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Effects of subclinical mastitis on physicochemical, nutritional, and technological properties of bovine milk: A systematic review

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Abstract

Subclinical mastitis (SCM) is one of the most prevalent and economically important diseases affecting dairy cattle worldwide, causing substantial losses in milk quality and processing efficiency despite the absence of visible clinical signs. This systematic review synthesizes peer-reviewed evidence published from 2000 onwards to evaluate the effects of bovine SCM on the physicochemical, nutritional, and technological properties of milk and to assess its broader implications for dairy production, processing, and food safety. The reviewed literature demonstrates that SCM disrupts the integrity of the blood-milk barrier, resulting in elevated somatic cell count, increased milk pH and electrical conductivity, altered ionic balance, and reduced lactose concentration. These inflammatory changes impair mammary secretory function and modify the synthesis and composition of major milk constituents. Although total protein content may remain relatively stable, SCM reduces casein synthesis and promotes extensive proteolysis through activation of endogenous enzymes, thereby compromising protein functionality. Simultaneously, alterations in mineral composition, enhanced lipolysis, and increased oxidative stress reduce the nutritional quality and stability of milk. These compositional changes significantly impair technological performance, leading to prolonged coagulation time, reduced curd firmness, lower cheese yield, delayed fermentation, increased syneresis in cultured products, diminished heat stability, and shortened shelf life of dairy products. Beyond its effects on milk quality, SCM imposes considerable economic burdens through decreased milk production, reduced manufacturing efficiency, increased management costs, and loss of quality-based incentives. Furthermore, concerns regarding zoonotic pathogens, bacterial toxins, antimicrobial residues, and antimicrobial resistance highlight the importance of SCM within the One Health framework. Overall, the evidence establishes SCM as a major determinant of milk quality, processing functionality, and dairy industry profitability, emphasizing the need for early detection, precision monitoring, and integrated management strategies to improve udder health and ensure sustainable dairy production.

Keywords: Somatic cell count, casein degradation, rennet coagulation, blood-milk barrier, plasmin activity, cheese yield, dairy chemistry

Introduction

The global dairy sector is a vital component of agricultural economies and human nutrition, providing high-quality proteins, bioactive peptides, essential lipids, and highly bioavailable minerals (Narváez-Semanate, 2022) [42]. The dairy industry depends on the biochemical quality and physical stability of raw bovine milk to ensure efficient processing, product consistency, and desirable sensory characteristics (Darbaz *et al.*, 2023; Bobbo *et al.*, 2017) [16, 13].

However, milk quality is continually challenged by bovine mastitis, an inflammatory disease of the mammary gland commonly caused by bacterial pathogens (Bobbo *et al.*, 2017) [13]. While clinical mastitis presents with visible udder inflammation and obvious milk abnormalities, subclinical mastitis (SCM) remains clinically silent (Contreras, 2007; Olechnowicz, 2014) [14, 45]. Cows affected by SCM produce apparently normal milk from udders without visible lesions, yet experience reduced milk production, altered milk composition, and elevated somatic cell counts (SCC) (Samad, 2022) [60].

Subclinical mastitis is one of the most prevalent diseases in dairy herds, occurring 15-40 times more frequently than clinical mastitis (Darbaz, 2023) [16]. Because infected animals often remain undetected for extended periods, SCM serves as a persistent source of infection within herds and contributes substantially to economic losses in the dairy sector (Samad, 2022; Ndahetuye, 2023) [60, 43]. These losses arise from decreased milk yield, increased veterinary and replacement costs, higher culling rates, and the loss of milk quality incentives offered by processors (Martins, 2020; Antre *et al.*, 2019) [39, 2].

Beyond farm-level losses, SCM significantly affects dairy processing. Milk from affected cows introduces elevated SCC, altered mineral balance, and increased concentrations of heat-stable proteolytic and lipolytic enzymes into the bulk milk supply. These changes impair industrial performance by reducing cheese yield and coagulation efficiency (Politis & Ng-Kwai-Hang, 1988; Auld *et al.*, 1996; Leitner *et al.*, 2004) [52, 4, 37], delaying yogurt fermentation and increasing syneresis (Ma *et al.*, 2000; Barbano *et al.*, 2006) [38, 7], and decreasing the heat stability of milk intended for powder manufacture (Kelly *et al.*, 2006; Santos *et al.*, 2003) [30, 61].

Although numerous studies have investigated the effects of SCM on milk composition and processing characteristics, inconsistencies remain. Some reports describe significant reductions in milk fat and protein concentrations, whereas others report little change or apparent increases due to concentration effects associated with reduced milk volume (Le Maréchal *et al.*, 2011; Bobbo *et al.*, 2017) [34, 13]. In addition, the pathogen-specific effects of SCM on milk proteins, peptides, and cheese-making performance require further clarification (Leitner *et al.*, 2011; Cremonesi *et al.*, 2015) [36, 15].

Therefore, this systematic review synthesizes current peer-reviewed evidence to provide a comprehensive evaluation of the effects of bovine subclinical mastitis on the physicochemical, nutritional, and technological properties of milk, while highlighting key research gaps and future priorities for the dairy industry.

Search Strategy and Review Protocol

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines and the PRISMA-S search reporting framework to ensure transparency, reproducibility, and methodological rigor (Page *et al.*, 2021; Rethlefsen *et al.*, 2021) [50, 57].

Search Strategy

A comprehensive literature search was performed using five major databases: PubMed, Scopus, Web of Science, CAB Abstracts, and AGRICOLA. Additional searches were conducted through ScienceDirect, SpringerLink, Wiley Online Library, Taylor & Francis Online, MDPI, and Google Scholar. Literature published from January 2000 to the present was considered, with particular emphasis on high-quality peer-reviewed studies published after 2015. Database-specific search strategies were developed using controlled vocabulary and relevant keywords combined with Boolean operators (Higgins *et al.*, 2023) [25].

Search string

("Subclinical mastitis" OR "SCM" OR "intramammary infection" OR "udder health") AND ("milk composition" OR

"milk quality" OR "physicochemical properties" OR "nutritional composition" OR "technological properties" OR "cheese yield" OR "coagulation properties" OR "rennet coagulation time" OR "curd firmness" OR "heat stability" OR "yogurt fermentation" OR "somatic cell count" OR "SCC") AND ("bovine" OR "cow" OR "dairy cattle")

Eligibility Criteria

Predefined eligibility criteria were established to ensure the inclusion of scientifically robust studies (Higgins *et al.*, 2023; Page *et al.*, 2021) [25, 50].

Inclusion Criteria

Studies were included if they:

- Were peer-reviewed original research articles published in English.
- Investigated naturally occurring or experimentally induced subclinical mastitis in dairy cows.
- Compared healthy animals or quarters (typically SCC <100,000-200,000 cells/mL and negative bacteriological culture) with SCM-affected animals or quarters (SCC ≥200,000 cells/mL and/or positive California Mastitis Test, with or without pathogen isolation) (Ruegg, 2017; IDF, 2013) [58, 27].
- Reported quantitative data on physicochemical, nutritional, or technological properties of milk.

Exclusion Criteria

Studies were excluded if they:

- Focused exclusively on clinical mastitis.
- Investigated non-bovine dairy species.
- Were review articles, editorials, conference abstracts, book chapters, patents, or case reports without original data.
- Contained duplicate datasets or lacked adequate scientific validation (Higgins *et al.*, 2023) [25].

Study Selection Process

After duplicate removal, titles and abstracts were independently screened by two reviewers. Potentially relevant articles underwent full-text evaluation against the predefined eligibility criteria. Any disagreements were resolved through discussion and consensus (Page *et al.*, 2021; Higgins *et al.*, 2023) [50, 25].

Data Extraction

Data were extracted using a standardized template that recorded author, publication year, geographic region, breed, diagnostic methods, SCC thresholds, sample size, and quantitative outcomes related to physicochemical, nutritional, and technological milk properties (Higgins *et al.*, 2023) [25].

Quality Assessment

Methodological quality and risk of bias were assessed using criteria adapted from the *Cochrane Handbook for Systematic Reviews* and the SANRA guidelines (Higgins *et al.*, 2023; Baethge *et al.*, 2019) [25, 5].

Studies were evaluated based on sample size adequacy, accuracy of SCM diagnosis, use of microbiological confirmation, clarity of analytical procedures, and appropriateness of statistical analyses, including adjustment for potential confounding factors such as parity, stage of lactation, and herd effects.

PRISMA Flow Diagram

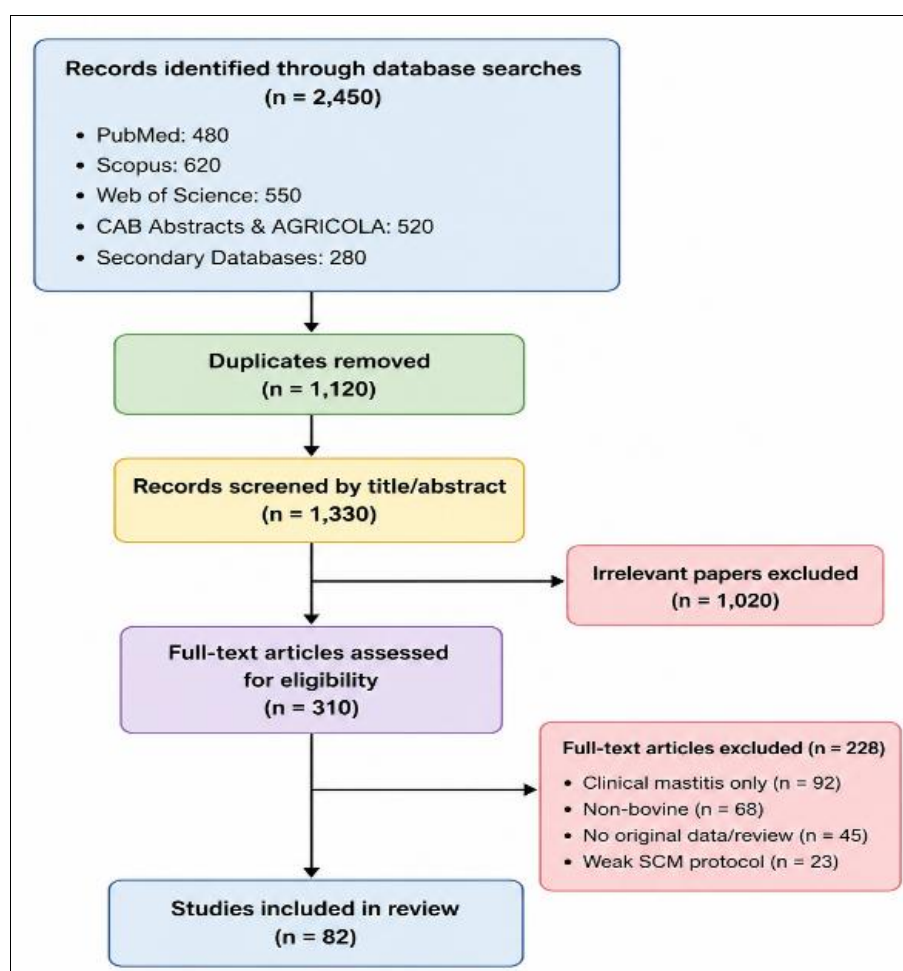


Fig 1: PRISMA 2020 Flow Diagram of Literature Search, Screening, Eligibility Assessment, and Study Inclusion for the Systematic Review

Effects of Subclinical Mastitis on Physicochemical Properties of Milk

The inflammatory processes associated with subclinical mastitis (SCM) induce measurable alterations in the physicochemical properties of bovine milk. These changes primarily result from disruption of tight junctions between mammary epithelial cells, allowing the non-selective transfer of extracellular fluid components and altering milk ionic composition (Leitner *et al.*, 2004; Silanikove *et al.*, 2014) [37, 65].

pH and Titratable Acidity

Healthy bovine milk is slightly acidic, with a normal pH ranging from 6.59 to 6.68. This acidity is maintained by natural buffering components, including caseins, dihydrogen phosphate (H_2PO_4^-), and citric acid (Fox *et al.*, 2015; Walstra *et al.*, 2006) [20, 70].

Numerous studies have reported a significant increase in milk pH during SCM, often reaching values between 6.69 and 7.07. This increase results from impaired secretion of acidifying constituents due to mammary epithelial damage and the influx of blood-derived sodium bicarbonate (NaHCO_3) and chloride (Cl^-) ions into the alveolar lumen (Kitchen, 1981; Leitner *et al.*, 2004; Sharma *et al.*, 2011) [31, 37, 64].

Conversely, titratable acidity decreases significantly during SCM. Normal bovine milk typically exhibits a titratable acidity of approximately 0.14% lactic acid, which may decline to about 0.105% in milk from affected quarters. This

reduction reflects losses of caseins and phosphate ions that contribute to the natural buffering capacity of milk (Batavani *et al.*, 2007) [9].

Density and Specific Gravity

The specific gravity and density of normal bovine milk generally range from 1.029 to 1.034 g/cm^3 and are largely determined by solids-not-fat (SNF) and fat content (Walstra *et al.*, 2006; Fox *et al.*, 2015) [70, 20].

During SCM, specific gravity decreases significantly, often approaching 1.024 g/cm^3 . This reduction is mainly attributed to decreased lactose and casein synthesis, resulting in lower SNF content and reduced milk density (Batavani *et al.*, 2007; Bansal *et al.*, 2005) [9, 6].

Electrical Conductivity

Electrical conductivity (EC) reflects the concentration and mobility of free ions in milk. In healthy milk, EC typically ranges from 4.0 to 5.5 mS/cm at 20°C because most calcium and magnesium remain bound within casein micelles, leaving relatively low concentrations of free conductive ions in the serum phase (Norberg *et al.*, 2004; Walstra *et al.*, 2006) [44, 70]. Subclinical mastitis causes a marked increase in EC, often exceeding 6.0 mS/cm . Inflammatory damage elevates sodium (Na^+) and chloride (Cl^-) concentrations while reducing potassium (K^+) levels. The increased abundance of highly mobile Na^+ and Cl^- ions enhances the electrical conductivity of milk (Kitchen, 1981; Norberg *et al.*, 2004; Pyörälä, 2003) [31, 44, 54].

Because of its rapid response to mammary inflammation, EC is widely used as a real-time indicator in automated and robotic milking systems for early detection of subclinical mastitis (Norberg *et al.*, 2004; Hovinen & Pyörälä, 2011) [44, 26].

Freezing Point

The freezing point depression (FPD) of bovine milk is a highly stable physical parameter, normally ranging from -0.520°C to -0.540°C . It is primarily regulated by lactose and dissolved minerals to maintain osmotic equilibrium with blood plasma (Fox *et al.*, 2015; Walstra *et al.*, 2006) [20, 70].

During SCM, lactose synthesis declines markedly, reducing its osmotic contribution. To maintain osmotic balance, sodium and chloride ions move from blood into milk. Although total osmotic pressure remains relatively constant, this shift in osmolyte composition alters the freezing point, which may move closer to zero under severe inflammatory conditions (Auldist & Hubble, 1998; Leitner *et al.*, 2004) [3, 37].

Viscosity

Milk viscosity is influenced by the concentration and size of fat globules, casein micelles, and somatic cells. Under normal conditions, bovine milk behaves largely as a Newtonian fluid; however, SCM can alter its rheological characteristics (Walstra *et al.*, 2006) [70].

The elevated somatic cell count associated with SCM introduces increased quantities of cellular proteins and high-molecular-weight DNA into milk, particularly following leukocyte lysis. Together with protein aggregation, this cellular debris can modify milk flow properties and increase viscosity (Silanikove *et al.*, 2014; Leitner *et al.*, 2004) [65, 37].

This phenomenon forms the basis of the California Mastitis Test (CMT), in which detergent-induced lysis of somatic cells releases DNA that produces a characteristic gel. The degree of gel formation is directly proportional to leukocyte concentration and serves as a practical indicator of subclinical inflammation (Schalm & Noorlander, 1957; Sharma *et al.*, 2011) [62, 64].

Milk Yield

Subclinical mastitis is consistently associated with reduced milk yield despite the absence of visible clinical signs. Production losses result from damage to secretory alveolar tissue caused by invading pathogens and their toxins, which reduces mammary secretory capacity. Additionally, the inflammatory response increases the demand for energy and glucose, thereby competing with lactose synthesis, the principal osmotic regulator of milk volume (Seegers *et al.*, 2003; Halasa *et al.*, 2007) [63, 22].

The extent of milk loss depends on the causative pathogen and duration of infection. Heikkilä *et al.* (2018) reported significant reductions in milk yield associated with subclinical infections caused by *Staphylococcus aureus* and *Streptococcus uberis*. Similarly, Gonçalves *et al.* (2020) [21] demonstrated that chronic SCM substantially reduced both milk production and total solids yield at the cow level. These findings highlight the considerable economic impact of persistent intramammary infections on dairy herd productivity (Gonçalves *et al.*, 2020) [21].

Fat, Lipid, and Oxidative Alterations

The effects of subclinical mastitis (SCM) on milk fat concentration are inconsistent. Several studies have reported

reductions in milk fat percentage due to impaired fatty acid synthesis by mammary epithelial cells and decreased uptake of blood-derived triglycerides (Auldist *et al.*, 1995; Bobbo *et al.*, 2017). In contrast, other studies have observed little change or slight increases in fat percentage, largely as a concentration effect resulting from reduced milk volume rather than increased fat synthesis (Safak *et al.*, 2023; Leitner *et al.*, 2004) [59, 37].

Beyond changes in fat concentration, SCM substantially alters lipid composition and stability. Inflammatory processes stimulate the release of lipolytic enzymes, particularly lipoprotein lipases derived from somatic cells and pathogenic bacteria, which hydrolyze triglycerides into free fatty acids (FFAs) and diacylglycerols. This enhanced lipolysis increases susceptibility to rancidity, off-flavors, and deterioration during storage (Ma *et al.*, 2000; Kelly *et al.*, 2006; Le Maréchal *et al.*, 2011) [38, 30, 34].

Subclinical mastitis is also associated with increased oxidative stress within the mammary gland. Activated polymorphonuclear neutrophils generate reactive oxygen species (ROS) as part of the immune response against invading pathogens. During persistent intramammary infections, ROS production may exceed the antioxidant capacity of mammary tissues, resulting in oxidative damage (Sordillo & Aitken, 2009; Turk *et al.*, 2017) [66, 69].

Oxidative stress promotes lipid peroxidation of milk fat globule membranes, accelerates fatty acid deterioration, and contributes to rancid flavors and reduced shelf life of dairy products. In addition, oxidative modifications of milk proteins, particularly caseins, can impair protein functionality, stability, and processing characteristics, further reducing milk quality (Bernabucci *et al.*, 2005; Sordillo & Aitken, 2009; Sharma *et al.*, 2011) [10, 66, 64].

Evidence also suggests that improving antioxidant status through dietary supplementation, including antioxidant nutrients and rumen-protected methionine, may help alleviate oxidative stress, support mammary tissue integrity, and improve lactational performance in dairy cows exposed to inflammatory challenges (Osorio *et al.*, 2013; Zhou *et al.*, 2016) [48, 72].

Total Protein, Casein, and Whey Fractions

Total crude protein concentration often remains unchanged or may increase slightly during SCM; however, this apparent stability masks substantial deterioration in protein quality and functionality (Auldist *et al.*, 1995; Bobbo *et al.*, 2017).

Milk proteins are broadly classified into caseins and whey proteins. Caseins (α s1-, α s2-, β -, and κ -casein) are synthesized within the mammary gland and play a critical role in dairy processing, whereas whey proteins include α -lactalbumin, β -lactoglobulin, immunoglobulins, and bovine serum albumin (Fox *et al.*, 2015) [20].

Concentrations of bovine serum albumin and immunoglobulins increase markedly because of enhanced vascular permeability and leakage of serum proteins into mammary secretions (Leitner *et al.*, 2004; Pyörälä, 2003) [37, 54]. In contrast, α -lactalbumin and β -lactoglobulin concentrations decline, reflecting reduced synthetic activity of damaged mammary epithelial cells (Leitner *et al.*, 2004; Auldist *et al.*, 1995).

Lactose

Lactose is the principal carbohydrate of milk and the primary osmotic regulator of milk secretion. Under normal conditions, its concentration remains relatively stable because of

continuous synthesis within the Golgi apparatus of mammary epithelial cells (Fox *et al.*, 2015; Walstra *et al.*, 2006) [20, 70].

A decline in lactose concentration is one of the most consistent compositional changes associated with SCM. This reduction results from impaired lactose synthesis, damage to mammary epithelial cells, leakage of lactose into the bloodstream through disrupted tight junctions, and bacterial utilization of lactose within the infected mammary gland (Kitchen, 1981; Leitner *et al.*, 2004; Silanikove *et al.*, 2014) [31, 37, 65].

Total Solids and Solids-Not-Fat (SNF)

Total solids (TS) and solids-not-fat (SNF) constitute the major dry matter components of milk. Because SCM reduces lactose synthesis, casein production, and overall mammary secretory activity, both TS and SNF generally decline during infection (Auldist *et al.*, 1995; Bansal *et al.*, 2005; Bobbo *et al.*, 2017). These reductions diminish the nutritional value of milk and adversely affect processing efficiency and product yield during the manufacture of cheese, yogurt, and milk powder (Le Maréchal *et al.*, 2011) [34].

Ash and Mineral Profile

The mineral composition of milk is highly sensitive to mammary gland health. Subclinical mastitis alters both macro- and micro-mineral concentrations through changes in epithelial permeability and mammary secretory function (Kitchen, 1981; Leitner *et al.*, 2004) [31, 37].

Calcium, Phosphorus, and Magnesium

In healthy milk, calcium, phosphorus, and magnesium are predominantly associated with casein micelles. Reduced casein synthesis and increased proteolytic degradation during SCM decrease the concentrations of these minerals (Auldist *et al.*, 1995; Leitner *et al.*, 2004; Fox *et al.*, 2015).

The resulting mineral depletion lowers the nutritional value of milk and destabilizes its colloidal structure, thereby impairing coagulation characteristics and overall processing performance (Politis & Ng-Kwai-Hang, 1988; Le Maréchal *et al.*, 2011) [52, 34].

Sodium, Potassium, and Chloride

The concentrations of sodium, potassium, and chloride are normally maintained by active transport mechanisms within mammary epithelial cells (Kitchen, 1981) [31].

Disruption of the blood-milk barrier during SCM permits passive exchange between blood plasma and milk, leading to increased sodium and chloride concentrations and decreased potassium levels (Kitchen, 1981; Pyörälä, 2003; Leitner *et al.*, 2004) [31, 54, 37].

This characteristic ionic shift contributes to the salty taste of mastitic milk and is largely responsible for the elevated electrical conductivity observed during intramammary infections (Norberg *et al.*, 2004; Hovinen & Pyörälä, 2011) [44, 26].

Trace Minerals and Total Ash

Trace minerals, including zinc, iron, and copper, are also influenced by mammary inflammation. During infection, host defense mechanisms alter the distribution of these micronutrients as part of nutritional immunity, thereby limiting their availability to invading pathogens (Moyes *et al.*, 2009; Kehrli & Shuster, 1994) [41, 28].

Experimental *E. coli* intramammary infections have demonstrated significant alterations in zinc, iron, and copper

metabolism associated with the acute-phase inflammatory response (Moyes *et al.*, 2009) [41].

Biological Mechanisms Underlying Milk Composition Changes

The physicochemical and nutritional alterations observed during SCM are direct consequences of inflammatory processes and tissue damage within the mammary gland (Sordillo & Streicher, 2002; Pyörälä, 2003) [67, 54].

Inflammatory Mechanisms, Blood-Milk Barrier Dysfunction, and Altered Lactose Synthesis

The compositional changes observed during subclinical mastitis (SCM) originate from inflammatory processes within the mammary gland. The disease is initiated when pathogenic microorganisms enter through the teat canal and activate host immune responses through bacterial components such as lipopolysaccharides (LPS) and peptidoglycans, triggering the release of inflammatory mediators and cytokines (Rainard & Riollet, 2006; Wellnitz & Bruckmaier, 2012) [55, 71].

These inflammatory signals recruit polymorphonuclear neutrophils (PMNs) from the bloodstream, resulting in a marked increase in somatic cell count (SCC). During mastitis, PMNs may account for more than 90% of somatic cells present in milk. Although essential for pathogen elimination, their activity contributes to tissue damage and impaired mammary secretory function, thereby exacerbating alterations in milk composition and quality (Paape *et al.*, 2002; Rainard & Riollet, 2006; Sordillo & Streicher, 2002) [49, 55, 67].

Inflammation also disrupts the integrity of mammary epithelial tight junctions, compromising the blood-milk barrier and increasing vascular permeability (Stelwagen *et al.*, 1997; Wellnitz & Bruckmaier, 2012) [68, 71]. As a result, sodium, chloride, serum albumin, immunoglobulins, haptoglobin, serum amyloid A, and other blood-derived constituents diffuse into milk, whereas lactose and potassium leak into the bloodstream. These exchanges are largely responsible for the physicochemical and nutritional alterations characteristic of SCM milk (Kitchen, 1981; Pyörälä, 2003; Leitner *et al.*, 2004; Eckersall *et al.*, 2006) [31, 54, 37, 19].

The decline in lactose concentration, one of the most consistent changes associated with SCM, is closely linked to mammary epithelial damage and impaired secretory activity. Lactose is synthesized from glucose within mammary epithelial cells through the lactose synthetase complex, and disruption of this process, together with leakage of lactose through damaged tight junctions, contributes to reduced lactose levels and impaired milk secretion during inflammation (Akers & Nickerson, 2011; Fox *et al.*, 2015; Leitner *et al.*, 2004) [1, 20, 37].

Proteolysis, Lipolysis, and Enzymatic Degradation

One of the most significant biochemical consequences of SCM is the acceleration of proteolytic activity within milk, leading to the degradation of functionally important proteins. This process is primarily mediated by activation of the endogenous plasminogen-plasmin system. Plasmin, a heat-stable serine protease naturally present in milk as inactive plasminogen associated with casein micelles, becomes increasingly activated during SCM. Enhanced release of tissue plasminogen activator (t-PA) and urokinase-type plasminogen activator (u-PA) from somatic and damaged epithelial cells promotes the conversion of plasminogen into active plasmin, thereby increasing proteolytic activity (Politis, 1996; Kelly & McSweeney, 2003; Le Roux *et al.*, 2003) [51, 29].

35].

Active plasmin preferentially hydrolyzes β -casein and α s1-casein, generating proteose-peptones and low-molecular-weight peptides, whereas κ -casein is comparatively less susceptible. In parallel, lysosomal proteases such as cathepsins released from somatic cells further contribute to protein degradation. These processes destabilize casein micelles, impair coagulation properties, reduce cheese yield, and promote the formation of bitter peptides that adversely affect milk flavor and product quality (Politis & Ng-Kwai-Hang, 1988; Kelly & McSweeney, 2003; Le Maréchal *et al.*, 2011) [52, 29, 34].

Subclinical mastitis also enhances lipolytic activity within milk. Under normal conditions, the milk fat globule membrane (MFGM) protects triglycerides from enzymatic hydrolysis. However, inflammatory damage and microbial phospholipases compromise MFGM integrity, allowing endogenous lipoprotein lipase and bacterial lipases to access triglycerides and hydrolyze them into free fatty acids (FFAs) and glycerol (Deeth, 2006; Ma *et al.*, 2000; Le Maréchal *et al.*, 2011) [18, 38, 34].

The accumulation of FFAs accelerates rancidity, impairs sensory quality, and reduces the storage stability of milk and dairy products. Consequently, increased lipolysis is a major contributor to the reduced technological and commercial value of SCM-affected milk (Deeth, 2006; Santos *et al.*, 2003) [18, 61].

Two principal mechanisms contribute to reduced lactose synthesis during SCM

Host-Pathogen Competition for Glucose: Intramammary pathogens utilize glucose as a major substrate for growth and replication. Simultaneously, activated immune cells require substantial amounts of glucose to support phagocytosis, respiratory burst activity, and cytokine production. This competition reduces glucose availability for lactose synthesis by mammary epithelial cells (Wellnitz & Bruckmaier, 2012; Sordillo & Streicher, 2002) [71, 67].

Biosynthetic Failure: Direct epithelial injury caused by bacterial toxins and inflammatory mediators impairs the

synthetic capacity of mammary epithelial cells. Reduced expression of α -lactalbumin, the regulatory component of the lactose synthetase complex, directly limits lactose production. Because lactose is the principal osmotic regulator of milk secretion, reduced lactose synthesis lowers osmotic pressure within the alveolar lumen, decreases water influx, and ultimately reduces milk yield (Akers & Nickerson, 2011; Leitner *et al.*, 2004; Silanikove *et al.*, 2014) [1, 37, 65].

Enzyme Leakage and Inflammatory Biomarkers

Subclinical mastitis promotes the release of intracellular enzymes from damaged mammary epithelial cells and infiltrating leukocytes into milk. Among the most widely studied indicators of mammary tissue damage are N-acetyl- β -D-glucosaminidase (NAGase) and lactate dehydrogenase (LDH) (Kitchen, 1981; Pyörälä, 2003) [31, 54].

NAGase is a lysosomal enzyme present in high concentrations within mammary epithelial cells and neutrophils. During SCM, cellular injury and leukocyte activation result in elevated NAGase activity in milk. Numerous studies have demonstrated strong positive associations between NAGase activity, somatic cell count, and intramammary infection status, supporting its use as a sensitive indicator of subclinical mastitis (Kitchen *et al.*, 1978; Pyörälä, 2003; Hovinen & Pyörälä, 2011) [32, 54, 26].

Similarly, lactate dehydrogenase (LDH), a cytoplasmic enzyme involved in glycolysis, is released into milk following epithelial cell damage and leukocyte disruption. Elevated LDH activity has consistently been associated with increased somatic cell counts, intramammary infection, and the severity of mammary inflammation (Batavani *et al.*, 2010; Pyörälä, 2003) [8, 54].

Because the activities of both NAGase and LDH correlate strongly with tissue damage, inflammatory intensity, and somatic cell count, these enzymes are widely recognized as sensitive and objective biomarkers for the detection of subclinical mastitis and for monitoring the recovery of mammary gland function following infection (Pyörälä, 2003; Hovinen & Pyörälä, 2011) [54, 26].

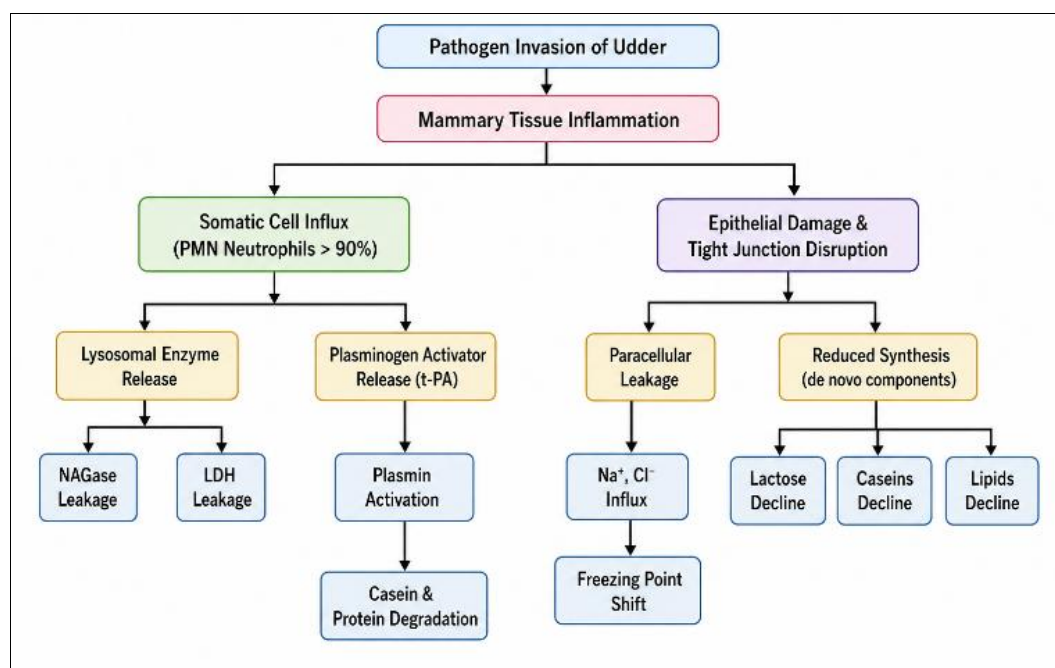


Fig 2: Biological Mechanisms Linking SCM to Changes in Milk Composition

Effects of Subclinical Mastitis on Technological Properties of Milk

The compositional changes associated with subclinical mastitis (SCM) directly impair the technological performance of bovine milk, creating substantial challenges for dairy processing and product manufacture (Auldish & Hubble, 1998; Le Maréchal *et al.*, 2011) [3, 34].

Cheese-Making Properties of SCM Milk

The manufacture of high-quality cheese depends on efficient rennet-induced coagulation of casein micelles, which is influenced by casein concentration, micellar integrity, and mineral balance. Subclinical mastitis (SCM) adversely affects all of these factors through reduced casein synthesis, plasmin-mediated degradation of α s1- and β -caseins, and disturbances in calcium and phosphorus balance, thereby compromising cheese-making performance (Politis & Ng-Kwai-Hang, 1988; Politis, 1996; Kelly & McSweeney, 2003; Leitner *et al.*, 2011; Le Maréchal *et al.*, 2011) [52, 51, 29, 36, 34].

As a result, SCM milk exhibits prolonged rennet coagulation time (RCT), slower curd-firming rate (k_{20}), and reduced curd firmness (a_{30}/a_{60}). Structural disruption of casein micelles,

elevated somatic cell counts, and increased concentrations of blood-derived whey proteins contribute to the formation of weak and fragile curd networks that are more susceptible to breakage during processing (Politis & Ng-Kwai-Hang, 1988; Bobbo *et al.*, 2017; Leitner *et al.*, 2011) [52, 13, 36].

These coagulation defects increase losses of fat and protein into the whey, leading to reduced cheese yield, altered composition, and lower processing efficiency. High-SCC milk has been shown to decrease the recovery of milk solids in cheese curd and increase moisture content, adversely affecting cheese texture, quality, and storage stability (Politis & Ng-Kwai-Hang, 1988; Auldish *et al.*, 1996; Bobbo *et al.*, 2017) [52, 4, 13].

Several studies have demonstrated that milk from SCM-affected quarters produces weaker gels and inferior clotting properties than milk from healthy cows. Furthermore, excessive proteolysis may alter cheese ripening and flavor development, with plasmin-derived peptides contributing to bitterness, texture defects, and reduced sensory acceptability of the final product (Bobbo *et al.*, 2017; Leitner *et al.*, 2011; Kelly & McSweeney, 2003; Le Maréchal *et al.*, 2011) [13, 36, 29, 34].

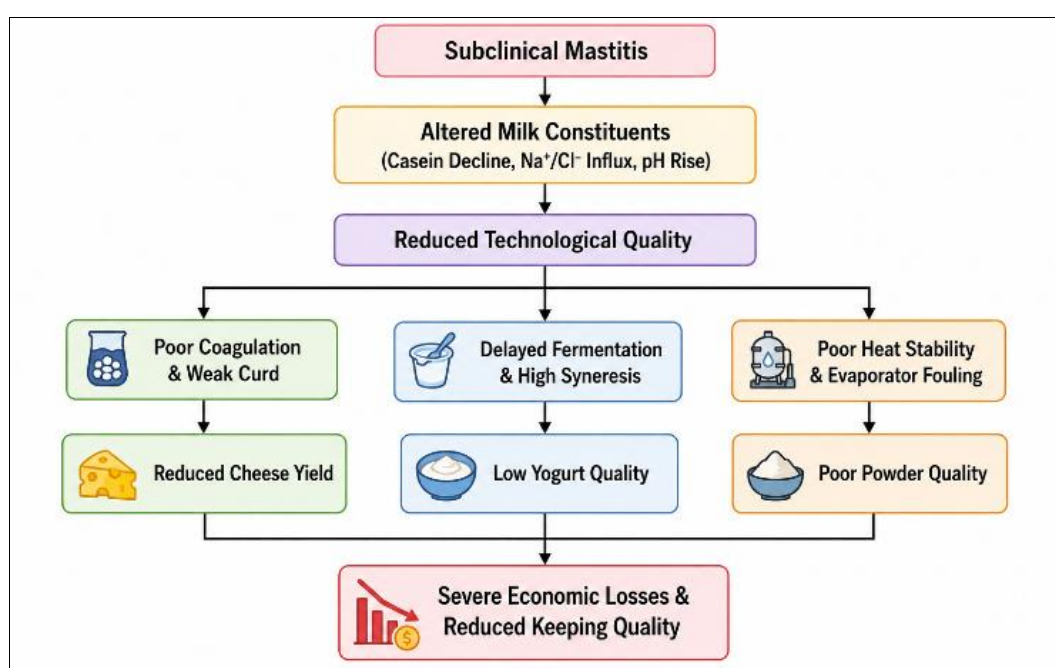


Fig 3: Impact of Subclinical Mastitis on Milk Quality and Processing

Yogurt Production and Fermentation Performance

The manufacture of yogurt and other fermented dairy products is also adversely affected by subclinical mastitis (Ma *et al.*, 2000; Le Maréchal *et al.*, 2011) [38, 34].

Fermentation Kinetics

The growth and acidification activity of yogurt starter cultures (*Lactobacillus delbrueckii* subsp. *bulgaricus* and *Streptococcus thermophilus*) may be delayed in SCM milk. This inhibition is attributed to elevated concentrations of antimicrobial proteins, including lactoferrin, immunoglobulins, lysozyme, and lactoperoxidase, which increase during mammary inflammation and can interfere with starter culture activity (Leitner *et al.*, 2011; Le Maréchal *et al.*, 2011) [36, 34]. Delayed acidification prolongs fermentation time, reducing processing efficiency and potentially affecting product consistency (Le Maréchal *et al.*, 2011) [34].

Product Quality, Texture, and Syneresis

The texture of yogurt depends on the formation of a stable acid-induced protein gel network capable of retaining water. Because SCM milk contains reduced casein concentrations and altered protein composition, the resulting gel network is often weaker and less stable (Auldish & Hubble, 1998; Le Maréchal *et al.*, 2011) [3, 34].

This weakened structure increases susceptibility to syneresis (whey separation), resulting in poorer visual appearance and reduced consumer acceptability. Although some studies have reported increased apparent viscosity in yogurt produced from high-SCC milk due to elevated whey protein concentrations and cellular material, overall texture quality and sensory acceptance are generally inferior to products manufactured from healthy milk (Le Maréchal *et al.*, 2011) [34].

Milk Powder Production and Heat Stability

The production of high-quality milk powders requires raw

milk with excellent thermal stability to withstand concentration and spray-drying processes. Under normal conditions, bovine milk possesses sufficient heat stability to tolerate severe thermal treatments without coagulation (Fox *et al.*, 2015) [20].

During subclinical mastitis, however, milk heat stability declines significantly. Elevated pH, altered mineral balance, increased proteolysis, and higher concentrations of serum-derived proteins contribute to reduced thermal resistance and increased susceptibility to heat-induced coagulation (Kelly *et al.*, 2006; Le Maréchal *et al.*, 2011) [30, 34].

These changes promote fouling of evaporators and heat exchangers, reduce processing efficiency, and negatively affect the solubility and functional quality of milk powders (Kelly *et al.*, 2006; Auldist & Hubble, 1998) [30, 3].

Pathogen Transmission

A variety of bacterial pathogens associated with subclinical mastitis may be shed into milk, including *Staphylococcus aureus*, *Escherichia coli*, *Streptococcus agalactiae*, *Streptococcus uberis*, and *Klebsiella pneumoniae*. Because

milk from subclinically infected quarters usually appears visually normal, contaminated milk may enter bulk tank systems if appropriate monitoring programs are not implemented (Oliver *et al.*, 2005; Ruegg, 2017) [47, 58].

The presence of pathogenic microorganisms in raw milk highlights the importance of effective mastitis control, hygienic milk production practices, and pasteurization for ensuring food safety (Oliver *et al.*, 2009) [46].

Heat-Stable Toxins

Although pasteurization effectively destroys viable bacterial cells, it does not necessarily eliminate all bacterial toxins. Certain strains of *Staphylococcus aureus* produce enterotoxins that are highly resistant to heat treatment and may remain biologically active after pasteurization and other processing operations (Hennekinne *et al.*, 2012; Le Loir *et al.*, 2003) [24, 33].

These staphylococcal enterotoxins are recognized causes of foodborne intoxication and represent a potential food safety concern when milk from infected animals is used in dairy processing (Hennekinne *et al.*, 2012) [24].

Table 1: Characteristics of Included Studies

Authors (Year)	Country	Breed	SCM Diagnostic Method	SCC Classification Used*	Sample Size	Main Outcomes Investigated
Safak <i>et al.</i> (2023)	Turkey	Holstein Friesian	California Mastitis Test (CMT), SCC, microbiological culture	Healthy vs SCM quarters	180 cows (720 quarters)	Effects of SCM pathogens on milk fat, protein, lactose, SNF, mineral profile, electrical conductivity, and specific gravity
Reitz <i>et al.</i> (2024)	Brazil	Jersey	SCC and blood metabolic profiling	Healthy vs SCM cows	59 cows	SCC linear score, lactose, protein, casein, SNF, milk urea nitrogen (MUN), inflammatory biomarkers
Bobbo <i>et al.</i> (2017)	Italy	Holstein, Jersey, Brown Swiss, Alpine Grey	SCC, aseptic bacteriological culture, HPLC protein profiling	Healthy vs SCM cows	1,508 cows (41 herds)	Casein fractions, whey proteins, milk coagulation properties (MCP), cheese-making characteristics
Heikkilä <i>et al.</i> (2018)	Finland	Holstein, Ayrshire	SCC, PCR bacteriology, herd health records	Healthy vs SCM cows	20,234 cows	Pathogen-specific milk yield losses and long-term production effects
Gonçalves <i>et al.</i> (2020)	Brazil	Holstein Friesian	SCC, repeated biweekly sampling, microbiological culture	Healthy vs chronic SCM cows	1,164 dairy records	Long-term effects on milk yield, total solids, and milk component production
Auldist <i>et al.</i> (1996)	Australia	Holstein Friesian	Bulk Milk Cell Count (BMCC), pilot-scale cheese manufacture	Low vs High BMCC milk	Seasonal herd cohorts	Milk protein profile, rennet coagulation, Cheddar cheese yield, sensory characteristics
Politis & Ng-Kwai-Hang (1988)	Canada	Holstein Friesian	SCC and individual cow milk composition analysis	Low vs High SCC milk	80 cows	Casein distribution, whey proteins, coagulation properties, cheese composition

Table 2: Effects of SCM on Physicochemical and Nutritional Properties

Parameter / Constituent	Healthy Control Milk (Typical Baselines)	Subclinical Mastitis Milk (Reported Trends & Values)	Main Underlying Mechanism	Key Direct References
Milk pH	6.59 - 6.68	6.69 - 7.07 (Significant rise)	Influx of blood-derived sodium bicarbonate and loss of natural phosphate/casein buffers.	[cite: 25, 36, 45]
Titrateable Acidity	~0.14% lactic acid	~0.105% lactic acid (Decline)	Decreased concentration of caseins and buffering dihydrogen phosphate ions.	[cite: 25]
Specific Gravity	1.029 - 1.034 g/cm³	1.024 g/cm³ (Significant decline)	Drop in lactose and structural casein concentrations, reducing overall milk density.	[cite: 25, 27]
Electrical Conductivity	4.0 - 5.5 mS/cm	> 6.0 to 11.0 mS/cm (Significant rise)	Influx of mobile <i>Na⁺</i> and <i>Cl⁻</i> ions accompanied by efflux of <i>K⁺</i> ions through leaky tight junctions.	[cite: 41, 46]
Milk Yield	Healthy baseline	4.3% to 24.5% decline	Pathogen-specific tissue destruction of secretory tissue and competition for glucose.	[cite: 8, 40]
Lactose	4.88%	4.14% - 4.37% (Consistently lower)	Impaired biosynthetic function of secretory cells and paracellular leakage into bloodstream.	[cite: 25, 32]
Casein	Baseline healthy levels	6.0% to 20.0% decline	Reduced cellular synthesis and post-secretory degradation by the endogenous plasmin system.	[cite: 20, 26]
Whey Proteins	Baseline levels (mostly)	Significant rise (BSA, IgG)	Disruption of the blood-milk barrier allowing serum	[cite: 36, 45]

	local)		albumin and immunoglobulins to flood raw milk.	
Total Solids / SNF	High solids baseline	Significant decline (TS/SNF drops)	Combined reductions in lactose, caseins, and synthesized lipids, reducing total dry matter.	[cite: 14, 25]
Minerals (Ca^{2+} , Mg^{2+} , P)	High physiological levels	Significant decline	Micellar breakdown and decreased permeability of damaged glandular tissue.	[cite: 25, 36, 45]
Minerals (Na^+ , Cl^-)	Low baseline levels	Significant increase	Passive diffusion of blood electrolytes down their concentration gradient into the alveolar lumen.	[cite: 25, 36, 45]

Research Gaps and Future Directions

While the immediate physicochemical, nutritional, and technological impacts of SCM on bovine milk have been extensively documented, several important knowledge gaps remain. Addressing these limitations will be essential for developing advanced diagnostic tools, effective control strategies, and innovative processing technologies capable of mitigating SCM-associated losses throughout the dairy value chain (Ruegg, 2017; Halasa *et al.*, 2007) ^[58, 22].

Geographic and Environmental Gaps

A substantial proportion of the current literature originates from intensive dairy production systems in Europe and North America. Consequently, information regarding the prevalence, pathogen distribution, and milk compositional changes associated with SCM under tropical, subtropical, arid, and smallholder production systems remains comparatively limited (Motaung *et al.*, 2017; Ruegg, 2017). Environmental stressors, particularly heat stress and elevated Temperature-Humidity Index (THI), are known to influence immune competence, pathogen susceptibility, and milk production. However, the interactions among climatic stress, pathogen-specific intramammary infections, and milk quality deterioration remain insufficiently characterized. Future epidemiological studies should integrate environmental variables, pathogen profiles, and milk quality indicators to improve predictive models of SCM occurrence and severity under changing climatic conditions (Polsky & von Keyserlingk, 2017) ^[53].

Breed-Specific and Genetic Research Gaps

Most published studies investigating SCM-associated milk quality changes have focused on Holstein-Friesian cattle because of their global predominance in commercial dairy systems. However, increasing evidence suggests that breed-specific differences exist in udder health, immune response, milk composition, and technological characteristics (Reitz *et al.*, 2024) ^[56].

Breeds such as Jersey and Brown Swiss produce milk with distinct protein, fat, and mineral profiles that may respond differently to inflammatory challenges. Furthermore, relatively little information is available regarding how genetic variants of milk proteins, particularly κ -casein and β -lactoglobulin polymorphisms, influence coagulation properties and technological resilience during SCM (Reitz *et al.*, 2024) ^[56].

Future research should focus on identifying genomic markers associated with both mastitis resistance and preservation of milk technological quality. Such information could support breeding programs aimed at improving udder health while simultaneously enhancing processing performance and dairy product quality (Bishop & Woolliams, 2014) ^[11].

High-Throughput Peptidomics and Molecular Research Gaps

The molecular mechanisms underlying SCM-induced

proteolysis remain incompletely understood. Although plasmin-mediated degradation of caseins has been widely investigated, the relative contributions of cathepsins, neutrophil-derived proteases, and pathogen-specific enzymes require further clarification (Kelly & McSweeney, 2003; Le Roux *et al.*, 2003) ^[29, 35].

Recent advances in proteomics, peptidomics, and mass spectrometry have revealed substantial alterations in the milk peptidome during mammary inflammation. However, pathogen-specific peptide signatures and their relationships with disease progression, technological defects, and product quality remain largely unexplored (Martins *et al.*, 2020) ^[39]. Identification of these molecular fingerprints could facilitate the development of rapid diagnostic platforms capable of detecting pathogen-specific milk alterations before major quality deterioration occurs, thereby improving mastitis management and raw milk quality control (Martins *et al.*, 2020) ^[39].

Technological Gaps and Innovative Processing Solutions

Current industrial management of SCM-affected milk largely relies on dilution within bulk milk systems rather than targeted technological interventions. Consequently, relatively little research has evaluated processing approaches designed specifically to mitigate the adverse effects of elevated SCC and inflammatory enzymes on dairy product quality (Le Maréchal *et al.*, 2011) ^[34].

Membrane-based technologies such as microfiltration and ultrafiltration offer promising opportunities to remove somatic cells, bacteria, and enzyme-containing cellular debris from raw milk. However, their effectiveness in restoring coagulation properties, improving heat stability, and recovering cheese-making performance under commercial processing conditions remains insufficiently investigated (Leitner, 2004; Le Maréchal *et al.*, 2011) ^[37, 34].

Future research should evaluate whether advanced separation technologies, alone or combined with enzymatic stabilization strategies, can reduce SCM-associated processing losses while maintaining nutritional and sensory quality.

Novel Non-Antibiotic Interventions

The growing global concern regarding antimicrobial resistance has increased interest in alternative approaches for mastitis prevention and treatment. Development of non-antibiotic interventions is now considered a major priority within dairy health management and One Health frameworks (Ruegg, 2017) ^[58].

Emerging technologies including vaccines, bacteriophage therapy, probiotics, immunomodulators, antimicrobial peptides, and physical therapies such as Acoustic Pulse Technology (APT) have shown potential for improving udder health while reducing reliance on conventional antimicrobials (Leitner *et al.*, 2011; Blum *et al.*, 2021) ^[36, 12].

Although preliminary findings are encouraging, robust large-scale longitudinal studies are required to determine whether these interventions can effectively eliminate intramammary

infections, restore mammary tissue function, normalize milk synthesis, and reverse the physicochemical and technological defects associated with SCM. Future research should also evaluate the long-term economic feasibility and practical adoption of these technologies under commercial farming conditions (Leitner *et al.*, 2011; Ruegg, 2017) [36, 58].

Conclusion

Subclinical mastitis (SCM) is a major constraint to sustainable dairy production, exerting profound effects on the physicochemical, nutritional, and technological quality of bovine milk despite the absence of visible clinical signs. The evidence synthesized in this review demonstrates that SCM disrupts mammary epithelial integrity and blood-milk barrier function, resulting in altered milk composition characterized by increased somatic cell count, elevated pH and electrical conductivity, reduced lactose concentration, and disturbed ionic balance.

SCM also compromises the nutritional quality of milk through reduced casein synthesis, enhanced proteolysis, altered mineral composition, increased lipolysis, and oxidative stress. These changes significantly impair the technological functionality of milk, leading to prolonged coagulation time, weaker curd formation, reduced cheese yield, impaired fermentation performance, decreased heat stability, and shortened shelf life of dairy products. Consequently, SCM adversely affects the efficiency and profitability of dairy processing operations.

Beyond its impact on milk quality, SCM imposes substantial economic losses through reduced milk yield, lower product recovery, increased management costs, and loss of quality-based incentives. Furthermore, the potential presence of zoonotic pathogens, bacterial toxins, antimicrobial residues, and antimicrobial-resistant microorganisms highlights its importance as a critical One Health issue.

Overall, SCM should be recognized not only as an udder health disorder but also as a significant milk quality and dairy processing challenge. Future progress will depend on the integration of precision monitoring technologies, early diagnostic biomarkers, genetic improvement strategies, and effective non-antibiotic interventions to enhance udder health, preserve milk quality, and ensure the long-term sustainability of the dairy industry.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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