Mycotoxins and Gut Integrity: Strengthening the Intestinal Barrier to Secure Performance

By Elise Nacer-Khodja, Toxin Solution Product Manager EW Nutrition

The gut under siege: understanding the direct assault on epithelial integrity

The gastrointestinal tract (GIT) is the primary site of interaction between animals and ingested mycotoxins, playing a pivotal role in the absorption and oral bioavailability of these contaminants. While high-dose clinical mycotoxicosis is rare in modern production, the chronic ingestion of low to moderate levels triggers a cascade of metabolic, physiological, and immunological disorders. The intestinal epithelium, a single layer of cells, is the animal's most critical interface, functioning simultaneously as a nutrient harvester and a frontline barrier against pathogens and toxins.

Mycotoxins, specifically trichothecenes like deoxynivalenol (DON), and fumonisins (FB1), but also aflatoxins (AFLA) and ochratoxins (OTA) directly sabotage this barrier. They downregulate the mRNA expression of tight junction proteins, compromise cell viability, and degrade the protective mucus layer. Beyond this structural damage, mycotoxins induce a pro-inflammatory cytokine response and disrupt the gut microbiota. These alterations do more than just damage the gut; they increase susceptibility to secondary infections such as coccidiosis, necrotic enteritis, salmonellosis and many others.

Protecting the gastrointestinal tract from mycotoxins becomes an essential pillar for health and performance because GIT is not just an organ for digestion; it is the largest immune organ in the body. When its integrity is compromised, the animal's entire biological priority shifts from growth to defense, leading to hidden performance losses that are often only noticed at the end of the production cycle.

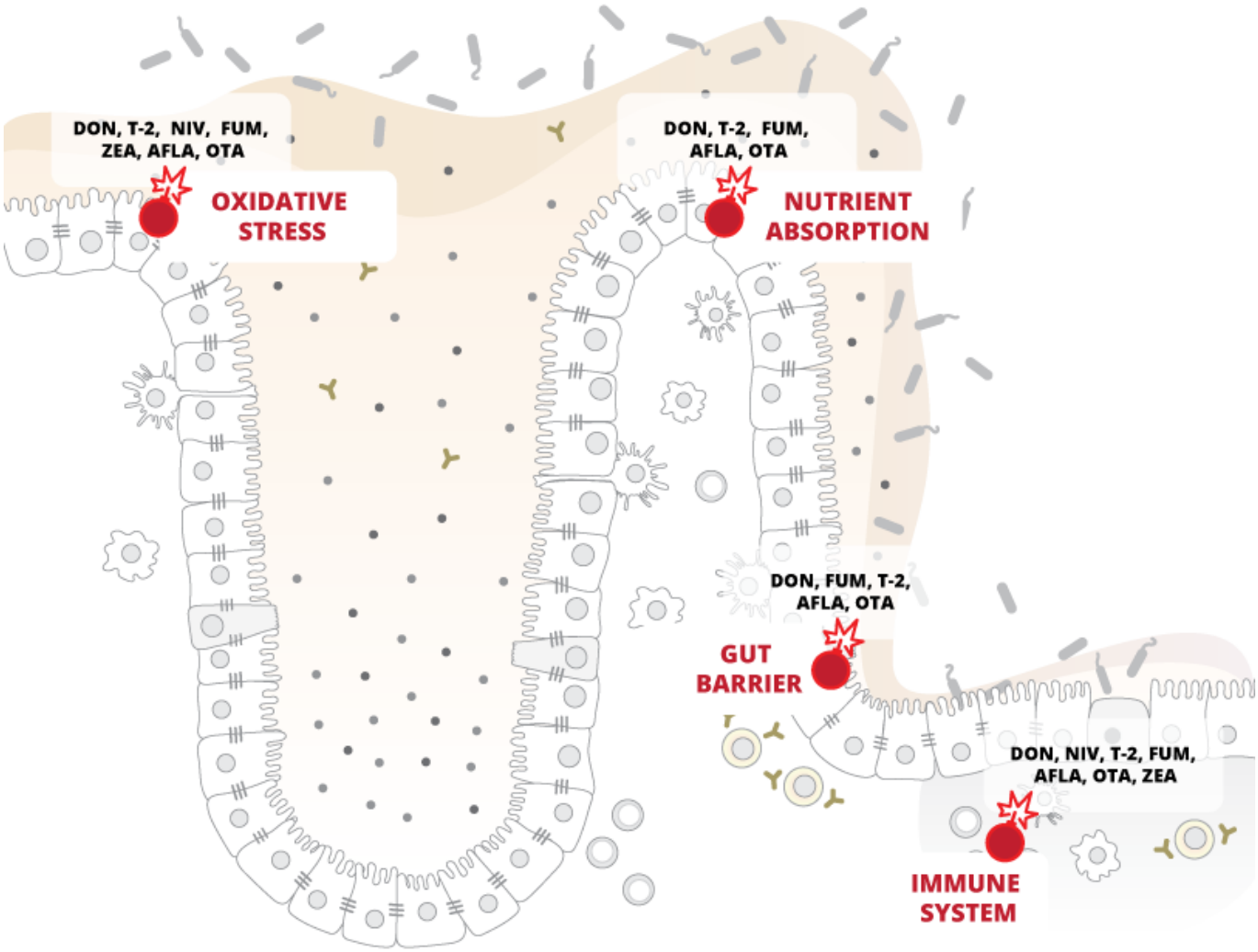

Figure 01 – Impact of mycotoxins on different functions of the GIT

Beyond physical damage: the catalyst for antibiotic resistance

Recent research highlights a critical link between mycotoxins and the global rise of antimicrobial resistance (AMR). While the misuse of drugs is the primary driver of AMR, toxins such as deoxynivalenol (DON) act as potent environmental catalysts. DON significantly disrupts the microbial balance of the gut, providing a survival advantage to bacteria carrying resistance genes. Furthermore, mycotoxins have been shown to activate specific bacterial resistance genes and accelerate horizontal gene transfer, allowing resistant strains to spread more rapidly through the microbiota. Bacteria employ molecular defense mechanisms against mycotoxins (such as efflux pumps and detoxification enzymes) that are similar to those used against antibiotics. This cross-resistance not only weakens therapeutic effectiveness but also creates a systemic “One Health” challenge.

From gut porosity to hepatic stress

In a study led by EW Nutrition in a research center in 2025, the oral exposure to 2 ppm of DON and 5 ppm of Fumonisin B1 from day 11 to 42 of 480 broiler chickens (Ross 308) acted as a direct assault on their intestinal and hepatic functions. Specifically:

- Intestinal porosity was increased: a significant downregulation of the tight junction protein ZO-1 ($p<0.001$) expression was observed, compromising gut integrity.

- Systemic leakage was revealed: an increased level of serum *E. coli* lipopolysaccharide (LPS), indicated that pathogens bypassed the degraded epithelial barrier.
- Hepatic damage was observed: severe hepatocellular necrosis, fibrosis, and a massive upregulation of IL-6 (inflammatory interleukin) and NOX-4 (marker of oxidative stress) was measured in the liver.

The liver is the primary metabolic hub for birds. By forcing the liver to deal with an influx of intestinal pathogens and oxidative damage, mycotoxins divert energy away from muscle protein synthesis. This redirection of resources is a primary driver of poor feed conversion rates, even when the animals do not show obvious signs of illness.

Economic consequences: the true cost of a compromised barrier

The biological sabotage detailed in the EW Nutrition trial translates directly into technical failure and heavy economic losses. The exposure to DON and FB1 significantly hindered performance during the growing-finishing period:

- Feed efficiency: The Feed Conversion Ratio (FCR) increased by 5 points (3%, $p<0.01$) from 11 to 42 days,
- Growth inhibition: At 42 days, challenged birds weighed 67g less (2.5%) than the control group,
- Productivity drop: The European Production Efficiency Factor (EPEF) decreased by 7% ($p<0.01$),
- Mortality: mortality rates more than doubled, jumping from 2.50% in the control group to 6.67% in the challenged group.

For the producer, this resulted in an average loss of 0.18€ per head. In a large-scale commercial operation, these “sub-clinical” losses can represent tens of thousands of euros in lost revenue per house, largely driven by the indirect effects of gut leakage and liver stress.

Research led by Kolawole (2025) suggests that poultry producers lose $0.30 per broiler chicken due to subclinical mycotoxin exposure. By damaging gut health and weakening immune responses, these toxins reduce feed efficiency and trigger “hidden” financial leaks. Even when contamination appears low, the cumulative impact on profitability remains severe.

Securing gut barrier: a shield for profitability

To counteract these effects, the trial evaluated the capacity of EW Nutrition toxin risk solution to mitigate these mycotoxin-induced damage. The results showed that the inclusion of this solution acted as a definitive shield for the animals:

- Restored gut integrity: EW Nutrition solution significantly improved the gut condition, reducing inflammation and restoring the intestinal barrier,
- Reduced lesions: mucosal ulceration and lesion scores were greatly reduced compared to the challenged group,
- Liver protection: supplementation returned the hepatic markers IL-6 and NOX-4 to control levels, effectively neutralizing the metabolic burden and oxidative stress on the liver.

Most importantly, this biological protection translated into a full recovery of animal performance. Birds receiving the supplementation reached higher body weights (2,782g vs 2,686g in the challenge group) and mortality was halved. Overall, groups treated with EW Nutrition toxin risk solution showed the highest productivity, with an EPEF 5% to 11% higher than their respective controls.

Conclusion

Effective mycotoxin management requires a multi-layered approach. While general biosecurity measures and raw material monitoring are essential to reduce initial exposure, they are rarely enough to eliminate the risk entirely in commercial environments. This study demonstrates that even moderate levels of toxins can trigger systemic metabolic stress and gut failure. Therefore, in addition to standard preventive measures, the use of EW Nutrition's advanced solutions, such as Solis Max 2.0, represents a highly effective lever.

With a Return on Investment (ROI) of 5:1, EW Nutrition's approach proves that protecting the intestinal epithelium and the liver is a fundamental technical and economic requirement. By ensuring nutrients are used for growth rather than inflammation, producers can secure the profitability and health of the broiler cycle, even under significant mycotoxin challenges.

References available upon request.

Ionophores: An Overlooked Risk for the Spread of Medically Relevant Antibiotic Resistance

Author: Dr. Inge Heinzl, Editor EW Nutrition

Antibiotic resistance is one of the biggest threats to global health today. When bacteria become resistant to antibiotics, infections that were once easily treatable can become deadly. For decades, the discussion surrounding the causes of antimicrobial resistance (AMR) has primarily focused on the misuse of antibiotics in human medicine and agriculture. But some antibiotics have escaped critical scrutiny—until now.

Ionophores, a special group of antibiotics

Ionophores are a group of antibiotics used as feed additives in ruminants and pigs as growth promoters and in poultry as anticoccidials since the early 1970s (Chapman et al., 2010). They are among the most widely used classes of antibiotics in animal production. In the US, e.g., more than 4 million kilograms were sold in 2016 (Wong, 2019).

Unlike many other antibiotics, ionophores are not used in human medicine because of their toxicity. For this reason, regulators have often assumed that ionophores pose little to no threat to human health. In North America, for example, ionophores are officially classified as having low or no importance for human medicine, which means their use is less strictly regulated than antibiotics that are directly relevant for human health.

However, new scientific findings challenge this assumption. A research team led by Asalia Ibrahim (2025) has provided compelling evidence that the use of ionophores in agriculture may indirectly contribute to the spread of resistance to antibiotics crucial for treating human infections.

What did the researchers discover?

The researchers focused on two specific genes, narA and narB, transporters which enable Enterococcus faecium to resist ionophores like narasin, salinomycin, and maduramicin. Initially, these genes were found in bacteria isolated from Swedish broiler chickens AND on the same plasmid as vancomycin resistance genes (Nilsson et al., 2012). More recent studies have identified the NarA and NarB genes in other countries as well, raising questions about their global distribution and their connection to resistance to medically important antibiotics.

To investigate, Asalia Ibrahim (2025) analyzed publicly available genome data from the NCBI Pathogens database, a massive resource that collects bacterial genome sequences from around the world. They identified more than 2,400 bacterial isolates from 51 countries that carry both narA and narB. The bacteria were found in various host animals, including poultry, swine, and cattle, but also in humans. Alarmingly, over 500 of the samples containing these resistance genes came from human sources!

Why is this a problem?

The core concern is that these ionophore resistance genes do not exist in isolation. Instead, they are often genetically linked with other resistance genes that protect bacteria from antibiotics that are critical for human medicine.

This can happen in two ways:

- Cross-resistance, where a single gene provides resistance to multiple drugs at once. In this case, it appears unlikely because ionophores belong to a class (polyether antibiotics) that is not used for humans.
- Co-selection occurs when different resistance genes sit close together on the same piece of genetic material (like a plasmid) or in the same bacterial genome. If one gene is selected because the antibiotic it resists is used, then the other genes hitch a ride and spread too.

The researchers found clear evidence for co-selection. Many narAB-carrying bacteria also contained resistance genes for vancomycin, a last-resort antibiotic (Nilsson et al., 2012), erythromycin, tetracycline

(Pikkemaat et al., 2022), and other antibiotics. On average, each narAB isolate carried more than 10 additional resistance determinants, including both resistance genes and mutations.

The link is not just theoretical. When the Norwegian poultry industry stopped using narasin in 2016, the levels of vancomycin-resistant Enterococcus dropped significantly (Simm et al., 2019). This real-world example suggests that the use of ionophores can indeed help maintain resistance to medically relevant antibiotics in animal populations, potentially allowing these bacteria to enter the food chain and reach humans.

What does this mean for food safety and public health?

The study's findings highlight how actions taken in agriculture can have far-reaching effects on human health. Suppose bacteria carrying narAB genes also carry resistance to life-saving human antibiotics. In that case, the routine use of ionophores in animal feed can indirectly contribute to maintaining a reservoir of resistant genes. These bacteria can spread from animals to humans through direct contact, contaminated meat, or environmental exposure.

This raises questions about the long-held belief that ionophores are risk-free. In reality, they might be acting as a hidden driver for the maintenance and spread of resistance genes that severely limit our treatment options in human medicine.

What should be done?

The researchers argue that ionophores need to be reevaluated within the broader framework of the "One Health" approach, which recognizes that the health of people, animals, and ecosystems are deeply interconnected. Simply because ionophores are not used in hospitals does not mean they are harmless to human health.

Possible steps could include:

- Stricter monitoring of ionophore use in livestock.
- Better surveillance of resistance genes like narA and narB in both animal and human bacterial isolates.
- Considering limits or alternatives to routine ionophore use in industrial farming.
- More research to understand how these resistance genes move between bacteria, species, and environments.

The bottom line

Ionophores play a crucial role in intensive animal production worldwide, helping to maintain the health and productivity of animals. But this convenience comes at a potential cost. The research of Ibrahim et al. (2025) serves as a clear reminder that the use of antibiotics—whether for humans or animals—can have unintended consequences for global health.

Prudent, science-based management of all antibiotics is crucial to slowing the spread of antimicrobial resistance and preserving the effectiveness of life-saving drugs for future generations.

References

Chapman, H.D., T.K. Jeffers, and R.B. Williams. "Forty Years of Monensin for the Control of Coccidiosis in Poultry." Poultry Science 89, no. 9 (September 2010): 1788–1801. https://doi.org/10.3382/ps.2010-00931.

Ibrahim, Asalia, Jason Au, and Alex Wong. “The Ionophore Resistance Genes narA and narB Are Geographically Widespread and Linked to Resistance to Medically Important Antibiotics.” mSphere, June 17, 2025. https://doi.org/10.1128/msphere.00243-25.

Nilsson, O., C. Greko, B. Bengtsson, and S. Englund. “Genetic Diversity among VRE Isolates from Swedish Broilers with the Coincidental Finding of Transferrable Decreased Susceptibility to Narasin.” Journal of Applied Microbiology 112, no. 4 (March 5, 2012): 716–22. https://doi.org/10.1111/j.1365-2672.2012.05254.x.

Pikkemaat, M.G., M. Rapallini, J.H.M. Stassen, M. Alewijn, and B.A. Wullings. “Ionophore Resistance and Potential Risk of Ionophore Driven Co-Selection of Clinically Relevant Antimicrobial Resistance in Poultry.” Food Safety Report, Wageningen, 2022. https://doi.org/10.18174/565488.

Simm, Roger, Jannice Schau Slettemeås, Madelaine Norström, Katharine R. Dean, Magne Kaldhusdal, and Anne Margrete Urdahl. “Significant Reduction of Vancomycin Resistant E. Faecium in the Norwegian Broiler Population Coincided with Measures Taken by the Broiler Industry to Reduce Antimicrobial Resistant Bacteria.” PLOS ONE 14, no. 12 (December 12, 2019). https://doi.org/10.1371/journal.pone.0226101.

Wong, Alex. “Unknown Risk on the Farm: Does Agricultural Use of Ionophores Contribute to the Burden of Antimicrobial Resistance?” mSphere 4, no. 5 (October 30, 2019). https://doi.org/10.1128/msphere.00433-19.

Mycotoxins as contributors to antibiotic resistance?

By Dr. Inge Heinzl, Editor EW Nutrition and
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Antibiotic resistance is a growing global health concern, making infections more complicated to treat and increasing the risk of disease spread, severe illness, and death. While overuse and misuse of antibiotics are the primary causes, recent research has uncovered another unexpected contributor: mycotoxins. Among these, deoxynivalenol (DON), a toxin commonly found in contaminated grains, has been shown to significantly alter gut microbiota and promote antibiotic resistance. This article examines how DON impacts gut bacteria, influences antibiotic resistance, and highlights why this issue warrants urgent attention.

Mycotoxins – originators of antimicrobial resistance?

Actually, it would be logical…

Alexander Fleming discovered Penicillin when he returned after the summer holidays and saw that a mold had grown on the agar plate he had prepared. Around the mold, Staphylococcus was unable to proliferate. The reason was a substance produced by the mold – penicillin, which, like other toxins produced by molds, is a mycotoxin. In his article about the origin of antibiotics and mycotoxins, Shier (2011) stated that antibiotics and mycotoxins share considerable similarities in structure, metabolic roles, and biosynthesis.

A short excursus to antimicrobial resistance

In general, the primary mechanisms of resistance involve the prevention or limitation of the antimicrobial substance's uptake, modifying the drug target, inactivating the drug, or facilitating its discharge with efflux pumps.

There are two types of resistance: natural resistance, which is further divided into intrinsic and induced resistance, and acquired resistance.

Intrinsic resistance is a "characteristic" of a bacterial species and is not dependent on antibiotic exposure. An example is the reduced permeability of the outer membrane of gram-negative bacteria, which prevents certain antibiotics from entering.

Induced resistance, however, needs to be initiated by antibiotics. Here, multidrug-efflux pumps can be mentioned.

The third one, acquired resistance, refers to the process by which bacteria acquire genetic material, the resistance genes, from other bacteria that are resistant. The mechanisms include vertical transfer to daughter cells and horizontal transfer, such as the transfer from dead bacteria to living ones, by viruses, or the transfer of plasmids (Reygaert, 2018).

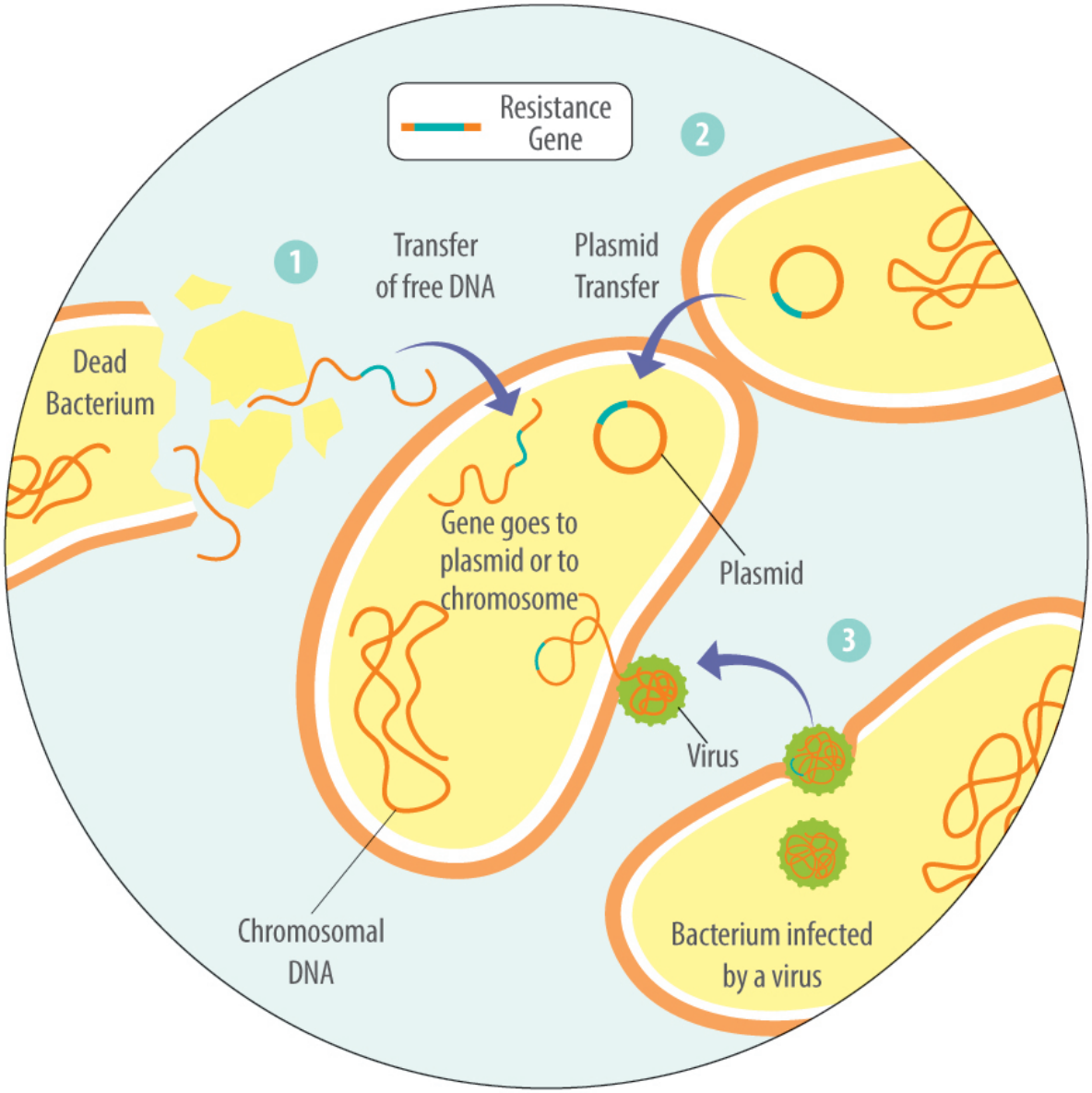


Figure 1: Different possibilities of transfer of resistance genes

Deoxynivalenol (DON) promotes resistance in gut microbiota

A Chinese group of researchers (Deng et al., 2025) examined for the first time the influence of DON on the intestinal microbiota of chickens. One of the most alarming findings is DON's ability to enhance antibiotic resistance. It contributes to this issue in several ways:

1. Encouraging resistant bacteria – By disrupting microbial balance, DON provides a survival advantage to bacteria that carry resistance genes.
2. Activating resistance genes – Studies suggest that DON can increase the expression of genes that help bacteria withstand antibiotics.
3. Enhancing gene transfer – Bacteria can share resistance genes through horizontal gene transfer. DON appears to promote this process, making antibiotic-resistant strains spread more rapidly.
4. Weakening antibiotic effectiveness – DON-induced changes in the gut environment can reduce the effectiveness of antibiotics, making treatments less successful.

A further indication that mycotoxins can enhance resistance is the significant overlap in the geographical distribution of antimicrobial-resistant bacteria and genes with that of mycotoxins, as noted by Deng et al.

Which protection mechanisms do bacteria have against mycotoxins?

In the case of mycotoxins, bacteria employ similar molecular mechanisms to those used against antibiotics. In an in vitro experiment, Hassan et al. (2019) challenged *Devosia mutans*, a gram-negative bacterium, with DON in the growth medium. DON inhibits protein synthesis, induces oxidative stress, and compromises cell membrane integrity in eucaryotic cells. Hassan et al. asserted three adaptive mechanisms as the response to the challenge:

1. Activation of cellular membrane proteins (adenosine 5'-triphosphate-binding cassette -ABC-transporters) responsible for the unidirectional transport of substrates, either outward or inward. These ABC transporters can work as drug efflux pumps.
2. Production of DON-specific deactivation enzymes, thereby engaging a toxin-specific pyrroloquinoline quinone-dependent detoxification pathway. This enables the bacterial isolate to transform DON to a non-toxic stereoisomer.
3. Upregulation of auxiliary coping proteins, such as porins (transmembrane proteins involved in metabolite exchange), glutathione S-transferases, and phosphotransferases, both of which are likely involved in the detoxification of xenobiotics.

Public health implications and preventive measures

Given the widespread presence of DON in food and animal feed, its potential role in antibiotic resistance poses a serious threat. The combination of increased bacterial resistance and weakened antibiotic efficacy could lead to more difficult-to-treat infections. This is particularly concerning in hospital settings, where antibiotic-resistant infections already cause high mortality rates.

To address the issue, several strategies can be implemented:

1. Reducing DON contamination: Implementing improved agricultural practices, such as crop rotation, the use of fungal-resistant crop varieties, and maintaining proper storage conditions, can help limit fungal growth and DON production.
2. Monitoring food and feed supply – Strict regulations and testing for DON contamination in grains and animal feed are essential to minimize human and animal exposure.
3. Effective mycotoxin risk management at feed mill and farm levels: Using tools such as MasterRisk and effective products combatting mycotoxins.
4. Maintaining gut health: A healthy diet rich in fiber, probiotics, and gut health-supporting feed supplements, such as Ventar D or products from the Activo line, may help counteract some of the adverse effects of DON on gut microbiota.
5. Developing new treatments: Research into alternative therapies and new antibiotics is crucial to combat the rise of antibiotic resistance.

Antimicrobial resistance: Be aware of the mycotoxins!

The connection between mycotoxins, such as DON, and antibiotic resistance underscores the need for a broader perspective on public health and food safety and once again brings the "One Health Concept" into focus. While antibiotic overuse remains the primary driver of resistance, environmental factors, such as

exposure to mycotoxins, should not be overlooked. By increasing awareness, enhancing food safety regulations, and investing in research, we can take steps to mitigate this emerging threat and safeguard the effectiveness of antibiotics for future generations.

References:

Deng, Fengru, Chuying Yao, Linyu Ke, Meichan Chen, Mi Huang, Jikai Wen, Qingmei Chen, Jun Jiang, and Yiqun Deng. “Emerging Threat to Antibiotic Resistance: Impact of Mycotoxin Deoxynivalenol on Gut Microbiota and Clonal Expansion of Extensively Drug-Resistant Enterococci.” Environment International 197 (March 2025): 109353. https://doi.org/10.1016/j.envint.2025.109353.

Hassan, Yousef I., Jian Wei He, Dion Lepp, and Ting Zhou. “Understanding the Bacterial Response to Mycotoxins: The Transcriptomic Analysis of Deoxynivalenol-Induced Changes in Devosia Mutans 17-2-E-8.” Frontiers in Pharmacology 10 (November 14, 2019). https://doi.org/10.3389/fphar.2019.01098.

Reygaert, Wanda C. “An Overview of the Antimicrobial Resistance Mechanisms of Bacteria.” AIMS Microbiology 4, no. 3 (2018): 482–501. https://doi.org/10.3934/microbiol.2018.3.482.

Shier, W. Thomas. “On the Origin of Antibiotics and Mycotoxins.” Toxin Reviews 30, no. 1 (January 28, 2011): 6–30. https://doi.org/10.3109/15569543.2011.550862.

Smith, William P., Benjamin R. Wucher, Carey D. Nadell, and Kevin R. Foster. “Bacterial Defences: Mechanisms, Evolution and Antimicrobial Resistance.” Nature Reviews Microbiology 21, no. 8 (April 24, 2023): 519–34. https://doi.org/10.1038/s41579-023-00877-3.

Enhancing Poultry Gut Health with Novel Xylanase: A Sustainable Path to Reduced Antimicrobial Use

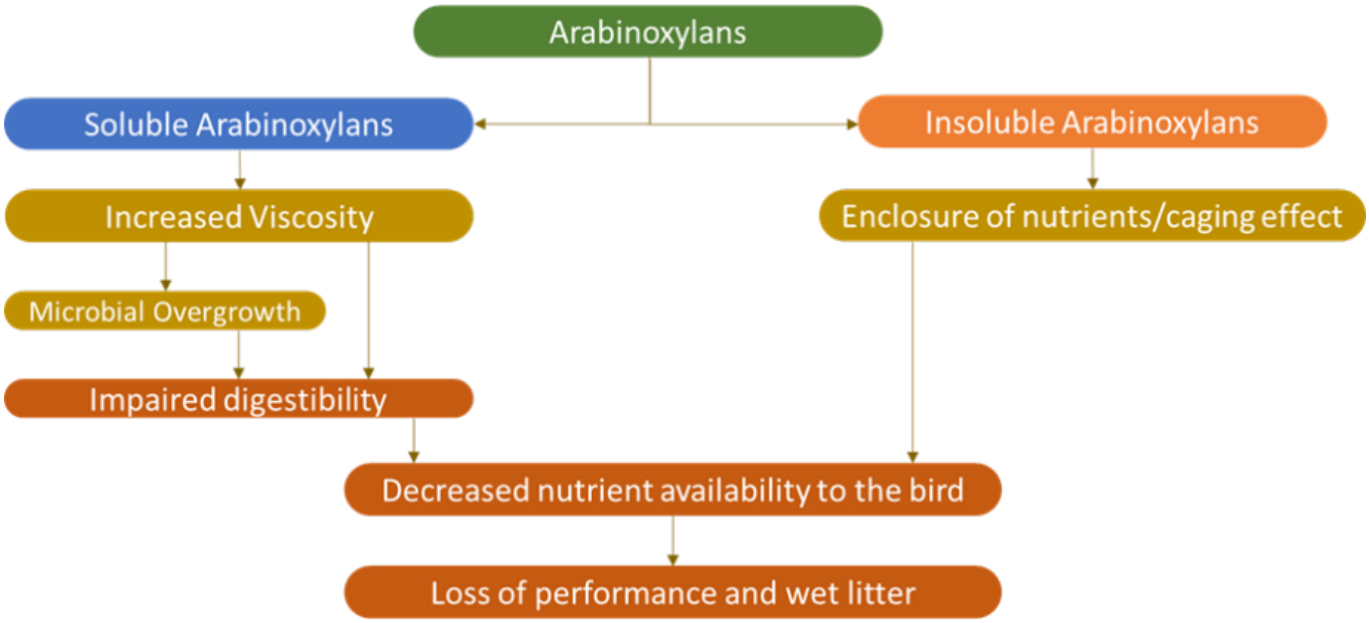
By **Ajay Bhoyar**, Senior Global Technical Manager, EW Nutrition

Gut health is pivotal to profitable poultry production, as the gastrointestinal tract (GIT) enables nutrient digestion and absorption while acting as a defense against pathogens. A healthy gut improves feed conversion, boosts immune resilience, and reduces reliance on antimicrobials—critical in the fight against antimicrobial resistance (AMR). With AMR posing significant threats to public health and animal agriculture, strategies like biosecurity, sustainable management, and effective dietary interventions are gaining traction. Feed enzymes have emerged as essential tools for managing feed costs, mitigating anti-nutritional factors, and improving nutrient utilization. Among these, feed enzymes like xylanase stand out. By breaking down xylan, a major component of non-starch polysaccharides (NSPs) in plant-based feed ingredients, xylanase reduces gut viscosity, enhances nutrient utilization, and supports optimal gut health and productivity. This article explores the innovative application of novel GH10 xylanases, such as Axxess XY, as a sustainable solution for improving feed efficiency and gut health in poultry production.

Xylanase in Poultry Nutrition

Xylanase plays a pivotal role in enhancing nutrient availability by addressing the limitations of endogenous enzyme synthesis in poultry. Xylanase enzymes belong to the carbohydrase class, catalyzing the breakdown of xylan, a major NSP in plant-based feed ingredients. They hydrolyze xylan into simple sugars like arabino-xylo-oligosaccharides (AXOs) and xylo-oligosaccharides (XOs), reducing the encapsulation of nutrients and digesta viscosity. These actions improve overall nutrient digestibility and bird performance.

Fig.1: Arabinoxylans – anti-nutrient mode of action in chicken



The primary benefit of feed xylanase lies in its ability to reduce digesta viscosity. By partially hydrolyzing NSPs in the upper digestive tract, xylanase ensures better nutrient absorption in the small intestine. Studies (Matthiesen et al., 2021; Choct & Annison, 1992) confirm that reduced viscosity enhances feed digestibility, leading to improved performance in poultry. Further, to realize the optimum benefits, it is crucial that xylanase efficiently degrades both soluble and insoluble arabinoxylans. The insoluble arabinoxylans are part of the cell wall structure of plant cells, resulting in a cage effect, entrapping nutrients like starch and protein. Effectively breaking down insoluble arabinoxylans ensures that the nutrients trapped in plant cell walls are released for growth and production.

Mechanisms Supporting Gut Health

Viscosity Reduction

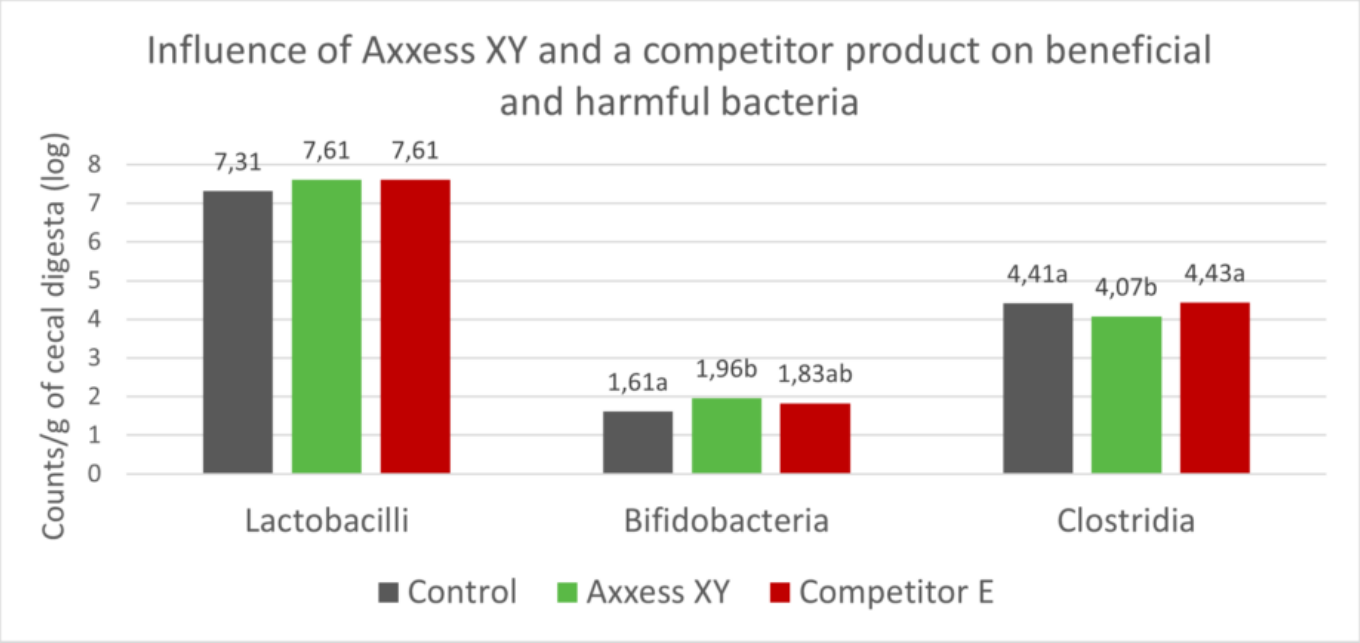
High NSP content increases digesta viscosity and slows digestion and nutrient absorption. Soluble arabinoxylan is not digested in the small intestine of broilers. It produces a viscous chime, leading to the proliferation of pathogenic bacteria, intestinal inflammation, impairment of barrier function in the intestine, and severe intestinal lesions (Teirlynck et al., 2009). Xylanase mitigates this by breaking down xylans, a major component of NSPs in common feed ingredients. This results in a better flow of digesta and reduced energy losses.

Microbial Metabolites

Xylo-oligosaccharides (XOS) can also be produced in the intestine of monogastric animals to some extent when exogenous enzymes, such as xylanase, are added to the feed (Baker et al., 2021).

The XOS generated by xylanase action on arabinoxylans can act as prebiotics, fostering beneficial bacteria like Lactobacillus and Bifidobacterium, which can outcompete harmful species. XOS can positively impact the gut microbiota, enhance short-chain fatty acid (SCFA) production, stimulate immune activity in the gastrointestinal tract, and improve energy utilization.

Fig. 2. Axxess XY improved beneficial microbes and reduced the clostridial population in broilers.



Barrier Function

By lowering inflammation and irritation in the intestine, xylanase helps maintain gut integrity, reducing the risk of pathogen translocation from the intestinal lumen. In a broiler study, xylanase decreases epithelial apoptosis index, up-regulates tight junction gene expression, and inhibits mucin synthesis in the small intestine, likewise alleviating the intestinal mucosal barrier impairment from *Clostridium perfringens* challenge (Liu et al., 2012).

Practical Considerations for Xylanase Use

Enzyme Stability

Enzymes are proteins that tend to lose their catalytic activity at high temperatures. When exposed to excessive heat, an enzyme's protein structure can irreversibly unfold, disrupting its active site and causing loss of function. Therefore, ensuring enzyme stability during feed processing is critical for maintaining its activity in the intestine. Intrinsically heat-stable enzymes have an inherent ability to withstand higher temperatures without the need for a protective coating and are immediately available for action upon ingestion.

Feed Composition

Xylanase efficacy is influenced by diet composition, particularly the NSP content and the presence of xylanase inhibitors in common feedstuffs. It is important to choose a xylanase that can resist the activity of xylanase inhibitors and is effective against both soluble and insoluble arabinoxylans.

The recommended energy matrix value for the xylanase enzyme should be used while formulating the feeds to create energy-deficient diets to reap the full benefits of xylanase use.

Optimal Dosage

Proper dosing is essential to maximizing the benefits of feed enzymes while avoiding unnecessary costs. It is important to follow manufacturers' recommendations and avoid underdosing an enzyme.

GH10 Xylanases: The Superior Choice for Animal Nutrition

Most feed xylanases are classified into glycoside hydrolase families 10 (GH10) and 11 (GH11) based on their substrate specificity, catalytic action, and structural features.

Why GH10 Xylanases Are More Effective

1. **Broader Substrate Specificity:**

   Unlike GH11 xylanases, GH10 xylanases can effectively hydrolyze both soluble and insoluble xylan substrates. This broader activity ensures an efficient breakdown of xylans in a wide range of feed ingredients.

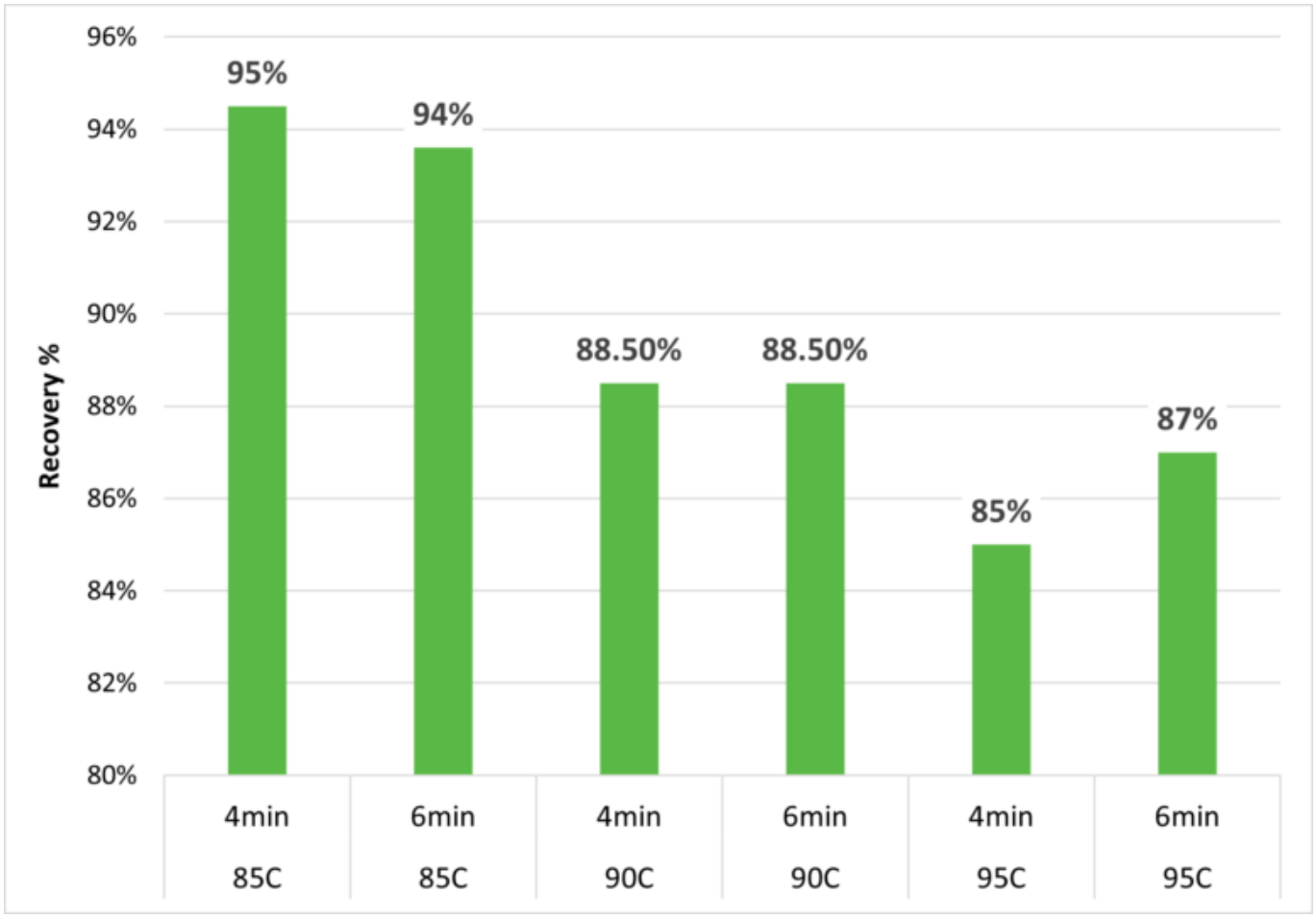
2. **Higher Catalytic Efficiency:**

   GH10 enzymes cleave xylan at substituted regions, yielding shorter xylo-oligosaccharides that can positively impact gut health and maximize nutrient availability.

3. **Thermostability:**

   Feed processing often involves high temperatures during pelleting. Axxess XY, a GH10 family xylanase, demonstrates remarkable thermostability, maintaining over 85% activity even at 95°C for extended conditioning times. This resilience ensures consistent enzyme performance during feed manufacturing and digestion.

Fig.3: Optimum recovery of Axxess XY at elevated conditioning time and temperatures



Novel Applications of Axxess XY: A GH10 Xylanase

Axxess XY exemplifies the advantages of GH10 xylanases in poultry nutrition. Its ability to efficiently act on both soluble and insoluble arabinoxylans makes it a versatile feed enzyme. The enzyme's high thermostability ensures efficient enzyme activity in the gut and subsequent optimum nutrient utilization under challenging processing conditions, promoting gut health and maximizing performance.

Key Benefits of Axxess XY

1. **Enhanced Nutrient Utilization:**

   By unlocking nutrients trapped in NSPs, Axxess XY promotes better feed conversion ratios (FCRs).

2. **Improved Gut Health:**

   Reducing the digest's viscosity reduces gut health challenges and predisposition to gut infections. Further, the short-chain oligosaccharides released by Axxess XY support beneficial gut microbiota, improving digestive health.

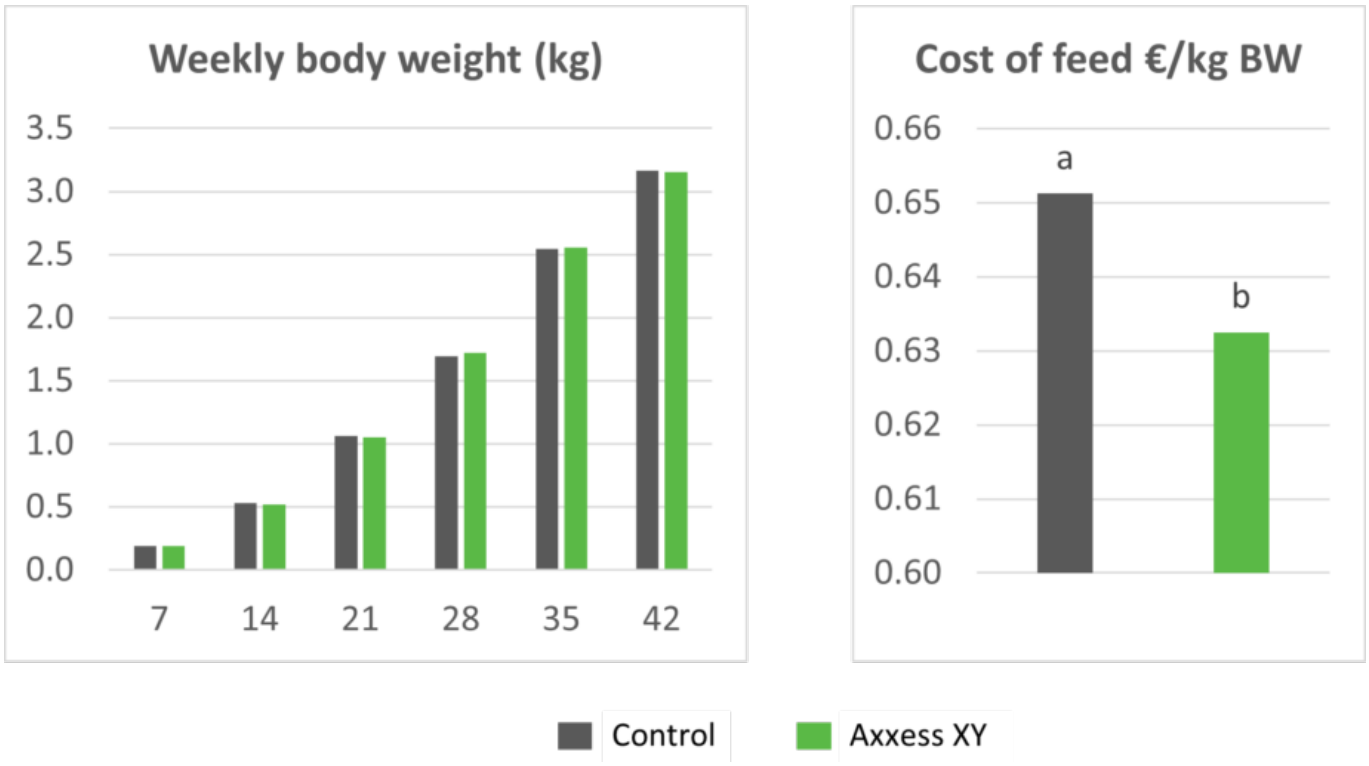
3. **Economic Efficiency:**

   Enabling the optimum use of high-fiber, cost-effective, locally available feed ingredients without

compromising performance makes Axxess XY an asset for profitability.

In a recently conducted 42-day trial at a commercial farm, Axxess XY maintained the average body weight of broilers with a 100 kcal/kg reduction in metabolizable energy while significantly reducing feed cost/kg body weight. The diets were based on corn, DDGS, and soybean meal.

Figures 4 and 5: Body weight and cost of feed in broilers fed a diet reduced by 100 kcal/kg in metabolizable energy compared to a standard diet without Axxess XY



Conclusion

Xylanase exemplifies how feed enzymes can transcend their traditional role in feed cost reduction to support enhanced gut health. Xylanase supports reduced antimicrobial use in poultry production by improving nutrient utilization, reducing digesta viscosity, and fostering healthy microbiota. Its integration into comprehensive gut health management strategies offers a sustainable pathway to combat AMR and ensure the long-term viability of poultry farming. By targeting NSPs, these enzymes enhance nutrient digestibility, reduce feed costs, and support sustainable production practices.

GH10 xylanases, particularly Axxess XY, stand out for their superior substrate specificity, catalytic efficiency, and thermostability. By incorporating **Axxess XY** into feed formulations, poultry producers can unlock the full nutritional potential of feed ingredients, ensuring optimal performance and profitability. As the poultry industry continues to evolve, adopting advanced enzyme technologies like Axxess XY represents a strategic step toward sustainable and efficient animal nutrition.

References:

Baker, J.T.; Duarte, M.E.; Holanda, D.M.; Kim, S.W. Friend or Foe? Impacts of Dietary Xylans, Xylooligosaccharides, and Xylanases on Intestinal Health and Growth Performance of Monogastric Animals. Animals 2021, 11, 609.

Choct, M., and G. Annison. “Anti-nutritive Effect of Wheat Pentosans in Broiler Chickens: Roles of Viscosity and Gut Microflora.” British Poultry Science 33, no. 4 (September 1992): 821–34.

https://doi.org/10.1080/00071669208417524.

Liu D, Guo S, Guo Y. Xylanase supplementation to a wheat-based diet alleviated the intestinal mucosal barrier impairment of broiler chickens challenged by Clostridium perfringens. Avian Pathol. 2012;41(3):291-8.

Matthiesen, Connie F., Dan Pettersson, Adam Smith, Ninfa R. Pedersen, and Adam. C. Storm. “Exogenous Xylanase Improves Broiler Production Efficiency by Increasing Proximal Small Intestine Digestion of Crude Protein and Starch in Wheat-Based Diets of Various Viscosities.” Animal Feed Science and Technology 272 (February 2021): 114739. https://doi.org/10.1016/j.anifeedsci.2020.114739.

Teirlynck, E.; Haesebrouck, F.; Pasmans, F.; Dewulf, J.; Ducatelle, R.; van Immerseel, F. The cereal type in feed influences Salmonella enteritidis colonization in broilers. Poult. Sci. 2009, 88, 2108–2112.