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A review of lameness in dairy cattle: Causes, impacts and prevention

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Abstract
Lameness is one of the most prevalent, multifactorial, and economically important health disorders affecting dairy cattle worldwide, with serious implications for animal welfare, productivity, and herd longevity. This review synthesizes current knowledge on the etiology, pathogenesis, risk factors, clinical manifestations, and herd-level management of lameness in dairy cows. Lameness is primarily associated with hoof and limb disorders, particularly claw horn lesions, digital dermatitis, and laminitis, and is most frequently observed during early lactation when cows experience negative energy balance, metabolic stress, and rapid physiological changes. The condition significantly reduces milk yield, milk solids, and reproductive efficiency, while increasing treatment costs, culling rates, and overall economic losses. Patho-physiologically, lameness involves complex interactions between metabolic disturbances, vascular dysfunction, mechanical overload, and inflammatory processes within the claw. Subclinical laminitis plays a crucial role in the development of chronic hoof lesions and often remains undetected until production losses become evident. Risk factors vary across herds and include nutritional imbalances, improper housing design, poor flooring and drainage, inadequate claw trimming, infectious pathogens, genetic predisposition, age, body condition score, and environmental stressors such as heat and humidity. The review also highlights the importance of accurate lameness detection through locomotion scoring and behavioural assessment. Effective control of lameness requires an integrated herd-level approach encompassing balanced nutrition, functional claw trimming, appropriate housing and flooring, regular foot bathing, and timely intervention. Addressing lameness as a herd problem through preventive management strategies is essential for improving dairy cow welfare, enhancing productivity, and ensuring long-term farm sustainability.

Keywords: Dairy cattle, lameness, hoof disorders, laminitis, digital dermatitis, animal welfare, herd management, milk production

Introduction
Cattle lameness is one of the most prevalent and costly health problems in dairy herds, adversely affecting animal welfare, milk production, fertility and longevity. It is primarily caused by hoof and limb disorders and often leads to increased culling, economic losses and reduced overall herd sustainability. Lameness markedly reduces dairy cow survival, particularly when it occurs during early lactation. Specific conditions such as foot rot and sole ulcers significantly increase the risk of culling or death, with the strongest effects observed when these disorders arise in the first few months after calving. (Booth *et al*., 2004) [5]. Lameness significantly reduces milk yield and milk solids across all major dairy breeds, with losses increasing as severity and recurrence rise. Early lactation shows a high prevalence of lameness, leading to marked production losses. Effective early detection and management of lameness remain essential for improving productivity and cow welfare. (Kofler *et al*., 2021) [16]. The economic losses associated with lameness include reduced milk production, treatment costs, reduced reproductive performance and increased risk of death and culling (Green *et al*., 2014) [13]. Lameness is seen to be the most important factor in animal health, second only to mastitis and reproductive issues. Bovines that are lame have lower reproductive efficiency.
Lameness is a widespread welfare and economic problem in dairy cattle, caused by multiple interacting factors related to housing, nutrition, genetics, and management. Effective prevention and timely treatment, supported by good stockman ship and appropriate housing

design, are essential to reduce lameness incidence and its long-term impacts on productivity and cow welfare (Ward, 2001) [32].

Lameness prolongs the interval between conception and calving by inducing physiological stress that disrupts normal reproductive hormone regulation. Stress associated with lameness reduces luteinizing hormone (LH) secretion while increasing adrenocorticotrophic hormone (ACTH) levels, leading to impaired heat expression, irregular or asynchronous LH surges and abnormal ovulation. Consequently, lameness results in substantial production losses in bovines. Most cases of lameness arise from foot lesions rather than disorders of the upper limbs (Thomsen *et al*., 2023) [29].

Bovines have two digits on their hoofs, making them cloven-footed animals. There are two claws: the inner medial claw and the outer lateral claw. The medial claw is the larger claw on the front feet of cattle, but the lateral claw is slightly more noticeable in the back feet.

In dairy herds, 20% of the cattle may get lame instantly, and 60% of the cattle may develop lame over the year.

Infectious pathogens from the underfoot slurry and foot lesions typically linked to laminitis syndrome account for more than 90% of lameness in cattle. The cow's body language, which is used to score the degree of lameness, is altered by foot pain. Pain is thought to be caused by a dense network of sensory nerve fibers and many nerve endings in the area of the sole heel bulb. Long periods of inactivity on concrete surfaces and psychological strain from interacting with a dominating animal are thought to be novel stresses. Dairy producers' ignorance and incompetence, as well as their lack of patience and kindness towards the animals raise the frequency of lameness. When estimating the cost of lameness, some of the aspects that are highly significant economically include negative effects on fertility, loss of body condition and milk, cost of medication, veterinary expenses, value of time spent nursing the cow, culling, and reduction of a cow's economic life. Lameness is a term used to describe abnormalities in the way cows move, and it is frequently included in the traditional clinical signs of animal pain perception. Asymmetrical movement is one of the motor diseases of variable severity that it manifests as rhythmic irregularities, a slowing pace, less weight bearing on the hooves, and poor posture. Lameness includes all disorders of the limbs and hooves, both viral and non-infectious, that cause pain sensations that drastically lower animal welfare indices, resulting in decreased milk production, reproductive losses, and even culling (Bran *et al*., 2018; Flower & Weary, 2009) [6, 10]. Lameness-related hoof illnesses are most prevalent in the first three to five months following calving (Kofler, 2013) [15], however 20% of the herd's cows exhibit mild signs in the second month of lactation (Olechnowicz & Jaśkowski, 2011; Urban-Chmiel *et al*., 2023) [21, 30].

Economic losses associated with lameness in dairy cattle are mainly attributed to three key components: reduced milk yield, accounting for approximately 40% of total losses; impaired reproductive performance, contributing around 30%; and expenses related to treatment, which make up the remaining 30%. These losses are further amplified because lameness often develops weeks or months before it is clinically detected and may persist for several weeks, or even up to five months, after treatment has ended (Neirurerová *et al*., 2021) [19]. Progressive degeneration of joints, ligaments, or bones structures essential for maintaining posture and balance can contribute to an increased incidence of lameness, especially in adult cattle. Additionally, cows with large, pendulous udders are more susceptible to lameness due to altered gait patterns and uneven distribution of weight on the hooves (Foditsch *et al*., 2016) [11].

Factors Involved in Hoof Diseases in Cattle

Hoof diseases and lameness in dairy cattle arise from a multifactorial and complex etiopathogenesis, with risk factors varying across countries, regions, and individual farms. The most commonly implicated factors are linked to herd management practices and housing conditions, encompassing both infectious and non-infectious causes. These include inadequate housing dimensions, high stocking density, unsuitable walking surfaces such as slippery or slatted floors, sharp turns at barn entrances or exits, and poorly designed or uncomfortable resting areas, all of which increase the risk of lameness (Bran *et al*., 2018; Browne *et al*., 2022) [6, 7].

Infectious hoof diseases are caused by a wide range of bacterial pathogens, including anaerobic species such as Fusobacterium spp., facultative aerobes like Campylobacter spp., and aerobic bacteria including Staphylococcus, Streptococcus, and Treponema spp. Infection is further facilitated by unhygienic environments rich in opportunistic organisms such as Escherichia coli, which frequently coexist with Staphylococcus species or members of the family Pasteurellaceae. Such conditions promote pathogen colonization, multiplication, and the subsequent development of lameness-related diseases (Refaai *et al*., 2013) [24].

Research indicates that the occurrence of infectious diseases within a herd largely depends on the individual resistance of animals. While some cows experience repeated infections, others within the same herd and even their offspring remain clinically unaffected. Breed susceptibility also plays a role, with Holstein-Friesian cows and their crossbreds showing a higher predisposition to infectious hoof diseases compared to other breeds (Palmer & O'Connell, 2015; Somers *et al*., 2005) [22, 28].

Digital dermatitis (DD) is a highly prevalent hoof disorder in dairy cattle in Western countries, whereas it is relatively uncommon in crossbred cattle and buffaloes in India. It is a chronic, erosive, and proliferative infection affecting the epidermis near the skin-horn junction in the flexor region of the interdigital space. DD is less frequently associated with lameness in older cows, possibly due to the development of stronger immunity. Affected animals often show marked lameness and tend to remain recumbent. Lesions primarily involve the hind limbs and are commonly located in the interdigital area or at the heel bulbs. The disease initially presents as superficial inflammation and may progress to ulcerative, granulomatous lesions extending into the interdigital skin. Contributing factors include imbalanced nutrition, high stocking density, and poor hoof hygiene, and if left untreated, DD can result in permanent limb damage (Barker *et al*., 2010) [2].

High milk production during peak lactation, combined with nutritional deficiencies in the postpartum period, significantly increases the risk of lameness, particularly in Holstein-Friesian (HF) dairy cattle. Loss of body weight and reduced body condition during early lactation further

exacerbate susceptibility to hoof disorders and lameness. Studies have shown that HF cows with a body condition score (BCS) of ≤ 3.0 experience a higher frequency of lameness compared to cows in better body condition (Alawneh *et al*., 2014; Bran *et al*., 2018; Green *et al*., 2014) [1, 6, 13]. However, Newsome and colleagues reported that rapid and substantial loss of body weight, along with a decline in body condition score (BCS), is closely associated with the development of lameness. This relationship may be partly explained by reduced willingness of lame cows to access the feed table, further aggravating negative energy balance and body condition loss (Newsome *et al*., 2017) [20].

Nutritional factors play a critical role in the development of lameness in dairy cows. Adequate intake of minerals such as calcium, copper, and zinc, along with vitamins A and D, B-complex vitamins (including niacin), essential amino acids like cysteine and methionine, and fatty acids particularly linoleic and arachidic acids is essential for proper hoof horn formation and keratinization. Diets that are nutritionally imbalanced increase susceptibility to infectious diseases and impair normal hoof wall development. Furthermore, feeding highly fermentable, energy-dense rations with insufficient roughage can promote ruminal acidosis and metabolic disturbances. Reduced rumen pH enhances lactic acid production, disrupts normal fermentation, and leads to endotoxin and histamine release, resulting in vasoconstriction within the hoof and deterioration of horn quality. These processes ultimately contribute to laminitis and the manifestation of lameness.

Cow age is a significant determinant of lameness, with animals in their second or subsequent lactations showing a higher prevalence of hoof abnormalities and gait disorders, regardless of breed, including Jersey, Holstein-Friesian, or crossbred cattle. In adult cows, progressive degeneration of joints, ligaments, and bones structures essential for posture and balance contributes to an increased incidence of lameness. Additionally, cows with large, pendulous udders are more susceptible due to altered gait patterns and uneven hoof loading. Environmental conditions, particularly high temperature and humidity, further compromise hoof integrity by promoting ulcer formation, microbial proliferation, and greater disease severity. The relative importance of these risk factors varies according to the housing and management systems employed on individual farms (Foditsch *et al*., 2016) [11]. Concurrent limb and hoof disorders are important contributors to lameness in dairy cows. These include white-line disease, sole injuries such as toe and sole ulcers, digital dermatitis, interdigital inflammation, heel erosion, and laminitis, which arises from damage to the vascular structures of the hoof. The presence of these conditions significantly increases the likelihood and severity of lameness in affected animals (Boettcher *et al*., 1998) [4]. At the herd level, several management and environmental factors contribute to the risk of lameness, including herd size, the extent of grazing areas, the presence of stones or slatted surfaces along cow walkways, and the slope and sharpness of turns on routes used by cows after milking. These factors can increase mechanical stress on the hooves and predispose animals to lameness (Browne *et al*., 2022) [7]. Lameness assessment should be carried out by observing cows walking on a flat, firm, and non-slippery concrete surface. Trained observers are substantially more accurate up to 2.5 times than untrained farmers in identifying lame animals. Cows should be evaluated from multiple angles, including front and rear views, while standing still, walking forward, and turning in both directions.

A normal gait consists of three phases: protraction, weight bearing, and retraction. Healthy cows maintain a level back posture and place their hind feet close to the position of their forefeet. In contrast, lame cows exhibit distinct behavioral and postural changes, such as lowered head carriage, shortened stride length, and uneven weight distribution. Pain in a limb is often indicated by elevation of the opposite shoulder or hip. Cows with front-limb lameness typically raise their head when weight is placed on the affected limb, while hind-limb lameness is characterized by elevation of the corresponding hip and outward swinging of the affected leg to avoid weight bearing (Garvey, 2022; Werema *et al*., 2022; Kimeli, 2014) [12, 33, 14].

Table 1: The Lameness Index Score (LIS), accompanied by its clinical description and evaluation criteria, was utilized for assessing lameness in crossbreed dairy cows before and after hoof trimming (HT)

LIS	Clinical description	Assessment criteria
1	Normal	The cow maintains a level-back posture while both standing and walking, and her gait appears normal.
2	Mildly lame	While standing, the cow maintains a level-back posture, but during walking, she adopts an arched-back stance, even though her gait remains unaffected.
3	Moderately lame	An arched-back posture is evident both while standing and walking. Her gait is affected and is best described as short striding with one or more limbs.
4	Lame	The presence of an arched-back posture is consistent, accompanied by a gait that can be characterized as deliberate, with the cow tending to favour one or more limbs/feet.
5	Severely lame	Furthermore, the cow displays an incapacity or significant hesitancy to support weight on one or more of her limbs/feet

Different stances adopted by a lame animal

- **Camping forward:** Pain in the apex of the claw;
- **Walking narrow:** Subclinical laminitis mainly in the medial claws;
- **Crossing either fore or hind feet:** Acute pain;
- **Knuckling of the fetlock:** Pain in the heel;
- **Hanging leg lameness:** Septic arthritis of the pedal joint or a fractured pedal bone;
- **Cattle reluctant to rise on forelimbs:** Very acute laminitis;
- **Cow hock posture:** Overburdening of heel of the lateral claw.
- **Camping back:** Pain in the heel region in rear claws.
- **The Laminitis syndrome:** In cattle, laminitis is a systemic illness that causes claw signs. It can manifest in a variety of different ways, including acute or subacute, chronic forms similar to those in horses, and subclinical types unique to cattle.

Acute laminitis is an uncommon condition in cattle and is most often associated with excessive intake of grains or accidental consumption of cereals. Affected animals show increased heart and respiratory rates, along with pale, watery feces. The severity of clinical signs depends on the quantity

of grain ingested; most cattle exhibit staggering gait, while severely affected animals may become recumbent. Additional signs include distension of superficial limb veins, crawling on knees, crossing of limbs, and a characteristic camped stance with forelimbs and hind limbs positioned abnormally. Excessive grain intake overwhelms the rumen's buffering capacity, leading to a drop in ruminal pH to 5 or below, cessation of ruminal motility, and development of rumenitis, ultimately predisposing the animal to laminitis (Randhawa, 2006) [23]. Subacute laminitis refers to a mild, transient laminitis-like condition in which the affected animal appears to recover clinically after a brief episode. It causes subtle discomfort, characterized by cautious placement of the feet and frequent shifting of weight from one foot to another. Diagnosis is often difficult because pain is distributed evenly across all limbs. In some cases, swelling of the foot with pink, edematous skin around the dewclaws and above the coronary band may be observed. The condition is commonly associated with improper feed intake during the post-calving period and usually resolves without treatment. Weeks after the initial insult, a horizontal groove may appear on the hoof wall parallel to the coronary band, indicating a past episode. Sudden, short-term dietary changes are believed to trigger vascular dilation within the hoof, increasing intra-digital pressure and resulting in pain; however, due to its delayed onset and self-limiting nature, therapeutic intervention is generally unnecessary (Van Metre, 2017) [31].

Subclinical Laminitis (SCL), or Pododermatitis Aseptica Diffusa (PAD)

As the name implies, subclinical laminitis does not show obvious clinical signs during the early stages of pathological change. However, structural and functional integrity of the claw horn is compromised, making it more susceptible to damage from environmental factors. Simultaneously, the supportive structures of the pedal bone and the suspensory apparatus of the digit undergo degenerative changes. This condition is particularly prevalent in well-managed, high-yielding dairy cows.

Lesions may appear weeks after the initial pathological insult, as the claw capsule horn gradually softens. Continuous exposure of the sole to slurry further weakens the horn, increasing vulnerability when cows walk on hard surfaces. Although not conclusively demonstrated, hemorrhage within the sole horn is considered part of the pathophysiology of SCL. When the pedal bone sinks sufficiently to exert pressure on blood vessels, spontaneous bleeding may occur, highlighting the role of mechanical overload in disease etiology. Yellow discoloration of the sole has also been suggested as a clinical indicator of SCL.

At the herd level, subclinical laminitis should be suspected when the combined incidence of sole ulcers, toe ulcers, and white line disease exceeds 5-10%.

Pathogenesis of Laminitis

The pathogenesis of laminitis is best described as alternating metabolic disturbances followed by progressive mechanical degradation of internal foot structures. This process can be divided into three distinct phases.

Phase 1 (Initial Activation Phase)

This phase is initiated by a systemic metabolic insult that lowers systemic pH, activating vasoactive mechanisms and increasing digital pulse and blood flow. Depending on the nature of the insult, endotoxins and histamine are released, causing abnormal vasoconstriction and vasodilation along with the formation of non-physiological arteriovenous shunts. These changes elevate intravascular pressure. Histamine-mediated vascular alterations result in blood pooling within capillaries, vessel rupture, serum leakage, and hemorrhage. Subsequent vascular damage leads to edema and hemorrhage of the solar corium, causing corial expansion and severe pain.

Phase 2 (Local Mechanical and Vascular Damage)

Following the initial insult, vascular edema leads to ischemia and hypoxia of internal digital tissues, reducing oxygen and nutrient supply to epidermal cells. Ischemia further promotes arteriovenous shunting, which is intensified by trauma, stress, and vasoactive mediators. This self-perpetuating cycle progressively impairs local circulation, producing changes analogous to an ischemic infarction of the digit.

Phase 3 (Progressive Mechanical Damage to Bone and Support Structures)

Prolonged vascular compromise results in failure of the epidermal-dermal junction, causing separation of the stratum germinativum from the corium and breakdown of laminar support. As a consequence, the pedal bone becomes displaced, compressing underlying soft tissues. This compression leads to hemorrhage, thrombosis, edema, ischemia, and focal necrosis in the solar region. Continued tissue degeneration disrupts normal horn production, resulting in chronic lesions such as double sole, sole hemorrhages, bruising, and diffuse solar pulp lesions.

Activation of Matrix Metalloproteinases and Related Mediators

Matrix metalloproteinases (MMPs), which normally function in tissue remodeling, play a crucial role in the progression of laminitis when pathologically activated. Endotoxins, cytokines, and hypoxia stimulate excessive activation of MMPs particularly MMP-9 leading to collagen degradation and weakening of the suspensory apparatus of the digit. This weakening contributes to displacement of the pedal bone and development of toe ulcers.

Activation of MMP-2 through a ~52 kDa gelatinolytic protease (hoofase) causes structural and functional disruption of connective tissue supporting the distal phalanx. Peak activity of this enzyme has been observed from two weeks pre-calving to 4-6 weeks post-calving. Notably, MMP-9 typically associated with inflammatory processes is not detected at significant levels in maiden or first-lactation heifers, suggesting that classical rumen acidosis-induced laminitis is unlikely to be the primary cause of lameness in these animals.

Additionally, epidermal growth factor (EGF) concentrations increase following ruminal epithelial damage during subacute ruminal acidosis. EGF receptors are present in the epidermal basement membrane of the claw, indicating a potential role in altered horn growth and lamellar weakening.

Other Factors Contributing to Subclinical Laminitis

Several additional factors influence the development and progression of SCL:

a) Local factors such as season, inadequate exercise, and prolonged exposure to concrete flooring
b) Animal-related factors including weight distribution, age, growth rate, genetics, body and foot conformation, and behavior
c) Relaxin hormone release during parturition, which may loosen the suspensory apparatus and promote rotation of the third phalanx
d) Systemic diseases such as mastitis, ketosis/acetonemia, metritis, udder edema, and retained placenta
e) Deficiencies of trace minerals including zinc, copper, manganese, and cobalt
f) The interaction of multiple biogenic agents and bioactive molecules that collectively produce diverse clinical manifestations

Long-term disruption of horn production results in softer-than-normal sole horn due to reduced cell adhesion caused by alterations in intercellular cementing substances. Increased horn flexibility leads to excessive pressure on the flexor process of the pedal bone, predisposing to sole ulcer formation. Poor-quality horn at the white line initiates white line disease, while softer horn in the heel region predisposes to heel erosion. Damage to the suspensory apparatus fibers may cause sinking or displacement of the pedal bone; however, permanent sinking occurs only in a small but significant proportion of SCL cases.
Treatment of subclinical laminitis is generally not feasible; therefore, preventive strategies at the herd level are essential.

Herd-Level Management
Lameness is fundamentally a herd-level problem and must be monitored regularly through appropriate management practices. Three fundamental management practices to address lameness in dairy animals are:

Balanced Nutrition
A nutritionally balanced ration with an appropriate energy-protein ratio is essential for preventing lameness in dairy cows, with concentrate supplementation adjusted according to milk yield. At least one-third of total dry matter intake should consist of fodder or roughage to stimulate rumination and saliva production, thereby aiding ruminal buffering and maintaining optimal rumen pH. Routine supplementation with mineral mixtures is crucial, as deficiencies of trace elements such as zinc, copper, manganese, and cobalt negatively affect hoof horn formation. Additionally, biotin supplementation has been shown to improve hoof horn quality by enhancing intercellular cementing between keratinocytes, thereby increasing horn strength and resistance to lesions (Bicalho & Oikonomou, 2013) [3].

Claw Trimming
Regular claw trimming at approximately six-month intervals is a fundamental component of effective hoof health management in dairy cattle. The Dutch trimming method, commonly used for both preventive and therapeutic purposes, focuses on restoring optimal toe length (about 7-8 cm) and achieving balanced weight distribution between claws, particularly by relieving excessive load on the outer hind claw. Trimming of the axial and abaxial walls helps redirect weight-bearing forces to stronger and healthier claw regions.
In therapeutic trimming, diseased or damaged horn is carefully removed to expose and drain lesions such as abscesses, thereby promoting healing. In severe cases, wooden or rubber blocks are applied to the healthy claw to offload the affected claw, typically remaining in place for 5-6 weeks or until adequate recovery is achieved (Manske *et al*., 2002; Shearer & van Amstel, 2001) [18, 26].

Management: Frequent Footbaths
Regular foot bathing is an effective management practice for controlling hoof lesions, particularly contagious skin conditions such as digital dermatitis. Formalin footbaths at a concentration of 4% have been shown to be highly effective. An ideal footbath should measure approximately 15 cm in depth, 1 m in width, and 3 m in length, with concrete footbaths being the most durable and cost-effective option. A standard 4% solution can be prepared using 5 liters of 39-40% formalin in 120 liters of water.
Foot bathing is recommended on three consecutive days every two weeks. Prior to foot bathing, hooves should be thoroughly cleaned to enhance efficacy. After treatment, animals should be kept on a clean concrete floor for at least 30 minutes to allow proper action of the disinfectant. In herds with fewer animals, formalin spray (40 ml per liter of water) may be applied on the first, second, and third day of each fortnight.
Alternative non-antibiotic footbath solutions include 5% copper sulphate and 20% zinc sulphate; however, their use is generally discouraged due to environmental contamination following disposal. Antibiotic footbaths using oxytetracycline or tetracycline hydrochloride at 6 g/L have also been used, with no evidence of systemic absorption, making them effective for localized treatment (Shearer *et al*., 2015; Roche *et al*., 2024) [27, 25].

Housing and Nutritional Management for Lameness Prevention
Optimal underfoot conditions and effective drainage are essential to maintain hoof integrity and reduce the risk of lameness. Cubicles should be adequately sized, with a minimum width of 1.2 m and a length of 2.5-2.8 m depending on cow stature, to allow comfortable lying and rising movements. Sudden dietary changes should be avoided; instead, a well-formulated transition ration should be introduced two to three weeks before calving and continued through the first six weeks of lactation, with concentrates increased gradually.
Concentrate feeding should not exceed 4 kg per feeding to prevent ruminal acidosis. At least 21% of the neutral detergent fibre (NDF) in the ration should be derived from forage sources to support rumen function. During early lactation, dietary buffers such as sodium bicarbonate should be included at approximately 1% of total ration dry matter to stabilize rumen pH.
In herds with high-producing cows and a high prevalence of lameness, targeted nutritional supplementation is recommended. Biotin supplementation at 1 g per animal per day (2% premix) has been shown to improve hoof horn quality and support healing of sole lesions. Additionally, zinc supplementation in the form of zinc methionine or zinc sulphate at 3 g per animal per day should be provided alongside the regular mineral mixture to enhance hoof horn strength and resilience (Lischer *et al*., 2002; Cook, 2017; Cook *et al*., 2012) [8, 9, 17].

Conclusion

Lameness remains one of the most significant health, welfare and economic challenges in dairy cattle, with far-reaching consequences for productivity, reproduction and herd sustainability. Its multifactorial etiology, predominantly linked to hoof and foot lesions, underscores the importance of integrated management strategies addressing housing, nutrition, genetics and stockmanship. The adverse effects of lameness are particularly pronounced during early lactation, when reductions in milk yield, fertility and survival are greatest, often culminating in increased culling and economic losses. Moreover, the physiological stress associated with lameness disrupts normal reproductive hormone dynamics, further compounding production inefficiencies. Given its strong association with reduced animal welfare and farm profitability, lameness should be regarded as a priority condition alongside mastitis and reproductive disorders. Early detection, prompt treatment and effective preventive measures are essential to minimize its incidence and severity. Improving lameness management will not only enhance dairy cow welfare but also support long-term productivity and economic viability of dairy herds.

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