period). If a cow produces, say, 30 liters a day and is out of tank

for 5 days, that's 150 liters lost from one case. Across a herd, this adds up – it's been estimated that discarded milk can account for roughly one-third of the total cost of a mastitis case (Paramasivam & Gopal, 2023).

Treatment costs are another direct expense: the price of antibiotics (intramammary tubes or injectables), anti-inflammatory drugs, and other medicines, as well as veterinary service fees if a vet is involved in treatment. Treating a simple case with a few intramammary tubes is relatively low cost (perhaps \$10-20), but treating a severe toxic mastitis can be expensive if IV fluids, multiple drugs, and intensive care are needed (in the hundreds of dollars). On average, treatment and drug costs are often cited to make up around 10–15% of the cost of a mastitis case (Paramasivam & Gopal, 2023).

Additionally, there is the **labor cost** associated with attending to mastitic cows – extra time to milk them separately or more frequently, to administer treatments, to monitor them – and potential **milk quality penalties**. In many milk pricing systems, a farm with high bulk tank SCC or with antibiotic residue violations will face financial penalties or even exclusion from the market until issues are resolved. Thus, mastitis can indirectly hit revenue through quality downgrades or rejected milk tankers.

Indirect Costs: These are often even larger than the direct costs and include longer-term or less immediately visible consequences. A major indirect cost is **increased culling and replacement**. Cows that have repeated mastitis or a particularly severe case may be culled earlier than they would have been, which means the farm must raise or purchase replacements. The cost of raising a heifer to first calving is substantial, so each cow lost prematurely to mastitis is a loss of that investment and the future milk she would produce. Mastitis is one of the leading reasons for involuntary culling in dairy herds, often second only to fertility problems. Studies in various countries have found that a significant fraction (up to ~20%) of dairy cow disposals are related to udder health problems. The economic model needs to account for the depreciation of a cow's value and the cost of bringing in a new cow. Some models have put the cost of culling due to mastitis at about \$150 to \$300 per case when averaged across all cases (since not every case leads to culling, but a fraction do) (Hogeveen *et al.*, 2011).

Another indirect cost is the impact on **reproductive performance**. Mastitis, especially clinical cases around breeding time, has been associated with decreased fertility – longer intervals to conception and higher likelihood of pregnancy loss. The stress and inflammation can disrupt hormonal balance. Halasa *et al.* (2007) noted that mastitis can extend calving-to-conception intervals, adding subtle costs in terms of extended lactations or more inseminations per pregnancy. Quantifying this, a cow that conceives later due to mastitis might

produce less milk in her lifetime or incur more insemination costs, which is an indirect economic hit.

Moreover, mastitis affects **milk quality and product yields**. High SCC milk has lower cheese yield (because enzymes from somatic cells break down milk proteins and reduce cheese curd formation) and can have off-flavors. On a farm level, high SCC can reduce the price per liter that farmers receive if their milk is classed in a lower quality bracket. At the industry level, the presence of mastitis can lead to wastage or lower efficiency in dairy product processing.

From a broader perspective, one can also consider **economic losses at the national or global scale**. For example, it has been estimated that mastitis accounts for about **70% of the total economic losses from all dairy cattle diseases** in some analyses, highlighting its predominance (Paramasivam & Gopal, 2023). As mentioned earlier, global losses have been put around \$19.7–\$32 billion annually (Fortune Business Insights, 2020; Paramasivam & Gopal, 2023). In the United States alone, mastitis is often cited to cost around \$2 billion per year to the dairy industry (Paramasivam & Gopal, 2023). In India, calculations of aggregated losses have yielded figures like 13,000 crore rupees (approximately \$1.8–\$2.2 billion USD) per year (Paramasivam & Gopal, 2023; Bardhan, 2013). These big numbers include all components: reduced national milk production, costs of treatments, losses from culling, etc.

To break down the cost of a single mastitis case, many studies use modeling. An example breakdown might be: production loss ~60-70% of total cost, discarded milk ~15-20%, treatment ~10%, and extra labor and culling risk comprising the rest (Hogeveen *et al.*, 2011; Paramasivam & Gopal, 2023). One cited average is around **\$350 per clinical mastitis case** in US conditions (Fortune Business Insights, 2020), though this can vary widely. Mild cases could be under \$100, while a severe case that causes a cow's death could be thousands of dollars lost.

There are also **intangible or harder-to-quantify costs**: Mastitis can predispose to other health issues or result in permanent quarter damage. It also has a cost in terms of animal welfare – cows with mastitis experience pain and discomfort, which though not directly an economic metric, is a concern for ethical farming and can influence consumer perceptions (and thus indirectly marketability of dairy products).

From a farm management standpoint, the economic impact of mastitis is often summarized in terms of **cost per cow per year** or **cost per lactation**. For instance, if a herd has an average of 30% subclinical mastitis and 20 cases of clinical mastitis per 100 cows, one can calculate the combined losses across those and come up with an average cost per cow. Cheng & Han (2020) noted an estimate that mastitis can constitute up to 11–18% of the gross margin per

cow per year – a very significant portion of potential profit being eroded.

Importantly, mastitis control measures themselves have costs (teat dips, dry cow antibiotics, etc.), so economically optimal mastitis level is not zero incidence (achieving zero would likely cost more than the disease itself in preventative effort). Instead, farmers aim to keep mastitis to a low level where marginal cost of further prevention would exceed marginal benefits. Generally, the investments in good mastitis prevention (like teat dipping and dry cow therapy) have shown **high return on investment** because they prevent much larger losses. For example, blanket dry cow therapy costs maybe \$10-20 per cow, but can prevent infections that would cost far more.

Finally, on an industry level, **mastitis affects supply and competitiveness**. Countries or regions with high mastitis levels have lower milk output and may struggle with quality issues that impede dairy product exports. Conversely, improvements in udder health contribute to efficiency gains in the dairy sector.

In summary, the economic impact of bovine mastitis is multifaceted: it reduces the volume and quality of milk produced (the primary income source), increases the costs of production (medicines, labor, herd replacement), and can shorten the productive life of cows. It is often cited as the most costly disease in dairy cattle. This is why mastitis control is not just about animal health but also about economic viability of dairy farming. Investments in mastitis prevention and prompt response are strongly justified by the avoidance of these losses. As an illustration of that, one study in the Netherlands showed that several mastitis preventive measures had a positive net benefit (Hogeveen *et al.*, 2011) – meaning the money spent on them was less than the money saved from prevented mastitis cases. Farmers are encouraged to view spending on mastitis control as an investment with tangible financial returns in addition to improving animal well-being.

Conclusion

Bovine mastitis continues to be a pervasive challenge in dairy farming, but advances in knowledge and management practices have significantly improved our ability to combat this disease. In this review, we have highlighted the current state of knowledge on mastitis, from its etiology and risk factors to diagnostic innovations, control strategies, and economic implications. Several key themes emerge from this overview:

Firstly, **mastitis is a multifactorial disease**. Its occurrence is influenced by a complex interplay of pathogen exposure, cow immunity, and environmental conditions. The primary causative agents are a variety of bacteria – notably *Staphylococcus aureus*, *Streptococcus agalactiae*, *Streptococcus uberis*, *Escherichia coli*, among others – with different modes of transmission and clinical patterns. Understanding whether a case stems from a contagious pathogen or an

environmental source is critical, as it dictates the control approach. Meanwhile, host factors like stage of lactation and parity, as well as farm management practices, strongly determine a herd's mastitis incidence. This underscores that no single “fix” will eradicate mastitis; rather, a **comprehensive herd management approach** is required, addressing all angles from hygiene to cow comfort to therapy.

Secondly, great strides have been made in **mastitis detection and monitoring**. Farmers and veterinarians now have tools at their disposal that previous generations did not, such as rapid milk culturing techniques, PCR-based pathogen identification, and automated sensors that monitor milk and cow parameters in real time. These innovations enable earlier and more targeted interventions. For example, identifying the causative organism allows for more judicious use of antibiotics (treating only when likely effective) and for specific control measures (like segregation if *Mycoplasma* is found). The continuing development of diagnostic biomarkers – like acute phase proteins and differential cell counts – and on-farm devices will further enhance our ability to detect subclinical cases before they flare into clinical problems. Future research in this area, including point-of-care tests and perhaps cow-side biosensors, is likely to make mastitis management even more proactive.

Thirdly, **preventive management remains the cornerstone** of mastitis control. The adage “prevention is better than cure” holds true; it is far more effective and economical to prevent new mastitis infections than to treat established ones. Time-tested practices such as teat disinfection after milking, proper milking routines, maintaining clean and dry bedding, and blanket dry cow therapy (or selective therapy combined with teat sealants) have dramatically lowered mastitis rates in herds that rigorously apply them. These measures, augmented by genetic selection for mastitis resistance and vaccine use where applicable, form a multi-layered defense system against mastitis. The concept of “herd immunity” to mastitis is not as straightforward as for viral diseases, but increasing overall resistance through breeding and possibly immunization will gradually tilt the balance in favor of the cow. Additionally, educating farm personnel and establishing standard operating procedures ensures consistency in applying these preventive measures daily. The **success stories** from countries that have implemented national mastitis control programs (like the Nordics) illustrate that with concerted effort, mastitis levels can be pushed down significantly – benefitting farmers through higher milk quality and yield, and benefitting cows through better health.

Fourthly, given the rise of antimicrobial-resistant bacteria and societal concern about antibiotic use, there is a strong impetus to explore and adopt **alternatives to antibiotics** in mastitis treatment. Our review touched on promising avenues like

plant-based therapies, bacteriophages, and immune modulators. While these are not yet mainstream, they represent important research frontiers. In the coming years, we can expect more refined approaches such as selective therapy protocols (treating only those cases that need treatment, and doing so with the narrowest effective spectrum) and possibly combination approaches (e.g., using a phage or biofilm enzyme along with a reduced antibiotic dose to clear a chronic infection). The **post-antibiotic era** does not imply ceasing all antibiotic use – rather, it envisions a scenario where preventive practices keep mastitis incidence low and where treatment, when needed, is precise and responsible (El-Sayed & Kamel, 2021). Achieving this will require continued research and field validation of alternative therapies, as well as enhancements in vaccine efficacy for mastitis pathogens (Rainard *et al.*, 2022). It will also require farmers and veterinarians to embrace a mindset of antimicrobial stewardship. Economically, the incentive to control mastitis is clear: the disease incurs substantial costs, but most of these are avoidable with known interventions. The economic analyses consistently show that investments in mastitis control yield favorable returns by reducing milk losses and other costs. As milk pricing systems increasingly reward quality (low SCC, no residues) and as consumers demand high standards of animal welfare, maintaining excellent udder health aligns with both profitability and public expectations.

References:

- Ajose, D. J., Oluwarinde, B. O., Abolarinwa, T. O., *et al.* (2022). Combating bovine mastitis in the dairy sector in an era of antimicrobial resistance: Ethno-veterinary medicinal option as a viable alternative approach. *Frontiers in Veterinary Science*, 9, 800322. DOI: 10.3389/fvets.2022.800322
- Ashraf, A., & Imran, M. (2020). Causes, types, etiological agents, prevalence, diagnosis, treatment, prevention, effects on human health and future aspects of bovine mastitis. *Animal Health Research Reviews*, 21(1), 36–49. DOI: 10.1017/S1466252319000094
- Akinboye, A. O., Adeyemo, R. O., Karzis, J., Petzer, I. M., & McGaw, L. J. (2024). Susceptibility patterns of *Escherichia coli* and streptococcal isolates from bovine mastitis cases to antibiotics and selected South African plant extracts with known antibacterial activities. *South African Journal of Botany*, 166, 14–26.
- Abebe, R., Hatiya, H., Abera, M., Megersa, B., & Asmare, K. (2016). Bovine mastitis: prevalence, risk factors and isolation of *Staphylococcus aureus* in dairy herds at Hawassa milk shed, South Ethiopia. *BMC veterinary research*, 12(1), 270.
- Bardhan, D. (2013). Estimates of economic losses due to clinical mastitis in organized dairy farms. *Indian Journal of Dairy Science*, 66(2), 168–172.
- Biffa, D., Debela, E., & Beyene, F. (2005). Prevalence and risk factors of mastitis in lactating dairy cows in Southern Ethiopia. *International Journal of Applied Research in Veterinary Medicine*, 3(3), 189–198.
- Barkema, H. W., Schukken, Y. H., Lam, T. J. G. M., Galligan, D. T., Beiboer, M. L., & Brand, A. (1997). Estimation of interdependence among quarters of the bovine udder with subclinical mastitis and implications for analysis. *Journal of Dairy Science*, 80(8), 1592–1599.
- Cheng, W. N., & Han, S. G. (2020). Bovine mastitis: risk factors, therapeutic strategies, and alternative treatments – a review. *Asian-Australasian Journal of Animal Sciences*, 33(11), 1699–1713. DOI: 10.5713/ajas.20.0156
- Chandrasekaran, D., Nambil, A. P., Thirunavukkarasu, P. S., Venkatesan, P., Tirumurugan, K. G., & Vairamuthu, S. (2015). Incidence of resistant mastitis in dairy cows in Tamil Nadu, India. *J. Appl. Nat. Sci*, 7(1), 304–308.
- Dasohari, A., Somasani, A., Nagaraj, P., & Reddy, G. A. (2018). Comparative studies for efficacy of different diagnostic tests of sub-clinical mastitis in cows. *The Pharma Innovation Journal*, 7(4), 149–152.
- El-Sayed, A., & Kamel, M. (2021). Bovine mastitis prevention and control in the post-antibiotic era. *Tropical Animal Health and Production*, 53(2), 236. DOI: 10.1007/s11250-021-02680-9
- Fazal, M. A., Rana, E. A., Akter, S., Alim, M. A., Barua, H., & Ahad, A. (2023). Molecular identification, antimicrobial resistance and virulence gene profiling of *Staphylococcus* spp. associated with bovine sub-clinical mastitis in Bangladesh. *Veterinary and Animal Science*, 21, 100297.
- Farabi, A. A., Hossain, H., Brishty, K. A., Rahman, M. H., Rahman, M., Siddiqui, M. S. I., ... & Zowlaty, M. E. E. (2025). Prevalence, risk factors, and antimicrobial resistance of *Staphylococcus* and *Streptococcus* species isolated from subclinical bovine mastitis. *Foodborne Pathogens and Disease*, 22(7), 467–476.
- Fesseha, H., Mathewos, M., Aliye, S., & Wolde, A. (2021). Study on prevalence of bovine mastitis and associated risk factors in dairy farms of Modjo town and suburbs, central Oromia, Ethiopia. *Veterinary Medicine: Research and Reports*, 271–283.
- Gomes, F., & Henriques, M. (2016). Control of bovine mastitis: old and recent therapeutic approaches. *Current microbiology*, 72(4), 377–382.

- Hossain, M. K., Paul, S., Hossain, M. M., Islam, M. R., & Alam, M. G. S. (2017). Bovine mastitis and its therapeutic strategy doing antibiotic sensitivity test. *Austin J Vet Sci Anim Husb*, 4(1), 1030.
- Halasa, T., Huijps, K., Østerås, O., & Hogeveen, H. (2007). Economic effects of bovine mastitis and mastitis management: a review. *Veterinary Quarterly*, 29(1), 18–31. DOI: 10.1080/01652176.2007.9695224
- Hogeveen, H., Huijps, K., & Lam, T. J. G. M. (2011). Economic aspects of mastitis: new developments. *New Zealand Veterinary Journal*, 59(1), 16–23. DOI: 10.1080/00480169.2011.547165
- Hoque, M. N., Das, Z. C., Rahman, A. N. M. A., Haider, M. G., & Islam, M. A. (2018). Molecular characterization of *Staphylococcus aureus* strains in bovine mastitis milk in Bangladesh. *International Journal of Veterinary Science and Medicine*, 6(1), 53–60. DOI: 10.1016/j.ijvsm.2018.03.008
- Haider, A., Ikram, M., Shahzadi, I., & Asif Raza, M. (2023). Bovine mastitis. In *Polymeric Nanoparticles for Bovine Mastitis Treatment* (pp. 49-80). Cham: Springer Nature Switzerland.
- Krömker, V., & Leimbach, S. (2017). Mastitis treatment—Reduction in antibiotic usage in dairy cows. *Reproduction in Domestic Animals*, 52, 21-29.
- Kibebew, K. (2017). Bovine mastitis: A review of causes and epidemiological point of view. *Journal of Biology, Agriculture and Healthcare*, 7(2), 1-14.
- Kour, G., Chandra, M., Kaur, G., Narang, D., Gupta, D. K., Arora, A. K., & Sharma, N. S. (2017). Prevalence of mastitis causing organism and their antibiotic resistance pattern in dairy farms. *Indian Journal of Dairy Science*, 70(5), 491-95.
- Kavithaa, N. V., Rajkumar, N. V., & Manokaran, S. (2020). Constraints in dairy farming: a critical analysis among the dairy farmers of Tamil Nadu. *International Journal of Science, Environment and Technology*, 9(3), 407-412.
- Kayesh, M. E. H., Talukder, M., & Anower, A. K. M. M. (2014). Prevalence of subclinical mastitis and its association with bacteria and risk factors in lactating cows of Barisal district in Bangladesh. *International Journal of Biological Research*, 2(2), 35-38.
- Morales-Ubaldo, A. L., Rivero-Perez, N., Valladares-Carranza, B., et al. (2023). Bovine mastitis, a worldwide impact disease: Prevalence, antimicrobial resistance, and viable alternative approaches. *Veterinary and Animal Science*, 21, 100306. DOI: 10.1016/j.vas.2023.100306
- Mekibib, B., Furgasa, M., Abunna, F., Megersa, B., & Regassa, A. (2010). Bovine mastitis: Prevalence, risk factors and major pathogens in dairy farms of Holeta Town, Central Ethiopia. *Veterinary world*, 3(9), 397-403.
- Nielsen, C. (2009). *Economic impact of mastitis in dairy cows* (No. 2009: 29).
- Paramasivam, R., & Gopal, D. R. (2023). Is antimicrobial resistance in dairy products a threat to human health? An updated review on the origin, prevention, treatment, and economic impacts of subclinical mastitis. *Veterinary Medicine: Research and Reports*, 14, 117–142. DOI: 10.2147/VMRR.SXXXXX (Open Access Dove Press)
- Pedersen, R. R., Krömker, V., Bjarnsholt, T., Dahl-Pedersen, K., Buhl, R., & Jørgensen, E. (2021). Biofilm research in bovine mastitis. *Frontiers in veterinary science*, 8, 656810.
- Panchal, J., Patel, A., Patel, S., & Goswami, D. (2024). Understanding mastitis: Microbiome, control strategies, and prevalence—A comprehensive review. *Microbial pathogenesis*, 187, 106533.
- Paape, M. J., Wergin, W. P., Guidry, A. J., & Pearson, R. E. (1979). Leukocytes—second line of defense against invading mastitis pathogens. *Journal of Dairy Science*, 62(1), 135-153.
- Pyörälä, S. (2009). Treatment of mastitis during lactation. *Irish Veterinary Journal*, 62(Suppl 4), S40.
- Rainard, P., Gilbert, F. B., Martins, R. P., Germon, P., & Foucras, G. (2022). Progress towards the elusive mastitis vaccines. *Vaccines*, 10(2), 296. DOI: 10.3390/vaccines10020296
- Ruegg, P. L. (2017). A 100-year review: Mastitis detection, management, and prevention. *Journal of Dairy Science*, 100(12), 10381–10397. DOI: 10.3168/jds.2017-13023
- Rajarajan, G. (2020). Clean milk production practices and its impact on the prevention and control of mastitis in Tiruchirapalli district of Tamil Nadu. *Indian Journal of Dairy Science*, 73(5).
- Stanek, P., Żółkiewski, P., & Januś, E. (2024). A review on mastitis in dairy cows research: current status and future perspectives. *Agriculture*, 14(8), 1292. DOI: 10.3390/agriculture14081292
- Sharun, K., Dhama, K., Tiwari, R., Gugjoo, M. B., Iqbal Yatoo, M., Patel, S. K., ... & Chaicumpa, W. (2021). Advances in therapeutic and managerial approaches of bovine mastitis: a comprehensive review. *Veterinary Quarterly*, 41(1), 107-136.

- Singh, P. J., & Singh, K. B. (1994). A study of economic losses due to mastitis in India.
- Sserunkuma, P., McGaw, L. J., Nsahlai, I. V., & Van Staden, J. (2017). Selected southern African medicinal plants with low cytotoxicity and good activity against bovine mastitis pathogens. *South African journal of botany*, 111, 242-247.
- Senthilkumar, V., Safiullah, A. M., Kathiravan, G., Subramanaian, M., & Mani, K. (2016). Economic Losses due to Milk Fever in Dairy Farms in Tamil Nadu.
- Sekiya, T., Yamaguchi, S., & Iwasa, Y. (2020). Bovine mastitis and optimal disease management: Dynamic programming analysis. *Journal of theoretical biology*, 498, 110292.
- Santman-Berends, I. M. G. A., Lam, T. J. G. M., Keurentjes, J., & Van Schaik, G. (2015). An estimation of the clinical mastitis incidence per 100 cows per year based on routinely collected herd data. *Journal of dairy science*, 98(10), 6965-6977.
- Thirunavukkarasu, M., Kathiravan, G., Kalaikannan, A., & Jebarani, W. (2010). Quantifying economic losses due to milk fever in dairy farms. *Agricultural Economics Research Review*, 23(1), 77-82.
- Tomanić, D., Samardžija, M., & Kovačević, Z. (2023). Alternatives to antimicrobial treatment in bovine mastitis therapy: a review. *Antibiotics*, 12(4), 683.
- Touza-Otero, L., Landin, M., & Diaz-Rodriguez, P. (2024). Fighting antibiotic resistance in the local management of bovine mastitis. *Biomedicine & Pharmacotherapy*, 170, 115967.
- Thangadurai, R., Venilla, M. A., & Shanmugam, P. S. (2017). Management of mastitis in dairy cattle using herbal combination. *Journal of Krishi Vigyan*, 5(2), 164-167.
- Tommasoni, C., Fiore, E., Lisuzzo, A., & Ganesella, M. (2023). Mastitis in dairy cattle: on-farm diagnostics and future perspectives. *Animals*, 13(15), 2538.
- Tezera, M., & Aman Ali, E. (2021). Prevalence and associated risk factors of Bovine mastitis in dairy cows in and around Assosa town, Benishangul-Gumuz Regional State, Western Ethiopia. *Veterinary Medicine and Science*, 7(4), 1280-1286.
- Wani, S. A., Haq, U., Parray, O. R., Nazir, Q. U. A., Mushtaq, M., Bhat, R. A., ... & Yattoo, M. I. (2022). A brief analysis of economic losses due to mastitis in dairy cattle. *Indian Vet. J.*, 90, 27-31.
- Wang, L., Yang, F., Wei, X. J., Luo, Y. J., Guo, W. Z., Zhou, X. Z., & Guo, Z. T. (2019). Prevalence and risk factors of subclinical mastitis in lactating cows in Northwest China. *Isr J Vet Med*, 74(1), 17-22.
- Ucella-Filho, J. G. M., Ferreira, N. S., Alves, M. R., Ignacchiti, M. D. C., Júnior, A. F. D., & Resende, J. A. (2024). Evaluation of natural products as therapeutic alternatives for bovine mastitis and implications for future research. *South African Journal of Botany*, 167, 310-321.