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Laminitis as a cause of metabolic disorders in domestic animals (review paper)

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Abstract. *The article presents a comprehensive analysis of modern scientific data on laminitis in domestic animals, taking into account the specific features of the course of the pathological process in horses, cattle, sheep and goats. The approaches to understanding laminitis as a systemic multifactorial syndrome, which is formed as a result of the interaction of metabolic, endocrine, infectious-toxic and mechanical factors, are summarized. It is shown that the development of the disease is accompanied by deep microcirculation disorders in the lamellar apparatus of the hoof, degradation of the dermal-epidermal junction, activation of inflammatory and ultimately leads to structural destruction of the hoof apparatus and a decrease in animal productivity.*

Particular attention is paid to the analysis of pathogenetic mechanisms taking into account the type of digestion. It has been established that in horses, as monogastric animals, endocrine and metabolic disorders play a leading role, in particular hyperinsulinemia, insulin resistance and imbalance of carbohydrate metabolism, which causes the development of endocrinopathic laminitis. In cattle, the key triggering mechanism is subclinical rumen acidosis, which is accompanied by changes in the rumen microbiota, increased blood endotoxin levels and the development of a systemic inflammatory response. In sheep and goats, laminitis is formed as a result of a combination of metabolic, infectious and mechanical factors and determines the variability of clinical and morphological manifestations of the disease.

Biochemical changes characteristic of different stages of laminitis (subclinical, acute and chronic) is systematized, including increased concentrations of insulin, glucose, triglycerides, activation of tissue damage enzymes (lactate dehydrogenase, creatine kinase), pro-inflammatory cytokines, C-reactive protein. It is shown that these indicators can be used for early diagnosis, differentiation of stages of the disease and prediction of its course. Modern approaches to the diagnosis of laminitis are also summarized, based on a combination of clinical examination, laboratory studies and instrumental methods, in particular radiography, ultrasound diagnostics, computed tomography and motor activity monitoring systems. Morphological, histological and pathoanatomical changes are characterized, which reflect the progression of the pathological process from initial microscopic lesions to pronounced hoof deformation, tissue necrosis and fibrosis.

At the same time, modern approaches to the prevention of laminitis are separately considered, which include optimization of feeding, control of metabolic status, prevention of subclinical acidosis, improvement of housing conditions and implementation of early monitoring technologies. It is emphasized that effective management of laminitis risks is possible only under the conditions of an integrated approach that takes into account the species characteristics of animals, the level of their working capacity for horses and productivity for cattle, and technological conditions of maintenance.

The results of the generalization can be used to improve the system of early diagnosis, prevention and control of laminitis and will contribute to increasing productivity and improving the welfare of domestic animals.

Keywords: hoof, inflammatory process, acidosis, endotoxemia, impaired microcirculation, metabolism, lameness

Laminitis in domestic animals is a complex multifactorial disease characterized by damage to the lamellar apparatus of the hoof, impaired microcirculation and the development of degenerative changes in the tissues. This disease is accompanied by metabolic disorders,

endocrine dysfunctions, inflammatory processes and changes in the microbiota of the digestive tract (Nocek, 1997; Cook, 2004; Koziy, 2008; Enemark, 2008; Bojkovski, 2023).

Recent studies demonstrate that metabolic products of sugars, particularly methylglyoxal, can directly damage the lamellar tissue of the hoof, causing structural and histological changes characteristic of laminitis, as confirmed in ex vivo lamellar explant models in horses (Vercelli, 2021).

Laminitis is particularly relevant for working horses and high-yielding cows, where metabolic and hormonal mechanisms largely determine the severity of the disease, the level of productivity and the state of health. (Asplin, 2007; Bailey, 2013; Radzyhovskyi, 2024). Laminitis in sick animals causes lameness, reduced productivity, and a deterioration in the quality of life of animals, making it one of the key problems of modern animal husbandry (Clarkson, 1996; Cook, 2003; Greenough, 2007; Sertu, 2024).

Historically, the first scientific descriptions of laminitis in horses appeared in the second half of the 20th century, when researchers mainly associated the development of the disease with mechanical overload and circulatory disorders in the hoof. The classic works of Colles and Jeffcott laid the foundation for understanding laminitis as a vascular pathology. Subsequent studies have significantly expanded the understanding of the etiology and pathogenesis of the disease, proving that laminitis is a polyetiological process (Colles, 1977; Greenough, 2007; Vercelli, 2021). In high-yielding cows, the first detailed observations of laminitis as a factor in metabolic disorders were presented by Ukrainian researchers and emphasized the role of endocrine and cicatricial mechanisms in the development of the disease (Nocek, 1997; Koziy, 2008; Mulligan, 2008).

Laminitis is one of the most common causes of lameness in horses and cattle on all continents, although exact incidence rates vary considerably depending on the species of animal, housing conditions, type of farm and diagnostic methods (Wylie, 2011). Incidence analyses show that the incidence of laminitis in horses ranges from approximately 1.5% to 34% across populations, indicating significant geographical and technological variation (Wylie, 2011). In Finland, among horses presenting to veterinary clinics with laminitis, about 89% of cases were associated with endocrinopathies or metabolic disorders, rather than just mechanical injuries. (Karikoski, 2011).

In cattle, direct data on the incidence of laminitis on a global scale are less common, but the incidence of associated hoof lesions and lameness ranges from 10% to over 50%, depending on feeding and housing conditions (Webster, 2001; Bergsten, 2003; Oetzel, 2007; Capion, 2009). For example, in Turkey, 28.6% of cows were lame, with 82.7% of cases related to hoof lesions associated with subclinical laminitis (Belge, 2005; Bostanlik, 2025). Monitoring of dairy herds in Romania showed laminitis incidence of around 20.3% in 2021, 18.6% in 2022 and 17.4% in 2023, with a clear negative impact on productivity (Popescu, 2013; Kornienko, 2024).

In Ukraine, quantitative data on laminitis are less systematic, due to the limited number of dedicated open access studies. However, regional observations in Vinnytsia, Donetsk, Lviv and Cherkasy regions demonstrate a significant proportion of orthopedic hoof pathologies and lameness, which are often a consequence or accompany laminitis, in line with global trends (Koziy, 2008; Archer, 2010; Klymas, 2025).

Thus, laminitis is a widespread problem in livestock farming, which limits the performance of working and sports horses, the productivity of cattle, causes deterioration of the condition of animals and leads to significant costs, which provide treatment and preventive measures (Greenough, 2007; Sertu, 2024).

Analysis of recent research and publications. Laminitis in domestic animals is defined as a systemic multifactorial syndrome manifested by damage to the lamellar apparatus of the hoof and impaired microcirculation, with the subsequent development of degenerative changes in the tissues (Boosman, 1991; Greenough, 2007; Faustmann, 2025).

Modern research proves that laminitis is not only local, but also systemic in nature, affecting metabolism, the endocrine system, inflammatory processes and the microbiota of the digestive tract (Sadiq, 2020; Bezpalko, 2023).

Given the multifactorial nature of laminitis, further research focuses on its etiology, which combines systemic and local factors and determines the animal's predisposition to develop pathological changes.

Systemic ones include metabolic dysfunctions, endocrine disorders, insulin resistance and hormonal changes, which can lead to increased concentrations of insulin and glucose in the blood, and imbalance of lipid metabolism (Asplin, 2007; Bailey, 2013; de Laat, 2014; de Laat, 2019).

Local factors include mechanical overload of the limbs, hoof injuries, infectious processes and microcirculatory disorders, which contribute to ischemia and degradation of the lamellar layer (Boosman, 1991; Pollitt, 1996; Hood, 1999). The complexity of the etiology lies in the interaction of these factors: systemic disorders increase susceptibility to local damage, and injuries or inflammatory processes in the hoof exacerbate metabolic disorders.

In cows, the main risk factors are subclinical cicatricial acidosis, impaired mineral metabolism, a highly productive lactation period, and irrational feeding (Bergsten, 2003; Oetzel, 2007; Enemark, 2008). Local factors include mechanical overload of the hooves, injuries and improper care of the hooves (Greenough, 1997; Sadiq, 2020).

The mechanisms of laminitis development vary depending on the form of the disease. Acute laminitis in horses and cows is often associated with impaired microcirculation and inflammatory reactions in the lamellar tissue, leading to detachment of the stratum corneum of the hoof (Pollitt, 1996; Hood, 1999; Ding, 2020). Metabolic mechanisms include the effects of hyperinsulinemia on endothelial cells and a reduction in the ability of lamellar tissue to repair or regenerate (Asplin, 2007; de Laat, 2019).

In cattle, endotoxins and acid-base imbalances are of additional importance in subclinical cicatricial acidosis (Boosman, 1991; Nocek, 1997).

Clinical signs. Laminitis is characterized by variability of signs and depends on the form of the disease. Acute laminitis is manifested by sudden lameness, hoof pain, change in posture and refusal to move (Hood, 1999; Vercelli, 2021).

Chronic forms are characterized by progressive destruction of lamellar tissue, hoof deformation, and persistent lameness (Greenough, 2007).

Subclinical laminitis often has no obvious external symptoms, making it difficult to diagnose without the use of special research methods (Mudron, 1996; Koziy, 2008).

The inflammatory component of the disease is manifested by the activation of pro-inflammatory cytokines, endothelial dysfunction and tissue edema, and increased activity of matrix metalloproteinases contributes to the destruction of intercellular connections in the lamellar tissue (Capion, 2009; Ding, 2020).

Biochemical damage to the hoof is accompanied by increased activity of tissue damage enzymes and an imbalance of antioxidant defense.

Experimental studies in horses have confirmed the key role of metabolic factors in the development of laminitis: prolonged hyperinsulinemia itself is capable of inducing laminitis in clinically healthy animals, which emphasizes the importance of endocrine mechanisms in the pathogenesis of the disease (Asplin, 2007; de Laat, 2019).

Metabolic responses to high glycemic index diets in horses and ponies also suggest a link between feeding and the risk of laminitis (Bailey, 2013).

In this regard, timely and accurate diagnosis of the disease is of particular importance. Diagnosis of the disease is based on a comprehensive approach and includes clinical examination, assessment of gait and posture, laboratory studies of metabolic and inflammatory markers, as well as instrumental methods (radiography, ultrasound, thermography) (Greenough, 2007; Danscher, 2009; Danscher, 2010). For example, telemetric monitoring of movement allows you to detect early changes in gait that precede severe lameness, and behavioral changes can be a significant marker of the initial stages of laminitis (Danscher, 2009; Danscher, 2010).

Based on the identified risk factors and early changes, a comprehensive prevention of laminitis is formed, it is multi-component and includes feeding control with avoidance of excess of easily digestible carbohydrates, balancing the diet, prevention of subclinical cicatricial acidosis

(Oetzel, 2007; Enemark, 2008), monitoring of insulin resistance (Harris, 2006), optimization of housing conditions, timely hoof trimming (Sadiq, 2020) and other management measures (Bergsten, 2003). Veterinary monitoring of productivity, reliability and motor activity of animals allows for timely identification of risks for the development of laminitis and adjustment of feeding and housing.

Despite the implementation of preventive measures, laminitis and related forms of lameness have been shown to significantly reduce milk yields, reduce live weight gain and reduce reproductive performance (Archer, 2010; Sertu, 2024). Additional costs to the farm arise from the need for treatment, specialized care and hoof correction, as well as from the loss of working and sporting horses, which emphasizes the importance of an integrated approach to prevention and monitoring of animal health.

Therefore, current scientific publications emphasize and emphasize the need to consider and study the features of the development of laminitis as a systemic syndrome, including metabolic, hormonal and inflammatory components, and not only as a local pathology of the hoof (Eades, 2010; de Laat, 2019). This opens new perspectives for the development of comprehensive preventive programs that take into account both nutritional and hormonal metabolic risk factors.

That is why the purpose of the study is to determine the prevalence of laminitis, assess its impact as a factor in metabolic dysfunctions, and develop approaches to diagnosis and prevention to increase productivity and maintain the health of domestic animals.

Materials and methods. To prepare the review article, a systematic analysis of the scientific literature on the topic of laminitis in horses and cattle was conducted. The PubMed, Scopus, Web of Science and Google Scholar databases were used, in particular, studies related to the etiology, pathogenesis, risk factors, clinical manifestations and prevention of laminitis. Only works with reliable methodological data were selected, and information from different sources was subjected to comparative and analytical synthesis. This approach allowed us to systematize modern scientific data and identify the main patterns of the development of laminitis and its prevention.

Results. Pathogenesis of laminitis in domestic animals. Laminitis in domestic animals is the result of a complex interaction of pathogenetic mechanisms, leading to damage to the lamellar apparatus of the hoof and disruption of its structural integrity. The pathogenesis of the disease differs significantly depending on the species of animal, which is due to the characteristics of the digestive system, metabolism, endocrine regulation and conditions of detention.

In horses, which are monogastric animals, fiber fermentation occurs mainly in the cecum and colon. Excessive consumption of concentrated feeds, a high content of easily digestible carbohydrates and abrupt changes in the diet lead to a rapid increase in blood glucose levels, stimulating insulin secretion and the development of insulin resistance (Table 1).

Table 1

Risk factors for the development of laminitis in domestic animals

Group of factors	Examples	Animal species
Metabolic	Insulin resistance	Horses
Nutritional	Concentrates	Cattle
Mechanical	Hard floors	All
Infectious	Endotoxins	Cattle

Chronic hyperinsulinemia and insulin regulation imbalance disrupt the function of the endothelial microvessels of the lamellar apparatus, reduce vasodilation and increase capillary permeability. This leads to local edema, impaired blood supply and oxygen metabolism in the epidermal and dermal layers of the hoof. At the cellular level, pro-inflammatory signaling pathways

Section 3

are activated, the expression of cytokines (TNF- α , IL-1 β , IL-6) and inducible oxide synthase (iNOS) increases, which causes the accumulation of free radicals and oxidative stress, with the destruction of the basement membrane of the epidermis and the degradation of lamellar structures (Belknap et al., 2007).

Increased pressure on the hoof apparatus due to physical exertion, insufficient trimming or hard surfaces increases mechanical damage and contributes to the progression of degenerative changes (van Oldruitenborgh Oosterbaan, 1999; Hood, 1999; Asplin, 2007; Bailey, 2013).

Pathogenetic mechanisms of laminitis in cattle, which are polygastric animals, are mainly associated with impaired rumen digestion and endotoxin intoxication. Thus, in subclinical rumen acidosis (SARA), the rumen microbiota changes, the level of lipopolysaccharides (endotoxins) increases, which are absorbed into the bloodstream and activate a systemic inflammatory response. At the level of the lamellar apparatus, endotoxins cause endothelial dysfunction, increase leukocyte adhesion, enhance the expression of intercellular adhesion molecules (ICAM-1, VCAM-1) and activate the synthesis of pro-inflammatory cytokines. This leads to impaired capillary hemodynamics, local edema, ischemia and oxidative tissue damage. At the same time, high-yielding cows that stand on hard surfaces for long periods of time are exposed to mechanical stress, which increases stratum corneum degradation and the formation of chronic laminitis (Boosman, 1991; Oetzel, 2007; Enemark, 2008; Ding, 2020).

In sheep and goats, the pathogenesis of laminitis is often combined and includes metabolic, nutritional, mechanical and infectious factors. Excessive introduction of concentrates into the diet and a deficiency of coarse fibers can cause carbohydrate metabolism disorders and microcirculatory changes in the hoof, while prolonged stay on wet, dirty or hard litter increases the mechanical load on the lamellar apparatus. The lesion is accompanied by activation of the local inflammatory reaction, degradation of horny structures and disruption of the integrity of the basal membrane of the epidermis. In this group of animals, a combination of factors plays a particularly important role: for example, endotoxemia due to infectious-toxic processes in the gastrointestinal tract increases mechanical damage and degradation of the stratum corneum (Table 2) (Boosman, 1991; Lischer, 1994).

Table 2

Comparison of the pathogenesis of laminitis by animal species

Animal type	Key mechanism	Trigger	Main pathogenetic links
Cattle	Metabolic-toxic	Subclinical cicatricial acidosis (SARA)	Endotoxemia, inflammation
Horse	Endocrinopathic	Hyperinsulinemia	Microcirculation disorders, basement membrane degradation
Sheep/Goat	Combined	Feeding + conditions	Inflammation + mechanical stress

Thus, the pathogenesis of laminitis in domestic animals is multifactorial and specific for each species: in horses, endocrine and metabolic disorders play a leading role, in cattle – feed, infectious-toxic and hemodynamic changes, and in sheep and goats - a complex of metabolic, feed and mechanical factors. These processes determine local changes in lamellae, microcirculation disorders, stratum corneum degradation and systemic biochemical imbalances, which directly determine the formation of clinical manifestations and severity of laminitis.

Clinical picture of laminitis in domestic animals. In this regard, the clinical picture of the disease in domestic animals is characterized by species-specific characteristics, variability of symptoms, and different dynamics of the development of the pathological process (Table 3).

Table 3

Clinical symptoms (systematized)			
Symptom	Horses	Cattle	Sheep/Goats
Pain	Pronounced	Moderate/Extreme	Variable
Position	“Backward deviation”	Cautious gait	Constrained gait
Hoof Temperature	↑	↑	±
Performance	↓ impaired motor activity	↓ milk	↓ increments

In horses, the course of laminitis is closely related to endocrine and metabolic disorders, and in the early, subclinical stages may manifest only with minor changes in behavior and motor activity. Animals become less active, there is periodic weight transfer from one limb to the other, a slight increase in local hoof temperature and pulsation of the digital arteries, and often remains unnoticed without specialized control (Asplin, 2007; Belknap, 2007; Danscher, 2010; Bailey, 2013).

As the acute stage progresses, horses develop a characteristic clinical picture: pronounced lameness, reluctance to move, a typical “laminitis posture”, which is associated with local inflammation and increased expression of cytokines in the lamellae (Belknap, 2007) with weight transfer to the hind limbs and forelimbs forward, which is caused by pain in the hoof area. There is increased pulsation of the digital arteries, local hyperthermia, pain on palpation and testing of the hoof, and in severe cases, rotation or lowering of the third phalanx, which is confirmed by instrumental methods (Hood, 1999; Vercelli, 2021). The chronic course in horses is characterized by persistent morphofunctional changes: hoof deformation, formation of so-called “laminite rings”, expansion of the white line, decreased elasticity of the stratum corneum, and prolonged lameness, which significantly limits the working capacity and sporting use of animals. (O’Grady, 2008; Wylie, 2011).

In cattle, clinical signs of the disease are often subclinical or chronic, making timely diagnosis difficult. In the initial stages, decreased activity, changes in posture, cautious gait, increased lying time, and reduced feed intake are noted (Cook, 2003; Danscher, 2009). Subclinical laminitis in cows often manifests as hemorrhages in the sole of the hoof, yellow spots of the stratum corneum, and minor abnormalities in the growth of the hoof horn, which are detected only when examining or trimming the hoof (Mudron, 1996; Zhao, 2020). In clinically pronounced cases, lameness of varying degrees, gait asymmetry, pain when loading the limbs, and the development of complications in the form of plantar ulcers, double soles, and white lines are observed (Bergsten, 2003; Greenough, 2007). The chronic course is accompanied by persistent lesions of the ungulates, which leads to a decrease in milk yield, deterioration of reproductive performance and the general condition of the animals (Capion, 2009; Archer, 2010).

In sheep and goats, the clinical manifestations of laminitis are less specific, but are important in intensive housing conditions. In the early stages, lameness, limited mobility, frequent lying down, and changes in the shape and structure of the hoof are noted. Later, deformities of the hoof horn, its delamination, increased fragility and susceptibility to secondary infections develop, which can complicate the course of the disease (Lischer, 1994). In these species, housing conditions play a significant role, in particular, the humidity of the litter and the density of the animals, which contribute to the chronicity of the process.

Thus, the clinical course of laminitis in domestic animals is characterized by significant variability and depends on the type of animal, conditions of detention and stage of the disease. In the end, acute and endocrinopathic forms with pronounced pain syndrome and local inflammatory process in the lamellae predominate, which is confirmed by increased activity of IL-1 β , IL-6, TNF- α (Belknap, 2007), in cattle - subclinical and infectious course with gradual development of affected hooves, while in sheep and goats clinical manifestations are often associated with conditions of detention and tend to become chronic. This necessitates a differentiated approach to the diagnosis, treatment and prevention of laminitis in different types of domestic animals.

Biochemical changes in laminitis. Taking into account the stages of the pathological process, the development of laminitis in domestic animals is characterized by specific morphofunctional and biochemical changes. Laboratory methods include the determination of biochemical indicators that allow differentiating the stages of laminitis. At the subclinical stage, clinical manifestations are often absent, but biochemical changes already allow detecting the pathological process. Among them, a moderate increase in plasma insulin by 15–25% is observed, which reflects early metabolic control disorders, as well as an increase in glucose levels by 10–20% (Table 4) (Asplin, 2007; Bailey, 2013; Delarocque, 2021).

The concentration of triglycerides and free fatty acids in the blood increases by 5–15%, which signals initial disorders of lipid metabolism (Zhao, 2020). At the same time, markers of oxidative stress are noted - malondialdehyde (MDA) increases by 15–20%, and the activity of antioxidant enzymes, such as superoxide dismutase (SOD) and catalase, remains at the lower level of the norm (Treiber, 2009). In the blood plasma, a moderate increase in the pro-inflammatory cytokines TNF- α and IL-6 by 10–20% is observed, which indicates an early systemic inflammatory response (Belknap, 2007; Zhao, 2020).

Locally in the lamellar apparatus, edema, hyperemia, and initial degenerative changes in the basal membrane of the epidermis are noted (Treiber, 2009).

Moving on to acute (clinical) laminitis in cattle, the manifestations become more pronounced: animals demonstrate lameness, pain when supporting the limbs, the affected hooves become hot and sensitive to the touch. Biochemically, at this stage, a significant increase in insulin and glucose is noted - 50–70% above normal, which reflects a violation of endocrine control. The activity of the enzymes lactate dehydrogenase (LDH) and creatine kinase (CK) increases by 1.5–2 times, which indicates damage to the muscles and tissues of the hoof (Belknap, 2007; Delarocque, 2021). At the same time, markers of systemic inflammation increase - C-reactive protein (CRP) by 2–3 times, TNF- α and IL-6 by 50–70%, and the level of MDA increases by 40–50%, which indicates pronounced oxidative stress (Harris, 2006; Treiber, 2009). Local microcirculatory disorders lead to ischemia and necrosis of the stratum corneum. These changes confirm an active systemic inflammatory response, combined with local ischemia of the lamellar apparatus (Belknap, 2007; Harris, 2006).

Table 4

Clinical course and main biochemical changes in domestic animals during the development of laminitis

Animal type	Stage	Main biochemical changes	Indicators
Horses	Subclinical	↑ insulin, ↑ glucose (slightly), ↑ MDA, ↑ TNF- α , IL-6	Insulin 15–25 μ IU/mL, glucose 5.5–6.5 mmol/L
Horses	Acute	Significant ↑ insulin, ↑ LDH, ↑ CK, ↑ CRP, ↑ free radicals	LDH 400–600 U/L, CK 350–500 U/L
Horses	Chronic	Insulin resistance, fatty acid imbalance, low antioxidant activity	MDA ↑ 20–30%, SOD ↓ 10–15%
Cattle	Subclinical	↑ LPS, moderate ↑ TNF- α	LPS 0.5–1.0 EU/mL
Cattle	Acute	Significant ↑ lactate, ↑ cytokines, ↑ CRP	Lactate 2–4 mmol/L
Cattle	Chronic	Moderate ↑ inflammatory markers	TNF- α 5–10 pg/mL
Sheep, goats	Subclinical	Moderate changes in insulin and inflammatory markers	Insulin 12–20 μ IU/mL
Sheep, goats	Clinical	↑ inflammatory cytokines, metabolite imbalance	TNF- α 8–12 pg/mL

Chronic laminitis in cows and bulls is characterized by persistent hoof deformity, lamellar fibrosis, and gait mechanics disorders. Biochemically, persistently elevated insulin (60–90% above normal), prolonged lipid profile imbalance, and low antioxidant system activity (SOD, catalase 30–40% below normal) are noted (Zhao, 2020). Proinflammatory cytokines TNF- α and IL-6 remain moderately elevated by 30–50%, CRP by 20–40%. Locally, dystrophy of horny

spines, degeneration of the basement membrane, and chronic infiltration by lympho- and neutrophils are noted in the lamellae (Treiber, 2009).

In sheep and goats, the pathogenesis of laminitis has specific features due to the characteristics of the digestive system and housing conditions. In the subclinical stage, even in the absence of obvious lameness, a moderate increase in insulin (10–20%), glucose (5–15%), and triglycerides (5–10%) is noted (Zhao, 2020). There is activation of oxidative stress (MDA +10–15%, SOD – decrease by 5–10%) and a moderate increase in TNF- α and IL-6 (+10–15%). Locally, edema, hyperemia, and initial degenerative changes of the basement membrane are observed in the lamellae (Treiber, 2009).

In the acute stage, clinical signs become noticeable: lameness, soreness, increased hoof temperature. Biochemical indicators reflect pronounced disorders: insulin +40–60%, glucose +20–35%, LDH and CK are increased by 1.5–2 times, and the MDA level increases by 30–50% (Delarocque, 2021). Pro-inflammatory cytokines TNF- α and IL-6 increase by 40–60%, CRP – 2 times (Zhao, 2020).

In chronic laminitis in sheep and goats, hoof deformities, lamellar fibrosis and chronic inflammation are accompanied by biochemical disorders: constantly high insulin (50–80% above normal), lipid profile disorders, reduction of antioxidant activity by 30–40%, TNF- α and IL-6 +30–50%, CRP +20–40%. Locally – degeneration of the basement membrane, dystrophy of the horny spines, infiltration by lympho- and neutrophils and fibrosis of the lamellar apparatus (Treiber, 2009; Zhao, 2020).

Thus, biochemical markers in different species of domestic animals allow not only to diagnose laminitis in the early stages, but also to determine the stage of the disease and predict its course. In horses, the key is the control of insulin and markers of oxidative stress (Delarocque, 2021), in cattle - monitoring of endotoxemia, LDH, CK and inflammatory cytokines (Zhao, 2020), in sheep and goats - a combination of metabolic and inflammatory indicators together with an assessment of housing conditions and mechanical load on the limbs (Treiber, 2009; Zhao, 2020). A comprehensive analysis of these indicators allows for a differential assessment of subclinical, acute and chronic forms of laminitis and to direct preventive and therapeutic measures.

Diagnosis of laminitis. Given the variety of biochemical, morphological and functional changes, timely detection of laminitis requires the use of a comprehensive diagnostic approach. Diagnosis of laminitis in domestic animals includes clinical assessment, laboratory studies and modern instrumental methods that provide accurate identification of the stage of the disease and justification for the choice of therapeutic and preventive measures (Table 5) (O'Grady, 2008; Vercelli, 2021).

Table 5

Differential diagnosis of laminitis in domestic animals

Animal type	Method	What reveals	Morphological/histological changes	Pathological and anatomical changes
Horses	Ultrasound	Swelling of the lamellar layer, displacement of the 3rd phalanx	Swelling, hyperemia, degeneration of the basement membrane	Initial edema of the subcutaneous tissue, weak lymphocyte infiltration, minimal necrosis of the stratum corneum
Horses	X-ray / CT	Rotation or lowering of the 3rd phalanx	Necrotic changes of the lamellar layer	Deformation of the sole, bone erosions, displacement of the phalanx
Cattle	Laboratory (LPS, cytokines)	Subclinical inflammation	Plantar hemorrhages, edema	Initial hemorrhages in the dermis, mild infiltration by neutrophils
Cattle	X-ray	Extent of hoof damage	Ulcers, subendothelial edema	Degenerative changes in the dermis and epidermis, thickening of the stratum corneum
Sheep, goats	X-ray	Lameness, inflammation	Hoof deformity, initial fibrosis	Local necrosis of the stratum corneum, initial fibrous changes in the lamellar layer

In cattle, subclinical laminitis is often associated with subclinical cicatricial acidosis and elevated endotoxin levels, which trigger a systemic inflammatory response. Biochemically, moderate elevations in proinflammatory cytokines, markers of oxidative stress, and impaired microcirculation are observed (Boosman, 1991; Cook, 2004; Harris, 2006; Belknap, 2007; Zhao, 2020; Ding, 2020). The acute stage is accompanied by pronounced inflammatory changes, increased lipase, protease, C-reactive protein, and impaired hoof hemodynamics. The chronic stage shows long-term structural changes in the stratum corneum, rotation and lowering of the phalanx, impaired microcirculation, and decreased performance (Greenough, 2007; Koziy, 2008; Archer, 2010).

Instrumental diagnostics allows to detect pathological changes in the early stages.

Radiography is used to assess the angle of rotation and descent of the third phalanx in horses, the thickness of the stratum corneum and the presence of plantar defects in cattle (Cook, 2004; O'Grady, 2008; Vercelli, 2021).

Ultrasound (US) allows assessment of soft tissue edema, lamellar blood flow, and early degenerative changes, which is particularly useful in subclinical laminitis and is confirmed by increased expression of pro-inflammatory cytokines in lamellae (Hood, 1999; Asplin, 2007; Belknap, 2007; Vercelli, 2021).

Computed tomography (CT) provides three-dimensional assessment of bone and horn structures, accurate determination of phalanx rotation, and surgical planning (O'Grady, 2008; Vercelli, 2021).

Magnetic resonance imaging (MRI) provides high-resolution soft tissue detail, early detection of degenerative changes in lamellae, and impaired blood flow (Pollitt, 1996; Vercelli, 2021). Additionally, thermography and laser Doppler are used to assess local temperature and microcirculation, which allows differentiation of early stages of laminitis (Bailey, 2013; Vercelli, 2021).

Comprehensive diagnostics of laminitis, including clinical assessment, laboratory markers and instrumental methods, allows to accurately determine the stage of the pathological process, predict the risk of progression and choose the optimal therapeutic strategy. In horses, it is especially important for the early detection of endocrinopathic forms of laminitis, in cattle - for the assessment of the consequences of SARA and systemic endotoxemia, and in sheep and goats - for the control of combined nutritional, mechanical and infectious factors (Boosman, 1991; Lischer, 1994; Cook, 2004; Asplin, 2007).

Morphological changes in laminitis in domestic animals are complex, multi-level and reflect both local lesions of the lamellar apparatus of the hoof and systemic disorders that underlie the pathogenesis of the disease. At the macroscopic level, horses, cattle, sheep and goats exhibit characteristic changes in the hoof horn and underlying structures, the severity of which depends on the stage and duration of the pathological process. In the initial stages, especially in the subclinical course, macroscopic changes may be barely noticeable, but already at this stage, there are disturbances in the color of the stratum corneum in the form of yellowish or grayish areas corresponding to areas of impaired blood supply and microhemorrhages (Mudron, 1996; Enemark, 2008; Zhao, 2020).

In cattle, a typical sign is saline hemorrhages, localized mainly in the sole and white line, indicating chronic microcirculation disorders and increased pressure on the hoof dermis (Greenough, 2007; Enemark, 2008; Zhao, 2020).

In horses, in the acute stage, there is an increase in hoof temperature, soreness, and as the process progresses, rotation or distal displacement of the third phalanx, accompanied by deformation of the hoof capsule and a change in its configuration (Hood, 1999; Vercelli, 2021). Chronic laminitis is characterized by the formation of so-called "laminitis rings", thickening and deformation of the hoof wall, widening of the white line, development of a double sole, and in severe cases, stratum corneum detachment and the formation of chronic ulcers (Weaver, 2005; Greenough, 2007). In sheep and goats, macroscopic changes are often combined with signs of hoof rot and secondary infections, which complicates the course of laminitis and contributes to the development of necrotic processes (Lischer, 1994).

At the histological level, the key manifestation of laminitis is damage to the dermal-epidermal junction of the lamellar apparatus. Early changes include lamellar edema, capillary

dilation, disruption of the basement membrane, and epidermal cell disorganization (Pollitt, 1996; Hood, 1999; Belknap, 2007).

In horses, early changes include lamellar edema, capillary dilation, disruption of basement membrane integrity, and epidermal cell disorganization (Pollitt, 1996; Hood, 1999). An important pathomorphological mechanism is the activation of matrix metalloproteinases, which leads to degradation of basement membrane components and loss of dermis-epidermal junction, clinically manifested by a weakening of the attachment of the hoof wall to the underlying tissues (Pollitt, 1996; van Oldruitenborgh Oosterbaan, 1999).

In cattle, the histological changes are somewhat different and include hyperemia, blood stasis, microthromboses in the dermal vessels, perivascular infiltration by neutrophils and lymphocytes, and degeneration of keratinocytes (Boosman, 1991; Ding, 2020; Zhao, 2020). In subclinical laminitis in cattle, microscopic hemorrhages, impaired keratinization, and a decrease in the quality of the stratum corneum are observed, which creates the prerequisites for the development of secondary lesions, such as plantar ulcers or white line disease (Mudron, 1996; Greenough, 2007).

In sheep and goats, the histological changes are characterized by a combination of lamellar damage with pronounced inflammatory processes, often complicated by bacterial infection, which leads to tissue necrosis and destruction of the stratum corneum (Lischer, 1994).

Pathological and anatomical changes in laminitis reflect hoof damage, changes in internal organs associated with metabolic and inflammatory disorders.

In horses with acute laminitis, pronounced circulatory disorders in the lamellar apparatus are noted, including ischemia, venous stasis and edema, which leads to tissue necrosis and hoof wall detachment (Hood, 1999; Vercelli, 2021). In chronic cases, fibrous changes, tissue remodeling and persistent hoof deformation are formed.

In cattle, pathoanatomical changes are often associated with the sequelae of subclinical laminitis and manifest as plantar hemorrhages, ulcers, abscesses, and hoof deformities, accompanied by changes in the scar, including signs of acidosis, mucosal damage, and the presence of high levels of volatile fatty acids (Nocek, 1997; Enemark, 2008; Mulligan, 2008). Cattle may also exhibit systemic changes, including hepatodystrophy, inflammation, and signs of intoxication associated with endotoxemia (Boosman, 1991; Mulligan, 2008). In sheep and goats, pathological and anatomical changes are characterized by a combination of laminitis with infectious lesions of the hoof, which leads to the development of purulent-necrotic processes, the spread of inflammation to deeper tissues and, in severe cases, to systemic intoxication (Lischer, 1994).

Thus, morphological, histological and pathological and anatomical changes in laminitis in domestic animals form a single pathogenetic complex, in which local damage to the lamellar apparatus is inextricably linked with systemic metabolic, vascular and inflammatory disorders. The specific features of anatomy, physiology and digestion determine the nature and severity of these changes, which must be taken into account in the diagnosis, treatment and prevention of laminitis in horses, cattle, sheep and goats.

Laminitis prevention. Modern approaches to the prevention of laminitis in domestic animals are based on the understanding of it as a systemic multifactorial syndrome, requiring a comprehensive impact on metabolic, nutritional, mechanical and management risk factors.

In horses, control of endocrine status and carbohydrate metabolism is of key importance, since hyperinsulinemia and insulin resistance are considered to be the leading mechanisms for the development of endocrinopathic laminitis. Limiting the intake of non-structural carbohydrates with feed, using diets with a low glycemic index, gradual transition between feeding types and body weight control are basic preventive measures, the effectiveness of which has been confirmed by experimental and clinical studies (Harris, 2006; Asplin, 2007; Bailey, 2013). An important element is regular monitoring of insulin, glucose and lipid profile levels, which allows for timely identification of animals at increased risk of laminitis and early correction of metabolic disorders (Karikoski, 2011; de Laat, 2019). In addition to feeding, conditions of maintenance and operation play a significant role: a sufficient level of physical activity, avoiding prolonged standing on hard surfaces, proper forging and regular correction of hooves ensure optimal load distribution on the limbs and reduce the risk of damage to the lamellar apparatus (O'Grady, 2008; Sadiq, 2020).

In cattle, the prevention of laminitis is closely linked to the control of feeding and the prevention of subclinical ruminal acidosis (SARA), which is one of the key factors in the development of the pathology. Diets should be balanced in terms of fiber and concentrates, with sufficient structured fibers to stimulate the rumen and stabilize the pH of the rumen. The gradual introduction of concentrates, the use of buffering additives and control of the structure of the diet can reduce the risk of acidosis and the associated endotoxemia (Nocek, 1997; Oetzel, 2007; Enemark, 2008).

Herd management is also essential: optimizing housing conditions, providing comfortable flooring, sufficient resting space and regular hoof trimming are critical factors in preventing laminitis and associated hoof horn lesions (Webster, 2001; Bergsten, 2003; Sadiq, 2020).

In high-producing cows, special attention is paid to the transition period, when metabolic stress and dietary changes can contribute to the development of laminitis, which justifies the need for metabolic monitoring and early intervention (Mulligan, 2008; Koziy, 2008). Preventive measures in cattle also have a significant economic effect, as a decrease in the incidence of laminitis is directly related to increased productivity and reduced milk losses (Archer, 2010; Sertu, 2024).

In sheep and goats, the prevention of laminitis has its own characteristics, associated with a combination of metabolic, infectious and mechanical factors. The diet should be adapted to the physiological needs of the animals, limiting the excess intake of concentrates and providing a sufficient amount of roughage, which helps stabilize digestion and reduce the risk of metabolic disorders. Housing conditions play a significant role: dry, clean bedding, regular disinfection of premises and humidity control reduce the risk of infectious lesions of the hoof and the development of secondary laminitis (Lischer, 1994). Regular trimming of hooves, control of the load on the limbs and prevention of injury are necessary components of prevention, especially in conditions of intensive livestock farming. An important modern direction in the prevention of laminitis in all types of domestic animals is the introduction of early monitoring and diagnostic systems. The use of telemetric systems for monitoring activity, gait and behaviour allows for the detection of early changes characteristic of subclinical laminitis, even before the appearance of pronounced clinical signs (Danscher, 2009; Danscher, 2010). The combination of such technologies with laboratory monitoring of biochemical parameters, including insulin, glucose, markers of inflammation and oxidative stress, creates opportunities for an individualized approach to prevention and risk management (Bailey, 2013; de Laat, 2019).

Thus, modern prevention of laminitis in domestic animals is based on an integrated approach that takes into account the species-specific characteristics of digestion, metabolism, housing and exploitation. In horses, the key is the control of endocrine and metabolic factors, in cattle - the prevention of acidosis and the optimization of herd management, while in sheep and goats - a combination of measures regarding feeding, hygiene and infection control. The implementation of these measures allows not only to reduce the incidence of laminitis, but also to improve the overall health of animals and increase the efficiency of livestock farming.

Discussion. Laminitis is a serious disease that affects the performance and health of livestock, particularly horses and cattle. Studies show that the prevalence of lameness in dairy cows can range from 10–40%, depending on housing conditions and feeding systems (Clarkson, 1996; Cook, 2003; Capion, 2009). Laminitis is often associated with metabolic disorders, including insulin resistance and impaired carbohydrate metabolism, as supported by studies in horses and ponies (Asplin, 2007; Bailey, 2013; Stefaniuk, 2023).

One of the key mechanisms of laminitis development is increased prolonged hyperinsulinemia, which stimulates laminar inflammation and destabilization of the hoof basement membrane (Pollitt, 1996; de Laat, 2014; Vercelli, 2021). In cattle, acute and subclinical laminitis often occurs against the background of cicatricial digestive disorders, in particular subclinical cicatricial acidosis (SARA), which causes endotoxin release and inflammatory reactions (Boosman, 1991; Nocek, 1997; Enemark, 2008). Oligofructose overload has also been shown to have a provocative effect on the development of lameness in cattle (Danscher, 2009; Ding, 2020).

Clinical signs of laminitis include behavioral changes, lameness, increased sensitivity to hoof pressure, and loss of performance (Danscher, 2010; Archer, 2010). In addition, there is evidence of an association between infectious processes such as mastitis and the development of acute laminitis in cows (Zhao, 2020; Faustmann, 2025).

Prevention and control of the disease are based on a comprehensive approach: diet optimization, body weight control, restriction of high-glycemic feed intake, correction of metabolic disorders, regular hoof examination and shoeing (Webster, 2001; Harris, 2006; O'Grady, 2008). In addition, early diagnosis is an important aspect, which reduces the risk of chronic laminitis and loss of productivity (Greenough, 2007; Koziy, 2008; Bojkovski, 2023).

Current research highlights that laminitis is a multifactorial disease where genetic, metabolic, inflammatory and management factors interact. Further research should focus on the molecular mechanisms of inflammation in lamellar tissue, interactions with metabolic disorders and the development of new prevention and treatment strategies (de Laat, 2019; Delarocque, 2021; Menzies-Gow, 2025).

Thus, timely diagnosis, control of metabolic disorders and comprehensive preventive measures allow not only to reduce the risk of laminitis, but also to increase productivity and ensure the well-being of domestic animals.

Conclusions. Laminitis in domestic animals is a complex systemic multifactorial syndrome, the development of which is due to the interaction of metabolic, endocrine, infectious-toxic and mechanical factors. The key role in the pathogenesis is played by impaired microcirculation of the lamellar apparatus, degradation of the dermal-epidermal junction and activation of inflammatory and oxidative processes, which leads to the destruction of the hoof apparatus and reflects systemic changes in the body. The disease has a progressive nature: from subclinical metabolic and biochemical changes (increased insulin, glucose, markers of inflammation and oxidative stress) to clinically pronounced forms with deep structural changes, hoof deformation, tissue necrosis and chronic destruction of lamellae. Clear species-specific features of the pathogenesis of laminitis have been established: in horses, endocrine and metabolic disorders (hyperinsulinemia, insulin resistance) prevail, in cattle - subclinical cicatricial acidosis with endotoxemia and systemic inflammatory response, in sheep and goats - the combined effect of metabolic, infectious and mechanical factors, which determines the variability of manifestations. Differential diagnostics of laminitis should be based on a comprehensive approach, including the assessment of clinical signs and stage of the course, analysis of biochemical indicators, as well as morphological, histological and pathological-anatomical studies, which allows for timely detection of the disease and distinguishing it from other hoof pathologies. Effective prevention and control of laminitis are possible only under the conditions of a comprehensive approach, which involves optimizing feeding, controlling metabolic status, improving housing conditions and using modern monitoring methods. Early diagnosis of subclinical forms is key to preventing the development of severe and chronic stages and reducing economic losses.

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Ламініт як чинник метаболічних порушень у свійських тварин (оглядова стаття)

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Резюме. У статті проведено комплексний аналіз сучасних наукових даних щодо ламініту у свійських тварин із урахуванням видових особливостей перебігу патологічного процесу у коней, великої рогатої худоби, овець і кіз. Узагальнено підходи до розуміння ламініту як системного мультифакторного синдрому, який формується внаслідок взаємодії метаболічних, ендокринних, інфекційно-токсичних та механічних чинників. Показано, що розвиток захворювання супроводжується глибокими порушеннями мікроциркуляції у ламелярному апараті копита, деградацією дермально-епідермального з'єднання, активацією запальних і в кінцевому результаті призводить до структурної деструкції копитного апарату та зниження продуктивності тварин.

Особливу увагу приділено аналізу патогенетичних механізмів із урахуванням типу травлення. Встановлено, що у коней, як моногастричних тварин, провідну роль відіграють ендокринно-метаболічні порушення, зокрема гіперінсулінемія, інсулінорезистентність і дисбаланс вуглеводного обміну, що обумовлює розвиток ендокринопатичного ламініту. У великої рогатої худоби ключовим пусковим механізмом є субклінічний рубцевий ацидоз, що супроводжується змінами мікробіоти рубця, підвищенням рівня ендотоксинів у крові та розвитком системної запальної відповіді. У овець і кіз ламініт формується внаслідок поєднання метаболічних, інфекційних і механічних чинників та визначає варіабельність клінічних і морфологічних проявів захворювання.

Систематизовано біохімічні зміни, характерні для різних стадій ламініту (субклінічної, гострої та хронічної), включаючи підвищення концентрації інсуліну, глюкози, тригліцеридів, активацію ферментів тканинного ушкодження (лактатдегідрогенази, креатинкінази), прозапальних цитокінів, С-реактивного білку. Показано, що ці показники можуть використовуватися для ранньої діагностики, диференціації стадій захворювання та прогнозування його перебігу. Також узагальнено сучасні підходи до діагностики ламініту, що базуються на поєднанні клінічного обстеження, лабораторних досліджень і інструментальних методів, зокрема рентгенографії, ультразвукової діагностики, комп'ютерної томографії та систем моніторингу рухової активності. Наведено характеристику морфологічних, гістологічних і патолого-анатомічних змін, які відображають прогресування патологічного процесу від початкових мікроскопічних ушкоджень до вираженої деформації копита, некрозу тканин і фіброзу.

Водночас, окремо розглянуто сучасні підходи до профілактики ламініту, які включають оптимізацію годівлі, контроль метаболічного статусу, запобігання субклінічному ацидозу, покращення умов утримання та впровадження технологій раннього моніторингу. Підкреслено, що ефективне управління ризиками ламініту можливе лише за умов інтегрованого підходу, який враховує видові особливості тварин, рівень їх працездатності для коней і продуктивності для великої рогатої худоби та технологічні умови утримання.

Результати узагальнення можуть бути використані для удосконалення системи ранньої діагностики, профілактики та контролю ламініту і сприятиме підвищенню продуктивності і покращенню добробуту свійських тварин.

Ключові слова: копито, запальний процес, морфологічні зміни, ацидоз, ендотоксинемія, порушення мікроциркуляції, обмін речовин, кульгавість.

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