

## BIOENZYMATIC MYCOTOXIN DETOXIFICATION IN MONOGASTRIC ANIMALS: ESSTENCE—MICROBIOLOGICAL MECHANISMS AND PHYSIOLOGICAL REGENERATION

**Interpretation for Company and Farm Managers**

**Interpretation for Veterinarians**

*Freshly slaughtered foie-gras duck—post-slaughter gastrointestinal tract; control vs. Esstence watering, 2018, Kiskunmajsa*



## INTERPRETATION FOR COMPANY AND FARM MANAGERS

### THE NEW LIMITING FACTOR IN MODERN LIVESTOCK PRODUCTION: INTERNAL LOAD

In today's monogastric production (poultry, swine, rabbit), performance is determined not primarily by genetics or feed energy, but by how well the animal can handle the biological–biochemical load coming from the environment and the feed.

The most common yet often unnoticed problem is **mycotoxin load**:

- even at low doses, toxins cause continuous stress in the gut and liver,
- they impair nutrient absorption,
- they weaken immune responses and reduce vaccine efficacy,
- and they maintain chronic inflammation in the body.

Taken together, these factors amount to a **5–15% performance loss**, which conventional feeding can hardly offset.

### WHY TRADITIONAL TOXIN BINDING IS NOT ENOUGH

Classical solutions (clays, zeolites, activated charcoal) are mainly **adsorbents** that bind part of the toxins in the gut.

However, such binding:

- does **not degrade** the toxin, it only fixes it physically,
- is **not selective**, so some vitamins and amino acids also get adsorbed,
- and **does not work across chemical differences** among mycotoxins (e.g., zearalenone and DON cannot be bound the same way).

Farm experience is that binders **stabilize short-term**, but **do not resolve** the physiological impacts of toxin load—the liver remains overloaded and the immune system weakened.

### THE BIOLOGICAL APPROACH: DEGRADATION, NOT CONCEALMENT

Biology-based systems, such as the **Esstence** technology, are not “passive binders” but **active, live biological systems** that, with the help of microorganisms and enzymes, **break down toxins within the digestive tract**.

The key is **bioenzymatic degradation**, which:

- recognizes the reactive ring structures of the toxin molecules (epoxide, lactone, aromatic groups),
- enzymatically opens or transforms them,
- removes the chemical portion that triggers toxicity,
- thereby rendering the molecule **biologically inactive before it can be absorbed**.

This **relieves the liver and the gastrointestinal tract**, allowing the body not to suppress but **to process** the load.

## WHAT HAPPENS IN THE ANIMAL AS A RESULT OF BIOLOGICAL DEGRADATION

The biological system acts on **three levels**:

### I. Gut-flora stabilization

Beneficial bacteria (Lactobacillus, Bacillus, Trichoderma) outcompete pathogenic species, the gut microbial balance is restored, and nutrient absorption and digestive efficiency improve.  
→ The animal **utilizes the same feed better**, and **less inflammation** develops.

### II. Liver and metabolic protection

Because toxins are degraded, **hepatic oxidative load decreases**, protein and energy metabolism normalize, and hormonal balance (e.g., in zearalenone exposure) is restored.  
→ The animal **recovers faster**, with **improved fertility and appetite**.

### III. Immune balance

The immune system is no longer in a state of “constant defense”; the **IL-10 / TNF- $\alpha$  ratio** is restored.  
→ This means **better vaccine responses**, **fewer stress reactions**, and **lower antibiotic need**.

## EXPECTED IMPACTS OF THE BIOLOGICAL SYSTEM ON HERD PERFORMANCE AND STABILITY

Based on literature and biological observations, applying bioenzymatic systems in monogastric animals is associated with the following effects:

| Toxin                          | Main effect                | On-farm outcome                              |
|--------------------------------|----------------------------|----------------------------------------------|
| <b>Aflatoxin B<sub>1</sub></b> | improved liver function    | ALT/AST -60–70%, FCR +8–10%                  |
| <b>Zearalenone</b>             | reduced estrogenic effect  | better litter size, improved conception      |
| <b>DON</b>                     | decreased gut inflammation | feed intake +5–7%, mortality -30%            |
| <b>Ochratoxin A</b>            | reduced renal burden       | better water intake, improved growth         |
| <b>Fumonisin, T-2</b>          | reduced cytotoxicity       | fewer “stunted” animals, more uniform growth |

## ECONOMIC AND OPERATIONAL ADVANTAGES AT MANAGEMENT LEVEL

Introducing a biological system is not an extra cost but an **efficiency-enhancing investment**:

- **More predictable production**—less fluctuation in growth,
- **Better feed ROI**—more live weight from the same feed,
- **Reduced drug use**—healthier microbiome, fewer infections,



- **Greater stress resilience**—more consistent performance during summer heat, feed changes, or vaccination.

Thus, the system does **not further burden the animal**, it **serves its physiology**, so growth is **natural rather than forced**.

## SUMMARY

Biology-based feeding is **not a new additive** but a **new operating principle**: it does not treat symptoms (inflammation, poor appetite, liver load) but **addresses root causes** (toxic stress and microbial imbalance).

**Esistence** not only stabilizes production—it **creates health-based productivity**.

This is not “just another additive,” but the **body’s natural self-cleansing via feed**; when the organism is not in constant defense, **all energy serves growth**.

## INTERPRETATION FOR VETERINARIANS

Mycotoxins occurring in the feed of monogastric animals (poultry, pigs, rabbits, fish)—primarily aflatoxin B<sub>1</sub> (AFB<sub>1</sub>), zearalenone (ZEN), ochratoxin A (OTA), deoxynivalenol (DON), fumonisin B<sub>1</sub> (FB<sub>1</sub>), T-2/HT-2 toxins and patulin—represent a significant global economic and animal health risk (Ji et al., 2016; Liu et al., 2024).

These compounds, due to their **stable covalent bonds and epoxide/lactone ring structures**, are highly resistant to conventional feed processing, and the monogastric digestive system lacks an **endogenous enzymatic toolkit** capable of degrading them (Sun et al., 2023).

Mycotoxins induce **oxidative stress, ribosomal protein synthesis inhibition, and DNA damage** in enterocytes and hepatocytes.

Clinically, this manifests as **feed refusal, poor FCR, elevated hepatic enzyme levels (ALT, AST), enteritis, and immune or reproductive disorders** (Abraham et al., 2022).

The underlying issue is that monogastric species **lack natural microbial symbionts** capable of chemically inactivating these toxins.

## THEORETICAL BASIS OF MICROBIAL DETOXIFICATION

In nature, several microorganisms—such as *Bacillus subtilis*, *Trichoderma harzianum*, *Lactobacillus plantarum*, *Pseudomonas putida*, and *Rhodococcus erythropolis*—can degrade or modify mycotoxins **through bioenzymatic processes** (Nguyen et al., 2024; Liu et al., 2024).

These microbes express **multi-enzymatic systems** capable of breaking epoxide, aromatic, or lactone rings, thereby eliminating molecular reactivity.

**Oxidoreductases and peroxidases** (e.g., CotA laccase, DyP peroxidase, Mn peroxidase) inactivate aflatoxins and zearalenone through **oxidative ring cleavage** (Gao et al., 2024; Jia et al., 2024).

**Carboxypeptidases and amidases**—mainly in *Bacillus* and *Lactobacillus* species—remove **phenylalanine or ester bonds**, reducing OTA and ZEN toxicity (Abraham et al., 2022).

**Esterases and dehydrogenases**, especially in *Trichoderma* and *Streptomyces*, convert DON and T-2 toxins into less toxic metabolites through **reduction or hydrolysis** (Sun et al., 2023).

These enzymes often function in **synergistic cascades**, where one enzyme metabolizes a toxin and another further detoxifies the resulting intermediate (Liu et al., 2024).

The biochemical outcome is the elimination of **reactive functional groups** on the mycotoxin molecule, preventing its binding to proteins or DNA and blocking oxidative chain reactions.

## MAIN DEGRADATION PATHWAYS AND BIOCHEMICAL CONSEQUENCES

The **primary site of degradation** is the intestinal lumen, where **cell wall-bound enzymes** and **extracellular metabolites** of the microbial community interact with mycotoxins present in the feed.

Detoxification is not mere adsorption but an **active biochemical transformation**, converting toxic molecules into **biologically inert forms** that cannot be absorbed across the intestinal epithelium.

(See attached table for biochemical pathway details.)

## PHYSIOLOGICAL REGENERATION: RESTORATION OF THE GUT–LIVER–IMMUNE AXIS

As a consequence of detoxification, **multi-level regeneration** occurs within the animal organism:

1. **Hepatic protection:** lipid peroxidation decreases; glutathione system activity increases (improved GSH/GSSG ratio); hepatocyte redox homeostasis is restored (Sun et al., 2023).
2. **Intestinal stabilization:** expression of tight-junction proteins (occludin, claudin, ZO-1) increases, the intestinal barrier is restored, and endotoxin translocation decreases (Abraham et al., 2022).
3. **Immunomodulation:** beneficial microbiota (*Lactobacillus*, *Bifidobacterium*, *Faecalibacterium*) competitively exclude pathogenic species (*Clostridium*, *E. coli*), reducing IL-6 and TNF- $\alpha$  while increasing IL-10 levels (Liu et al., 2024).

As a result, the animal's **metabolic stability returns**, reflected in measurable performance parameters:

- improved feed conversion ratio (FCR),
- normalized liver enzyme profiles,
- reduced mortality,
- restored reproductive and immune functions (Nguyen et al., 2024; Sun et al., 2023).

Thus, the bioenzymatic system provides not only **chemical detoxification** but also **functional organ protection**.

## CLINICAL CONSIDERATIONS AND VETERINARY PERSPECTIVE

Conventional strategies for combating mycotoxins have relied on **adsorbents** (e.g., clays, zeolites, activated carbon), which can bind toxins but **do not degrade** them, and often bind nutrients as well.

In contrast, the **bioenzymatic approach** enables **active degradation** that:

- does not interfere with nutrient utilization,
- restores microbial equilibrium,
- and reduces long-term antibiotic demand (Abraham et al., 2022; Liu et al., 2024).

For monogastric species—whose fermentative and microbial diversity is inherently limited—such **enzyme-active microbial systems** provide **true physiological protection** against mycotoxins.

In veterinary practice, this approach can serve as a **preventive strategy**: it stabilizes gut microbiota, safeguards liver function, and enhances stress resilience during vaccination, feed transition, or heat stress.

Clinically, this manifests as **improved feed efficiency, lower FCR values, stronger immune responses, and enhanced reproductive performance**.

Experimental studies show that, in monogastric animals treated with bioenzymatic systems,

AFB<sub>1</sub>-related liver enzyme deviations decreased by **60–70%**,

ZEN-related estrogenic activity by **~80%**,

and DON-associated inflammatory markers by **~50%** (Nguyen et al., 2024; Sun et al., 2023).

## SUMMARY

In monogastric animal health, **microbial–enzymatic defense against mycotoxins** represents a **next-generation veterinary concept**.

The objective is not merely toxin removal but **biochemical deactivation** and **activation of the organism's regenerative capacity**.

This process is driven by the **Bacillus–Trichoderma–Lactobacillus–Pseudomonas axis**, capable of simultaneously protecting and stabilizing the gut–liver–immune system.

Hence, **bioenzymatic detoxification** functions not only as a chemical but also as a **physiological defense system**, contributing to the realization of **antibiotic-free and sustainable animal production**.

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| MONOGASTRIC ANIMALS                                       |                                                                  |                                                                                   |                                                                     |                                                                                                                           |                                                                                                                               |                           |                          |                                                                            |
|-----------------------------------------------------------|------------------------------------------------------------------|-----------------------------------------------------------------------------------|---------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------|---------------------------|--------------------------|----------------------------------------------------------------------------|
| Esstence Bio-Enzymatic Mycotoxin Detoxification Potential |                                                                  |                                                                                   |                                                                     |                                                                                                                           |                                                                                                                               |                           |                          |                                                                            |
| Mycotoxin                                                 | Effect of the toxin on the animal's body                         | Microorganisms proven effective in publications                                   | Taxa identified in Esstence                                         | Biochemical detoxification pathway                                                                                        | Result of the enzymatic/mechanistic process in the animal + combined Esstence effect                                          | Literature efficiency (%) | Expected Esstence effect | References (author + year)                                                 |
| Aflatoxin B <sub>1</sub> (AFB <sub>1</sub> )              | Hepatotoxicity, immunosuppression, reduced appetite, poor FCR    | Bacillus subtilis / licheniformis, Trametes / Cerrena, Pseudomonas, Mycobacterium | B. subtilis, P. putida, Rhodococcus erythropolis, Aspergillus niger | 8,9-epoxide → dihydrodiol; lactone hydrolysis (AFD <sub>1</sub> /AFD <sub>2</sub> ); C3-hydroxylation (AFQ <sub>1</sub> ) | Epoxide ring opening → decreased hepatotoxicity; Combined effect: ALT/AST normalization, improved immune function, FCR +5–10% | 70–98                     | 80–90%                   | Sun 2023; Liu 2024; Nguyen 2024; Gao 2024; Jia 2024; Ji 2016; Abraham 2022 |
| Zearalenone (ZEN)                                         | Estrogenic effect, cycle and fertility disorders                 | Trichoderma, Aspergillus niger, Bacillus                                          | T. harzianum, A. niger, B. amyloliquefaciens                        | Aromatic oxidation + ester hydrolysis → non-estrogenic metabolites                                                        | Reduced estrogenic activity; Combined effect: hormonal balance restored, fertility improved                                   | 60–100                    | 75–85%                   | Sun 2023; Liu 2024; Gao 2024; Jia 2024; Ji 2016                            |
| Ochratoxin A (OTA)                                        | Nephrotoxicity, immunodepression, decreased feed intake          | B. licheniformis, L. plantarum, A. niger                                          | B. licheniformis, L. plantarum, A. niger                            | Phenylalanine cleavage (OTA → OTα)                                                                                        | OTα formation → reduced nephrotoxicity; Combined effect: improved renal function, feed intake +6–8%                           | 50–85                     | 70–80%                   | Sun 2023; Liu 2024; Abraham 2022; Ji 2016                                  |
| Deoxynivalenol (DON)                                      | Intestinal epithelial damage, loss of appetite, ribotoxic stress | Streptomyces, Bacillus, Lactobacillus                                             | S. griseus, B. subtilis, L. casei                                   | Epoxide reduction → DON-ol / DON-3-glucoside                                                                              | DON-ol → reduced ribotoxic stress; Combined effect: mitigated enteritis, restored microbiome, improved appetite               | 60–80                     | 75–80%                   | Sun 2023; Liu 2024; Selim 2024; Ji 2016                                    |
| Fumonisin B <sub>1</sub> (FB <sub>1</sub> )               | Hepatic / intestinal damage, sphingolipid metabolism disruption  | Sphingomonas / Sphingopyxis, Streptomyces, Bacillus, A. niger                     | Streptomyces spp., B. cereus, A. niger                              | Side-chain hydrolysis → HFB <sub>1</sub> ; C2-deamination                                                                 | HFB <sub>1</sub> less toxic; Combined effect: liver membrane protection, intestinal wall regeneration                         | 55–90                     | 80–90%                   | Abraham 2022; Sun 2023; Ji 2016                                            |
| T-2 / HT-2                                                | Intestinal mucosal damage, inflammation, feed refusal            | Rhodococcus, Trichoderma, Pseudomonas                                             | R. erythropolis, T. harzianum, P. putida                            | Epoxide opening + deacetylation → less toxic metabolites                                                                  | Epoxide opening → reduced cytotoxicity; Combined effect: epithelial regeneration, improved nutrient utilization               | 60–85                     | 75–85%                   | Sun 2023; Selim 2024; Ji 2016                                              |
| Patulin                                                   | Intestinal epithelial damage, oxidative stress, loss of appetite | Candida, Aspergillus niger                                                        | Candida spp., A. niger                                              | Lactone ring hydrolysis → less toxic derivatives                                                                          | Lactone cleavage → mucosal protection; Combined effect: intestinal mucosa recovery, appetite normalization                    | 70–95                     | 85–90%                   | Sun 2023; Abraham 2022; Ji 2016                                            |