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Current Developments in Equine Medicine – Highlights from the Leipzig Veterinary Congress 2026

Vitamin E in the sport horse – nutritional relevance and deficiency pathology

Vitamin E is a fat-soluble micronutrient essential in equine nutrition. Physiologically, almost exclusively α-**tocopherol** is relevant, with the natural form **RRR**-α-**tocopherol** having the highest bioavailability due to its preferential hepatic processing.

As a lipid-soluble antioxidant, vitamin E protects cell membranes from oxidative damage and is of central importance for neuromuscular function. During physical exertion, vitamin E turnover increases due to greater formation of reactive oxygen species.

Vitamin E is synthesised exclusively by plants and is present in high concentrations in fresh grass. Preserved forage shows significantly reduced levels due to storage and oxidation processes, meaning horses without access to pasture cannot adequately meet their requirements.

The recommended daily requirement is **1–2 mg/kg body mass**, and at least **2 mg/kg body mass** for sport horses. In cases of increased oxidative stress or muscular disease, **4–6 mg/kg body mass** is recommended.

A chronic vitamin E deficiency can lead to severe neuromuscular diseases. These include **vitamin E deficient myopathy** with degenerative muscle changes, **Equine Motor Neuron Disease (EMND)** with progressive degeneration of motor neurones, and **Neuroaxonal Dystrophy/Equine Degenerative Myeloencephalopathy (NAD/EDM)**, a neurodegenerative disease of young horses in which vitamin E deficiency is considered a key predisposing factor.

Vitamin status is assessed via **serum α-tocopherol** concentration; values above **2.0 µg/ml** are considered adequate.

In the absence of grass intake, supplementation is mandatory. **Natural RRR-α-tocopherol** is superior to synthetic forms in terms of bioavailability. However, supplementation beyond requirements **does not show consistent benefits** in healthy sport horses regarding performance or markers of oxidative stress

Picture source: envatoelements

Myopathies in Icelandic horses – laboratory diagnosis and clinical relevance

Myopathies are an increasingly recognised cause of reduced performance and non-specific lameness in Icelandic horses. Due to breed-specific biomechanical characteristics and subtle clinical signs, muscular disorders are often diagnosed late. A recently described **chronic idiopathic myopathy** appears to represent an Icelandic horse-specific entity.

Affected horses mainly show non-specific clinical signs such as reduced performance, diffuse lameness, stumbling, increased respiratory rate, and reduced willingness to perform certain gaits. Acute clinical signs of classical exertional rhabdomyolysis are usually absent.

LABOklin LABORATORY FOR CLINICAL DIAGNOSTICS GMBH & CO. KG
Steubenstr. 4 • 97688 Bad Kissingen (Germany) • Tel.: +49 971 720 20 • Fax: +49 971 6 85 46 • E-mail: info@laboklin.com

Serum creatine kinase (CK) is the most important laboratory marker. Resting values are often normal or only mildly elevated. A diagnostically key finding is **exercise-dependent CK increase**, measured in a standardised manner at **4 and 24 hours** post-exercise. Peak CK values typically range between **2000–3000 U/L** and are therefore significantly lower than in horses with recurrent exertional rhabdomyolysis (RER).

CK kinetics have greater diagnostic value than single measurements. Reproducible, moderate post-exercise increases support a myopathic origin, even when absolute values are only mildly elevated. In addition, **aspartate aminotransferase (AST)** may be used to assess longer-standing muscle damage. Inflammatory parameters and metabolic routine profiles are primarily used to rule out systemic or inflammatory diseases.

Definitive diagnosis is confirmed via **muscle biopsy** from the semimembranosus muscle. Histopathology shows chronic degenerative and regenerative changes with centralised nuclei, without evidence of abnormal glycogen storage or myofibrillar defects.

In Icelandic horses with reduced performance, **exercise-dependent CK kinetics** should be included early in the diagnostic work-up. In combination with muscle biopsy, this enables reliable differentiation between myopathic and non-myopathic causes and provides a key basis for clinical decision-making.

Fever in horses: the role of laboratory diagnostics in clinical decision-making

Fever in horses is a common but non-specific clinical sign, the aetiological investigation of which requires a structured diagnostic approach (Fig. 1). The aim is the early identification of infectious or systemic diseases that require targeted treatment or further measures.

Following history-taking and clinical examination, **laboratory diagnostics** play a central role. Haematological and clinical chemistry tests allow an initial assessment of the inflammatory process as well as indications of possible organ involvement. **Acute-phase proteins** are of particular diagnostic importance in this context. **Serum amyloid A (SAA)**, as the horse's major acute-phase protein, responds rapidly and with high sensitivity to inflammatory stimuli and is suitable both for detecting acute processes and for monitoring disease progression. **Fibrinogen** shows a delayed, longer-lasting increase and is particularly relevant in subacute or chronic inflammation. Due to their high sensitivity but lack of specificity, acute-phase proteins must always be interpreted in a clinical context.

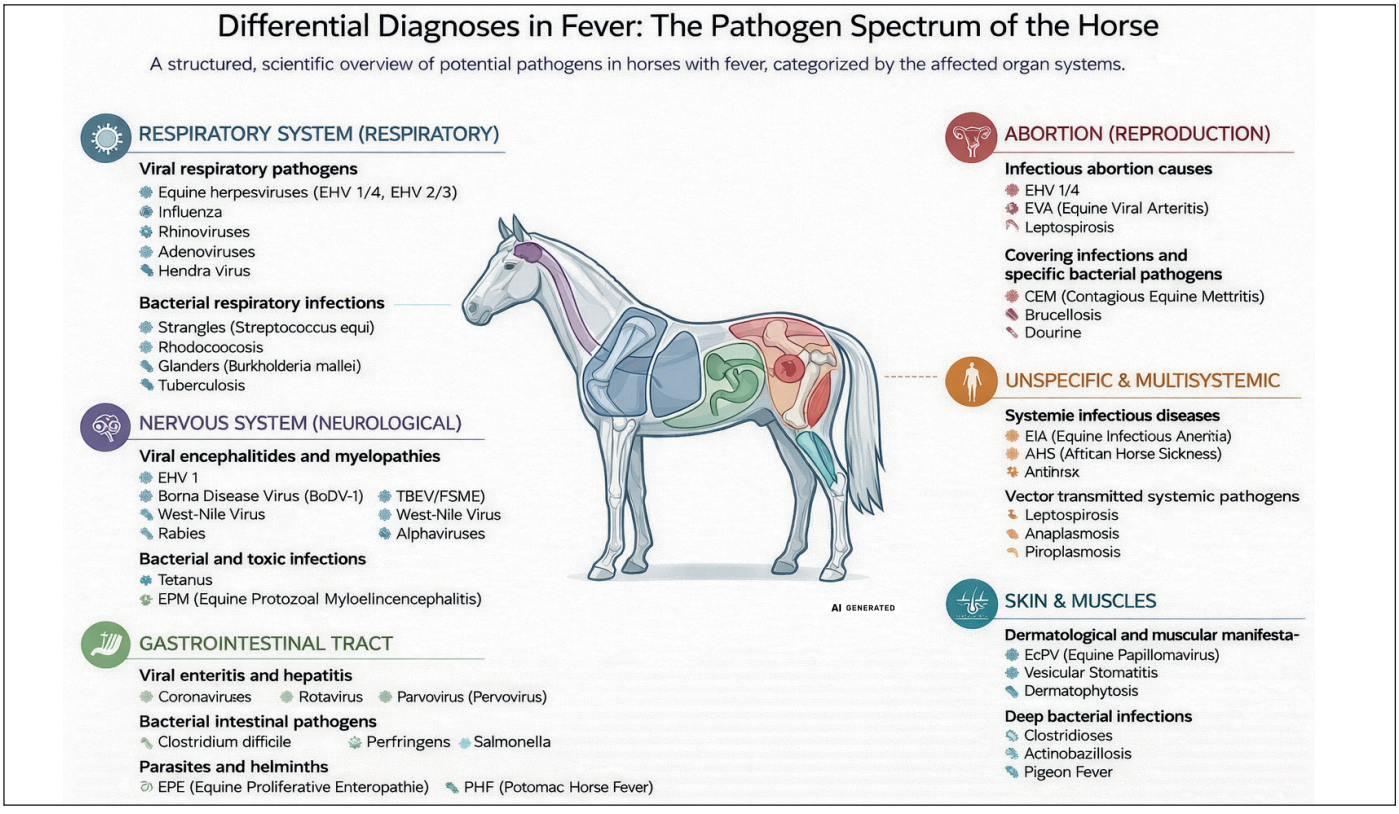

Fig. 1: Differential diagnoses of the most common pathogens in horses with fever, grouped by organ system *Picture source: Gemini AI*

For the aetiological investigation of infectious causes, specific laboratory diagnostic methods are required. **Molecular detection techniques**, in particular **PCR-based methods** in accredited laboratories, are considered the gold standard for direct pathogen detection. They are characterised by high analytical sensitivity and specificity and enable targeted differentiation of relevant bacterial and viral pathogens. **Point-of-care tests** can provide a rapid initial indication, but they neither replace comprehensive laboratory diagnostics nor quality-assured interpretation of results by specialised laboratories.

Depending on the laboratory findings and the clinical course, further diagnostic measures, including imaging techniques or targeted sampling, may be required. Overall, laboratory diagnostics represent an indispensable component of fever investigation in horses and form the basis for evidence-based treatment decisions as well as herd protection measures.

Genetic testing in equine myopathies: evidence-based application and genetic limitations

Genetic testing has become an established diagnostic tool in equine myopathies; however, it is currently carried out without binding regulatory oversight. This places veterinarians under the obligation to critically assess the validity of commercially available tests. A genetic test should only be used clinically if the underlying variant is demonstrably causally associated with the disease and meets internationally recognised validation criteria.

Fundamental requirements for valid genetic tests include a low allele frequency in the general population, a high genotype–phenotype concordance, a functionally relevant effect of the variant on the encoded protein, and reproducibility of results in peer-reviewed studies. To date, only a small number of genetic tests for equine myopathies meet these criteria.

Currently, there are five to six validated genetic tests for muscle disorders in horses, including **polysaccharide storage myopathy type 1 (PSSM1; GYS1), myosin heavy chain myopathy (MYHM; MYH1), hyperkalaemic periodic paralysis (HYPP; SCN4A), malignant hyperthermia (MH; RYR1), and glycogen branching enzyme deficiency (GBED; GBE1)**.

Identification of the causal variants was based on strictly phenotyped case–control studies using gold-standard diagnostics such as muscle histopathology or electromyography. The respective mutations show a strong disease association, low prevalence in healthy populations, and a clear functional effect on the affected protein.

In contrast, there are commercial genetic test panels for so-called **type 2 polysaccharide storage myopathies (PSSM2), myofibrillar myopathies (MFM), muscle integrity myopathy (MIM),** and **recurrent exertional rhabdomyolysis (RER)**. These tests are based on variants in genes such as *MYOT, FLNC, MYOZ3, PYROXD1, CACNA2D3*, and *COL6A3*, whose pathogenic relevance has not yet been convincingly demonstrated.

Several independent studies have failed to show a significant association between these variants and histopathologically confirmed myopathies. The prevalence of the tested variants was similar in affected horses and healthy controls, leading to high rates of false-positive and false-negative results. Particularly notable is the high allele frequency of these variants in the general population: population genetic analyses show that up to 50% of Warmblood horses carry at least one of these variants. Given the much lower prevalence of clinically manifest muscle diseases, a causal role is unlikely; rather, these are probably benign genetic polymorphisms.

Evidence is also limited for the genetic diagnosis of **recurrent exertional rhabdomyolysis**. The so-called Px variant, which has been proposed as causal, is synonymous and does not lead to an amino acid change. Its high prevalence in healthy Thoroughbreds contradicts a causal pathogenic role.

In summary, genetic tests in equine myopathies should only be used when they are strictly validated and in agreement with breed, clinical presentation, and laboratory and histopathological findings. Uncritical use of non-validated genetic test panels carries a significant risk of misdiagnosis and inappropriate selective breeding.

Development of new diagnostic tests for infectious endometritis in mares

Infectious endometritis is one of the most common causes of subfertility and infertility in mares. It occurs either as a persistent inflammatory response following covering or as a chronic bacterial infection. While the majority of mares effectively eliminate uterine bacterial contamination within 6–24 hours, approximately 10–15% develop chronic endometritis, which prevents successful pregnancy.

The classical diagnosis of infectious endometritis is based on **bacterial culture, cytological examination**, and **molecular biological methods** such as PCR. However, these methods primarily detect planktonic, metabolically active bacteria. Recurrent infections may still occur because pathogens can persist either in **anatomical reservoirs** (e.g. the clitoral fossa), in a

metabolically inactive (dormant) state deep within the endometrium, or as **bacterial biofilms**. These forms often evade both conventional diagnostics and antimicrobial treatment.

To detect dormant, persistent bacteria, a **diagnostic–therapeutic activation concept** has been developed, in which dormant bacteria in the endometrium are stimulated so that they can subsequently be detected using conventional culture or PCR and made accessible to treatment. This approach represents a significant advance in the diagnosis of subclinical and recurrent endometritis.

A previously unresolved diagnostic problem is the **reliable in vivo detection of bacterial biofilms** in the uterus. Biofilms effectively protect bacteria from the immune system and antimicrobial agents and promote the development of antimicrobial resistance. Although it has been experimentally demonstrated that common equine endometritis pathogens such as Streptococcus equi subsp. zooepidemicus, Escherichia coli, and Pseudomonas aeruginosa are capable of forming biofilms, there is currently no established clinical diagnostic method for uterine biofilms.

Current research approaches focus on detecting specific **components of the biofilm matrix** in low-volume uterine lavage samples. This matrix consists of proteins, lipids, polysaccharides, and nucleic acids. Proteomic and lipidomic analyses have already identified differences between planktonic and biofilm-forming bacteria, including biofilm-specific proteins and membrane lipids with potential diagnostic relevance. The aim is to develop a robust, clinically applicable test that enables targeted diagnosis and thus a differentiated, biofilm-specific therapy.

In summary, in cases of recurrent infectious endometritis, not only free-living bacteria but also **dormant persistent organisms** and **bacterial biofilms** should be considered in the diagnostic evaluation. The development of biofilm-specific diagnostic methods represents a crucial step towards optimising therapy and enabling more targeted and responsible use of antibiotics.

A selection of our services relating to the topics above:

- Individual vitamin analyses and vitamin profiles (basic and comprehensive)
- Basic and extended muscle screening
- Various PCR panels for pathogen detection by organ system (e.g. respiratory tract I–IV)
- A wide range of genetic tests (e.g. polysaccharide storage myopathy type 1 (PSSM1), immune-mediated myositis & MYH1 myopathy (MYHM), hyperkalaemic periodic paralysis (HYPP), equine malignant hyperthermia (EMH), glycogen branching enzyme deficiency (GBED))
- Breeding hygiene services

Jana Pohl

Further reading is available here:

https://short.laboklin.com/lit_lapf0526_de