

BIOENZYMATIC MYCOTOXIN DETOXIFICATION IN RUMINANTS: ESSTENCE MICROBIAL PATHWAYS, RUMEN PHYSIOLOGICAL STABILIZATION, AND MILK SAFETY

Interpretation for Company and Farm Manager

Interpretation for Veterinarians

Foie-gras duck—freshly slaughtered gastrointestinal tract; control vs. Esstence watering, 2018 Kiskunmajsa



INTERPRETATION FOR COMPANY AND FARM MANAGERS

LIMITATIONS OF THE TRADITIONAL APPROACH

The production performance of ruminants (cattle, sheep, goats) has traditionally been linked to feed energy content and rumen fermentation.

In recent years, however, it has become clear that fluctuations in production, digestive problems, and fertility disorders are driven not only by feeding practices but also by **biological imbalance**.

Fungal toxins—aflatoxin, zearalenone, ochratoxin, fumonisin, T-2 toxin—can be detected in almost all plant-based feed ingredients, and while the rumen can partially neutralize them, **this does not occur under all conditions**:

- pH shifts,
- feed changes,
- stress,
- or higher toxin concentrations can **collapse the rumen flora's defenses**.

At that point, toxins pass through the rumen system and reach the **liver, immune system, and mammary gland**. The consequences are **metabolic load, inflammation, deterioration of milk quality, and reproductive disorders**.

WHY "GOOD RUMEN FLORA" OR TRADITIONAL BINDERS ARE NOT ENOUGH

Many farm managers rightly say: "Our rumen flora is good; the cows eat well."

Yes—but rumen flora is not all-powerful. If rumen pH or microbial composition shifts (e.g., concentrate dominance, heat stress, feed transition), the **defense functions are the first to be impaired**. Toxins then pass through the rumen and, via the bloodstream, **end up in the liver**.

Traditional adsorbents (clay, zeolite, activated carbon) can capture this only partially—the molecular structure of most toxins is too small or does not adhere adequately. Moreover, these substances can also bind **vitamins and trace elements**, which further compromises the animal's metabolism.

In other words, the traditional approach is **symptomatic treatment**—it does **not** address the root of the biological problem.

THE BIOLOGICAL APPROACH: ACTIVE DEGRADATION, NOT PASSIVE BINDING

A biology-driven system, such as the **Esstence** technology, is not merely an additive but a **living microbial defense mechanism**.

The microorganisms present in the system (*Bacillus*, *Trichoderma*, *Lactobacillus*, *Aspergillus*, *Rhodococcus*) produce enzymes that **biochemically transform** toxins rather than merely "covering" them.

This process occurs at the **rumen–small intestine interface**, where:

- toxins are **broken down** into less reactive molecules,
 - the animal's body **does not absorb** them,
 - and the microflora **restores fermentative balance**.
- The end result: the organism **is not burdened but regenerates**.

IMPACT OF BIOLOGICAL FUNCTION ON THE RUMEN–LIVER–IMMUNE AXIS

In ruminants, the effect of biological protection manifests on three levels:

I. Rumen stabilization

The biological system supports cellulolytic bacteria and reduces pH fluctuations.

Thus, fiber degradation improves in the rumen, feed energy is utilized more efficiently, and the risk of acidosis decreases.

Consequence: more stable rumen fermentation and more even milk production.

II. Liver and metabolic protection

Degraded toxins no longer burden the liver, making protein, fat, and carbohydrate metabolism more efficient. Liver functions (e.g., ammonia clearance, hormone metabolism) are restored.

Consequence: better appetite, balanced milk yield, stable milk composition.

III. Immune stabilization

Microbial balance and antioxidant effects reduce inflammatory reactions.

The body spends **less energy on defense** and **more on production**.

Consequence: fewer stress symptoms, improved vaccine response, stronger reproductive metrics.

EXPECTED EFFECTS OF THE BIOLOGICAL SYSTEM ON HERD PERFORMANCE AND STABILITY

Based on literature and biological observations, applying bioenzymatic systems in ruminants supports the following processes:

Mechanism of action	Physiological outcome	Production consequence
Toxin degradation and microbial protection	Reduced toxin load on liver and gut	more consistent milk yield, better feed utilization
Rumen pH stabilization	More efficient fiber breakdown, improved fermentation	reduced acidosis, higher energy status
Improved liver function	Better ammonia and lipid metabolism	stable body condition, fewer metabolic diseases
Immune support	Decrease in inflammatory markers	less stress, better reproductive ratios
Antioxidant and microbial regeneration	Reduced cellular oxidative stress	more stable milk quality and longer productive lifespan



ECONOMIC INTERPRETATION FROM A MANAGEMENT PERSPECTIVE

Biology-based animal husbandry is **not an extra cost** but a **preventive investment**.

The system does **not add energy** to the feed; it **releases the energy** the body previously had to spend on defense.

A healthy rumen flora, a less burdened liver, and a stable immune system deliver **higher utilization even from less feed**.

Production becomes more predictable, animals recover faster, and stress situations (heat stress, feed transition, post-calving period) are better tolerated.

For managers, this translates into **not only biological but also financial stability**:

- more predictable production cycles,
- fewer losses,
- longer productive lifespan.

SUMMARY

The future of ruminant production lies not in increasing feeding intensity but in **preserving biological balance**. Biology-based feeding is **not a new additive** but a **new operating principle**: it does not treat symptoms but **eliminates root causes**—toxic stress and microbial imbalance.

Such a system does not “speed up” production; it **eliminates internal losses**, so the animal’s energy is directed not to defense but to **growth and regeneration**.

Microbial–enzymatic systems can:

- prevent the physiological consequences of toxic load,
- restore the balance of the **gut–liver–immune axis**,
- and support the body’s **own regenerative mechanisms**.

Thus, the biological approach is **not an alternative** to traditional husbandry but its **natural and sustainable complement**—ensuring production stability **through the animal’s health**. In this way, the system does not further burden the animal but **serves its physiology**, so **growth and production efficiency are natural**, not “forced.”

INTERPRETATION FOR VETERINARIANS

Ruminant animals, although partially capable of degrading mycotoxins via the rumen microbiome, are still exposed to toxin load in many cases.

The main risk compounds—aflatoxin B₁ (AFB₁) and its milk metabolite (AFM₁), zearalenone (ZEN), ochratoxin A (OTA), deoxynivalenol (DON), fumonisin B₁ (FB₁), T-2/HT-2 toxins, and patulin—enter the body through feed materials (Ji et al., 2016; Liu et al., 2024).

These toxins induce subclinical hepatic, renal, and reproductive disorders, as well as deterioration of milk and meat quality (Abraham et al., 2022).

Traditional degradation based on rumen fermentation is often insufficient, especially under high toxin concentrations, pH shifts, or feed transitions (Nguyen et al., 2024).

This justifies the development of microbial–enzymatic systems that, by supporting the natural function of the rumen microbiota, actively degrade toxins and thereby prevent systemic absorption.

THEORETICAL BACKGROUND OF MICROBIAL BIO-DETOXIFICATION

In the ruminant organism, mycotoxin degradation occurs on two levels:

- (1) through the partial inactivating activity of the rumen microbial community, and
- (2) via complementary exogenous microorganisms and enzymes that enhance degradation capacity (Liu et al., 2024; Sun et al., 2023).

Active contributors include strains of *Bacillus subtilis*, *Trichoderma harzianum*, *Lactobacillus plantarum*, *Aspergillus niger*, *Pseudomonas putida*, and *Rhodococcus erythropolis*, which produce enzymes such as CotA laccase, DyP peroxidase, carboxypeptidase, amidase, dehydrogenase, esterase, and epoxidase (Gao et al., 2024; Selim et al., 2024). These enzymes remain active even in the slightly reductive rumen environment with a pH of 6.2–6.8 and convert mycotoxins into less toxic, biologically inactive metabolites (Jia et al., 2024).

BIOCHEMICAL PATHWAYS AND ENZYME SYSTEMS (See: tabulated appendix)

I. Aflatoxin B₁ and M₁

CotA laccase and F420H₂ reductase produced by *Bacillus subtilis* and *Rhodococcus erythropolis* transform the 8,9-epoxide of AFB₁ into a dihydrodiol via oxidative ring opening (Gao et al., 2024). The resulting AFD₁ and AFD₂ metabolites no longer exhibit mutagenic effects and do not convert to AFM₁ in the mammary gland (Sun et al., 2023). The physiological consequence is decreased liver enzyme levels, up to a 60% reduction in milk AFM₁ contamination, and a 5–8% improvement in milk yield (Nguyen et al., 2024).

II. Zearalenone

The laccase and peroxidase systems of *Trichoderma harzianum* and *Aspergillus niger* degrade the estrogen-active structure of ZEN via aromatic oxidation, while esterases hydrolyze the lactone bond (Jia et al., 2024). The outcome is the formation of non-estrogenic metabolites, which restores the progesterone–estrogen ratio and thus improves fertility and lactational stability (Liu et al., 2024).

III. Ochratoxin A

Carboxypeptidases and amidases from *Bacillus licheniformis* and *Lactobacillus plantarum* cleave the phenylalanine side chain of OTA (Abraham et al., 2022). The resulting OTα metabolite does not bind to plasma albumin, is rapidly eliminated, and the toxic effect on renal tubules ceases. Clinically, this manifests as improved urinary excretion parameters and stable feed intake.

IV. Deoxynivalenol

Enzyme systems (epoxide reductase, dehydrogenase) of *Streptomyces griseus*, *Bacillus subtilis*, and *Lactobacillus casei* convert DON to DON-ol (Selim et al., 2024). This form no longer inhibits ribosomal peptide synthesis, thereby reducing the inflammatory cytokine response (IL-6, TNF- α) and restoring the balance of rumen fermentation (Sun et al., 2023). Some studies have also demonstrated an 8–12% reduction in methane emissions due to normalized fermentation (Nguyen et al., 2024).

V. Fumonisin B₁

Aminotransferases from *Streptomyces* spp. and *Aspergillus niger* produce HFB₁ via C2-deamination and carboxyl hydrolysis, which no longer disrupts sphingolipid synthesis (Ji et al., 2016). This improves hepatic and neural membrane integrity and stabilizes milk fat levels.

VI. T-2 / HT-2 toxins and Patulin

Enzymes of *Rhodococcus erythropolis* and *Trichoderma harzianum* open the epoxide ring and deacetylate, reducing cytotoxicity (Selim et al., 2024). The lactonase systems of *Aspergillus niger* convert patulin to an ascorbate analogue, thereby enhancing antioxidant capacity (Abraham et al., 2022).

PHYSIOLOGICAL REGENERATION AND PROTECTION OF THE RUMEN–LIVER–IMMUNE AXIS

Bioenzymatic degradation not only inactivates toxins but also induces functional regeneration of the organism:

1. **Rumen stabilization:** pH fluctuations diminish, the proportion of cellulolytic bacteria increases, and nitrogen utilization and fermentative efficiency improve (Sun et al., 2023).
2. **Liver regeneration:** lipid peroxidation and glutathione-oxidative stress decrease; mitochondrial activity of hepatocytes normalizes (Liu et al., 2024).
3. **Immune homeostasis:** IL-10 increases and TNF- α decreases, which suppresses chronic inflammation and improves vaccine response intensity (Abraham et al., 2022).
4. **Milk and meat quality:** AFM₁ levels in milk may decrease by up to 60%, while fat and protein fractions remain stable (Nguyen et al., 2024).

Together, these processes restore the homeostasis of the rumen–liver–immune axis, which in ruminants is the basis of metabolic health and production efficiency.

CLINICAL CONSIDERATIONS AND VETERINARY RELEVANCE

In ruminants, mycotoxin load often appears in a latent, chronic form, which reduces reproductive performance and the consistency of milk production. In contrast, the microbial–enzymatic approach supports the rumen microbiota’s natural defense mechanisms, does not impair nutrient absorption, and maintains the organism’s redox balance (Liu et al., 2024).

According to clinical observations, in herds applying bioenzymatic systems:

- the stability of milk yield increased,
- milk AFM₁ levels decreased by 40–60%,
- intestinal and hepatic enzyme profiles normalized,
- fertility and calving rates improved (Sun et al., 2023; Nguyen et al., 2024).

Such biological systems therefore provide complex, synergistic protection that combines toxin inactivation with physiological-level regeneration—while supporting antibiotic-free, sustainable milk and meat production.

SUMMARY

Mycotoxin management in ruminants can no longer be based exclusively on binders. According to scientific consensus, **bioenzymatic detoxification**—which, in cooperation with the rumen microbiome, employs multiple enzyme systems (laccase, peroxidase, amidase, esterase, dehydrogenase)—is capable of **chemical deactivation of toxins, functional restoration** of the organism, and **improved milk safety**.

In veterinary practice, this mechanism creates a new opportunity to manage chronic mycotoxin load and, in the long term, stabilizes the metabolic health of dairy and beef ruminant populations (Liu et al., 2024; Abraham et al., 2022).

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RUMINANT ANIMALS								
Esistence Bio-Enzymatic Mycotoxin Detoxification Potential								
Mycotoxin	Without Esistence: effect of the toxin on the animal's body	Microorganisms proven effective in publications	Taxa identified in Esistence	Biochemical detoxification pathway	Result of the enzymatic/mechanistic process in the animal + combined Esistence effect	Literature efficiency (%)	Expected Esistence effect	References (author + year)
Aflatoxin B ₁ / M ₁	Hepatotoxicity, milk-M ₁ contamination, performance decline	B. subtilis / licheniformis, Trametes / Cerrena, Mycobacterium	B. subtilis, R. erythropolis, P. putida	8,9-epoxide → dihydrodiol; lactone hydrolysis; inhibition of M ₁ formation	Epoxide opening → reduced milk- M ₁ source; Combined effect: milk- M ₁ -40–60%, liver function improvement, milk yield +5–8%	70–98	80–90%	Sun 2023; Liu 2024; Nguyen 2024; Gao 2024; Abraham 2022; Ji 2016
Zearalenone (ZEN)	Estrogenic effect, reduced fertility / milk yield	Trichoderma, Aspergillus niger, Bacillus	T. harzianum, A. niger, B. amyloliquefaciens	Aromatic oxidation + ester hydrolysis	Decreased estrogenic activity; Combined effect: improved fertility, stabilized milk yield	60–100	75–85%	Sun 2023; Liu 2024; Gao 2024; Jia 2024; Ji 2016
Ochratoxin A (OTA)	Nephrotoxicity, rumen flora inhibition, reduced feed intake	B. licheniformis, L. plantarum, A. niger	B. licheniformis, L. plantarum, A. niger	Phenylalanine cleavage (OTA → OTα)	OTα → reduced nephrotoxicity and microbial inhibition; Combined effect: stable rumen, improved ammonia turnover	50–85	70–80%	Sun 2023; Liu 2024; Abraham 2022; Ji 2016
Deoxynivalenol (DON)	Disturbed rumen fermentation, increased CH ₄ , loss of appetite	Streptomyces, Bacillus, Lactobacillus	S. griseus, B. subtilis, L. casei	Epoxide reduction → DON-ol / conjugates	DON-ol → reduced ribotoxicity; Combined effect: restored fermentation balance, CH ₄ -8–12%	60–80	75–80%	Sun 2023; Liu 2024; Selim 2024; Ji 2016
Fumonisin B ₁ (FB ₁)	Hepatic / neurological damage, reduced milk fat	Sphingopyxis / Sphingomonas, Streptomyces, Bacillus, A. niger	Streptomyces spp., B. cereus, A. niger	Side-chain hydrolysis → HFB ₁ ; C2- deamination	HFB ₁ less toxic; Combined effect: liver membrane regeneration, stable milk yield	55–90	80–90%	Abraham 2022; Sun 2023; Ji 2016
T-2 / HT-2	Rumen epithelial membrane damage, reduced appetite	Rhodococcus, Trichoderma, Pseudomonas	R. erythropolis, T. harzianum, P. putida	Epoxide opening + deacetylation	Epoxide opening → decreased cytotoxicity; Combined effect: rumen epithelial membrane restoration, increased feed intake	60–85	75–85%	Sun 2023; Selim 2024; Ji 2016
Patulin	Inhibition of gut flora, off-flavor/odor in milk	Candida, Aspergillus niger	Candida spp., A. niger	Lactone ring hydrolysis → ascorbate derivative	Lactone cleavage → mucosal protection; Combined effect: improved milk quality, stabilized gut homeostasis	70–95	85–90%	Sun 2023; Abraham 2022; Ji 2016