Egg antibody technology for nursery pig application

Pigs at birth having insufficient immunity are simply not able to cope with the stress situations they face early in life. They of course become susceptible to the many pathogens common in the farrowing house. The resulting negative effects are added medical costs for treating the pigs and often an increased mortality. Strengthening the immune system by applying egg antibodies (IgY) during the first days of piglet's life is a proven viable option.

Immunity in pigs

Humans and animals are protected against diseases by specific antibodies (AB). Newborns receive the antibodies maternally (**passive immunity**) and they produce them after contact with pathogens (**active immunity**). But unlike humans, who receive maternal AB within the womb, sows possess a multi-layered placenta which prevents the transfer of AB during gestation. Therefore, an early intake of AB from colostrum is essential. This intake should begin immediately after birth as absorption decreases with every hour. But, the maternal antibodies are only a “starter immune kit”. The young pigs immediately must begin to develop their own “active immunity”.

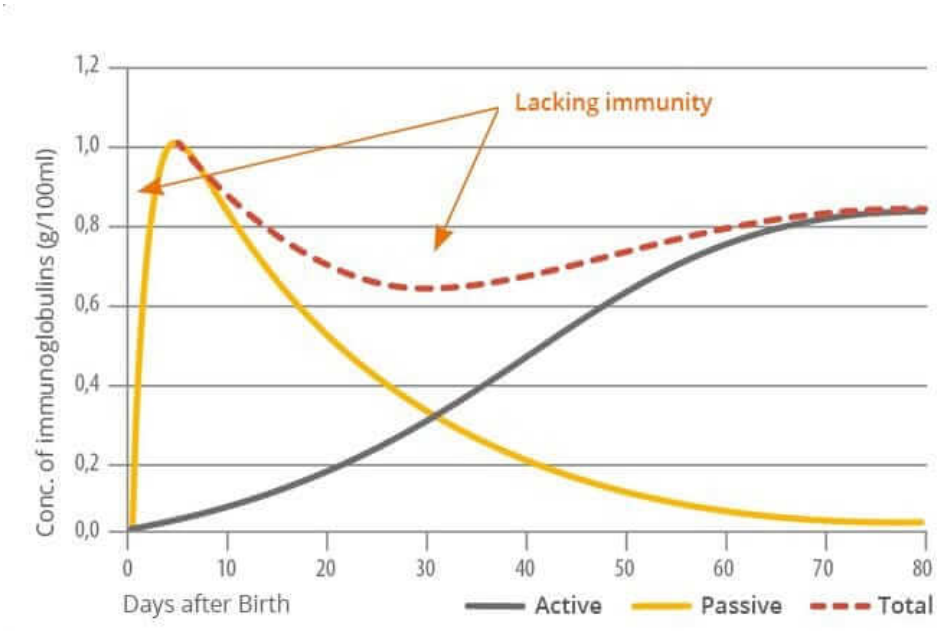


Figure 1: Immune status of the young pig (*Sieverding, 2000*)

Figure 1 shows gaps of low immunity shortly after birth and about six weeks after, as the level of passive immunity begins to drop and the active immunity starts to build up. The strength of the passive immune protection depends on quantity and quality of the colostrum consumed by the nursery pig. The quality is determined by the pathogens the sows have been confronted with during their life. Young gilts and sows with only short adaptation time into the herd often do not have the farm-specific antibodies needed to pass to their nursing pigs.

How can egg antibodies serve as a tool ?

Young pigs are challenged by different pathogens (see figure 2). From studies made by the German internist Felix Klemperer (*Klemperer, 1893*) we know that hens which come in contact with pathogens (in his studies with tetanus bacillus) produce antibodies against these pathogens. The antibodies are transferred to the egg yolk and are intended for being a starter protection kit for the chicks.

Technology allows us today to produce a highly valuable product based on egg powder. It contains significant amounts of natural egg immunoglobulins (IgY – **i**mmuno**g**lobulins from the **y**olk). These egg antibodies mainly act in the gut. There they recognize and tie up pathogens and in this way render them ineffective.

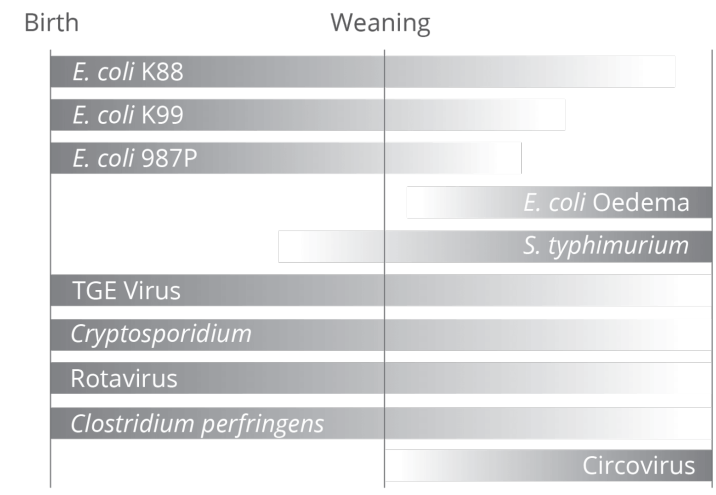
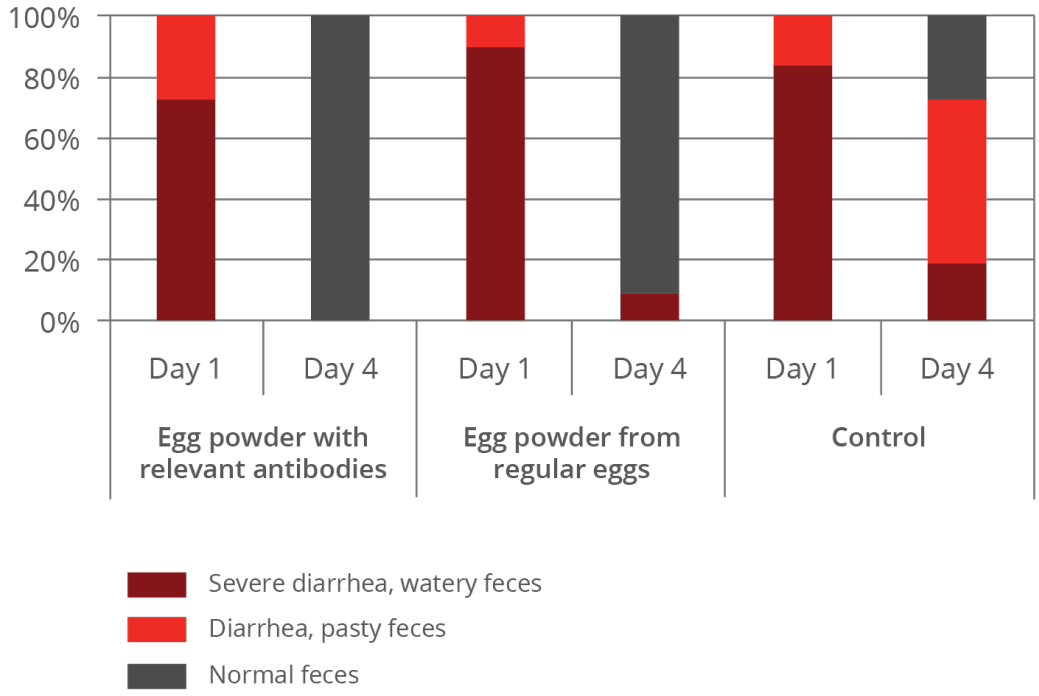


Figure 2: Commonly occurring pathogens causing diarrhea in pigs as they age

Not all egg powders are equal



Early work done by Kellner et al. (1994) showed the effectiveness of egg powder containing relevant antibodies against diarrhea causing pathogens in nursery pigs. In the trial they evaluated three groups receiving egg powder with relevant antibodies, egg powder from regular eggs or no additive (negative control).

Results:
(Figure 3: Effects of egg powder with relevant antibodies and egg powder from regular eggs in comparison to a negative control):

- The group that received egg powder containing relevant antibodies completely recovered from diarrhea on day 4.
- In the group fed normal egg powder on day 4 still 9 % suffered from severe diarrhea.
- In the control more than 70 % showed either severe or light diarrhea.

The results show that the effectiveness of egg powder depends on its content of antibodies.

Reducing mortality by oral administration of egg antibodies

The effectiveness of egg antibodies in pigs was demonstrated also in other studies (Erhard et al., 1996, Yokoyama et al., 1992, Nguyen et al., 2005, Yokoyama et al., 1997). One trial conducted in Germany showed promising results concerning reduction of mortality in the farrowing unit. For the trial 96 sows and their litters were divided evenly into three groups (32 sows each) and the pigs were treated as follows:

Group	Number of pigs	Treatment
Negative Control	530	no treatment

Group EP – 1+3	494	egg powder-based product Globigen Pig Doser, 4 ml on day 1, 2 ml on day 3
Group EP – 1, 2, 3	527	egg powder-based product Globigen Pig Doser, 4 ml on day 1, 2 ml on day 2 and 3

**EP = Egg powder-based product*

Results:
Figure 4 shows regardless of the frequency of oral application dosage given to pigs both were very supportive and significantly reduced mortality compared to the control. This resulted in a higher number of weaned pigs than in the control.

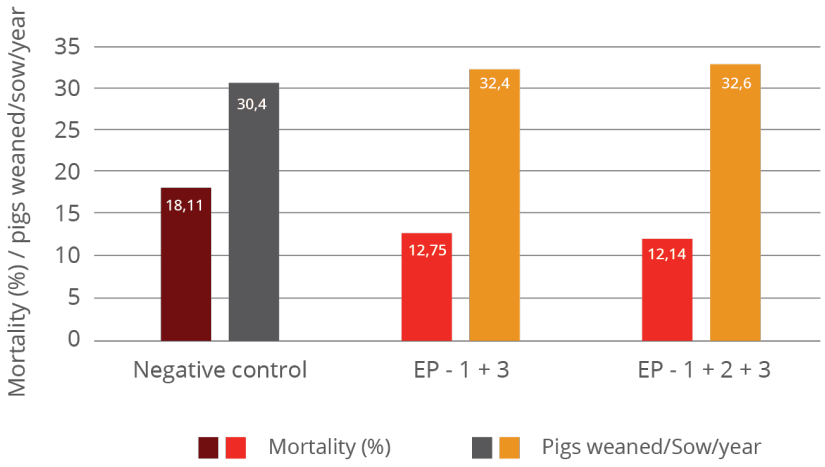


Figure 4: Mortality and resulting number of pigs weaned per sow and year

Conclusion

Using egg antibodies in pig nutrition is an effective tool to reduce mortality in young pigs. They can be applied individually by doser (newly weaned pigs) or via powder in the feed. Both practices have proven effectively in commercial operations.

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Understanding the dangers of mycotoxins for breeder hens

As the producers of hatching eggs and day-old chicks, breeding hens are the backbone of the poultry industry. Hence it is common practice to pay particular attention to this valuable asset's feed, selecting raw materials of high nutritional quality and safety. However, in any feed formulated for animals in production and reproduction, studies show that it is almost inevitable to find a certain level of mycotoxin contamination.

Mycotoxins exert toxic effects mainly on the gastrointestinal tract, liver, and kidneys and can accumulate in some tissues but also in the eggs. Mycotoxin contamination in breeder hens rations does not always lead to visible symptoms, such as when trichothecenes cause oral lesions. However, it may influence productivity, egg quality, hatchery performance, as well as chick quality and immunity. Mycotoxin risk management is thus an essential part of managing breeder hens. Mycotoxins can negatively affect eggshell quality and, as a consequence, embryonic mortality.

*By **Technical Team**, EW Nutrition.*

Type of mycotoxin and exposure time determine effect on egg production

Mycotoxicosis in hens can cause reduced egg production, most likely because it causes a decrease in protein synthesis. A lower synthesis of albumin results from a degeneration of the liver tissue due to aflatoxin, ochratoxin, T2 and DON exposure. The liver then may look pale, friable and occasionally shows superficial hemorrhages.

The contamination levels at which these effects can be observed are as low as 100ppb in feed, for example, during a 21-day exposure to ochratoxin (*Figure 1*). With increasing levels of the toxin, production further decreases. A similar effect is observed when breeder hens are exposed to aflatoxins.

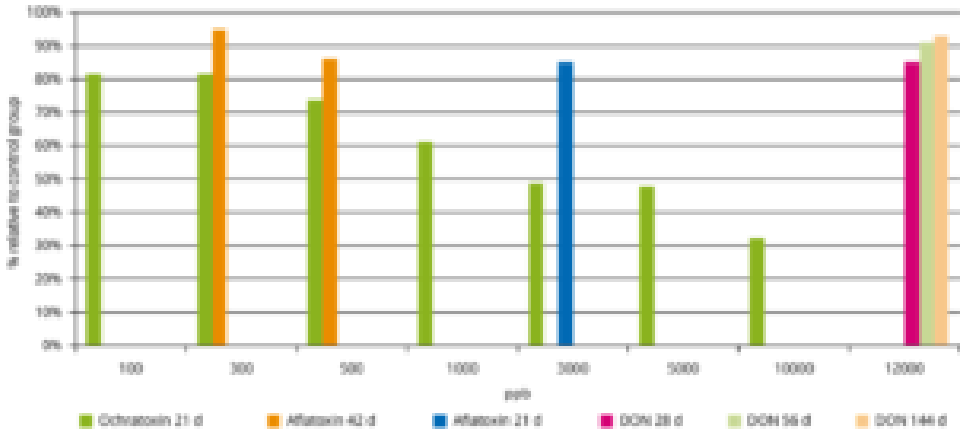

Figure 1 – Effect of mycotoxins on egg production, compared to non-contaminated control (=100 %)

Egg production, however, is not the only parameter that is affected when breeding hens are exposed to mycotoxins. Earlier on in the reproductive cycle, they already impact on embryonic mortality and hatchability. These effects are potentially more severe and may even occur without any noticeable change in the number of eggs produced.

Mycotoxins' insidious consequences for eggshell quality and embryonic mortality

The eggshell is important to protect the progeny: thin and fragile shells can increase embryonic mortality, lower embryonic weight gain and decrease hatchability. Eggshell quality is a function of the hen's calcium and vitamin D3 metabolism. The bioavailability of calcium and of vitamin D3 depends on intestinal integrity and on the production of enzymes and transporters that aid in feed metabolism. These processes can be adversely affected by aflatoxins, DON, T2, and Fumonisins.

The gastrointestinal tract is not the only site of mycotoxin action, however. Mycotoxins such as aflatoxins and ochratoxins have nephrotoxic effects, affecting calcium metabolism and increasing its excretion via the urine, while lowering its levels in blood serum.

Moreover, mycotoxins damage the liver, which plays a central role in egg production, being responsible for vitamin D3 metabolism and the synthesis of the lipids that make up the yolk. Moreover, the synthesis of transporters for lipids, calcium, and carotenoids − important components of the egg− also takes place in the liver. When liver function is impaired, the internal and external quality of the egg declines, which, in the end, affects the production of day-old chicks.

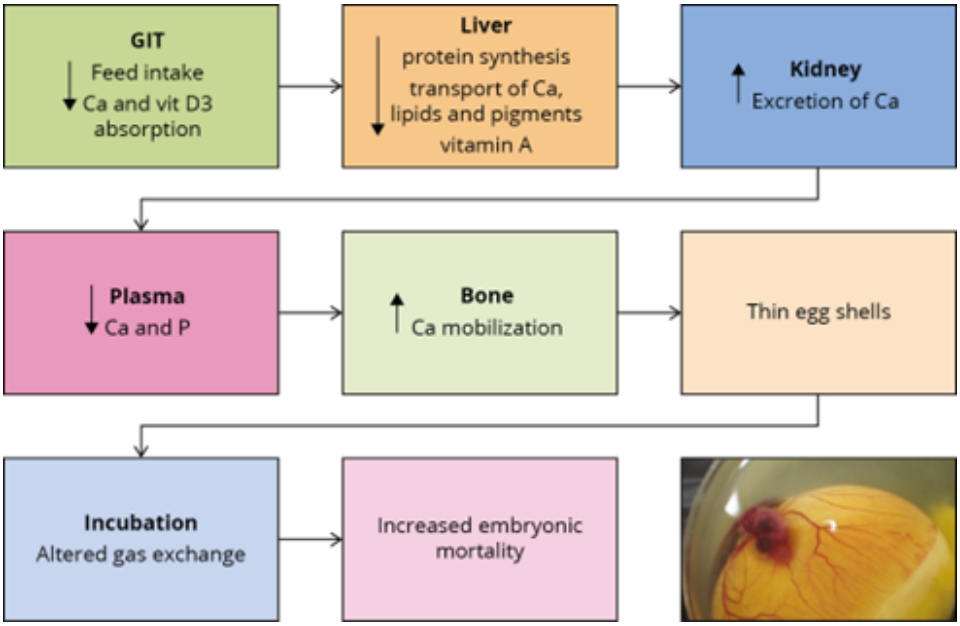

Figure 2 – Effects of mycotoxins on eggshell quality and embryonic mortality

Figure 2 summarises the possible ways in which mycotoxins can negatively affect eggshell quality and, as a consequence, increase embryonic mortality. If a hen's intestinal integrity is compromised, the utilization of nutrients decreases. Liver and kidney damage leads to a diminished availability of calcium and other nutrients necessary for egg formation. The birds' calcium (and phosphorus) levels in the plasma are then lower and may lead to a greater mobilization of calcium from the bones. However, this response cannot be maintained and the eggs get a thinner shell.

The thickness of the eggshell influences the egg's moisture loss and exchange with the environment during the incubation period. An eggshell of optimal quality does not allow the loss of nutrients and prevents bacterial contamination. Thinner eggshells are less able to fulfill these functions, leading to higher embryo mortality.

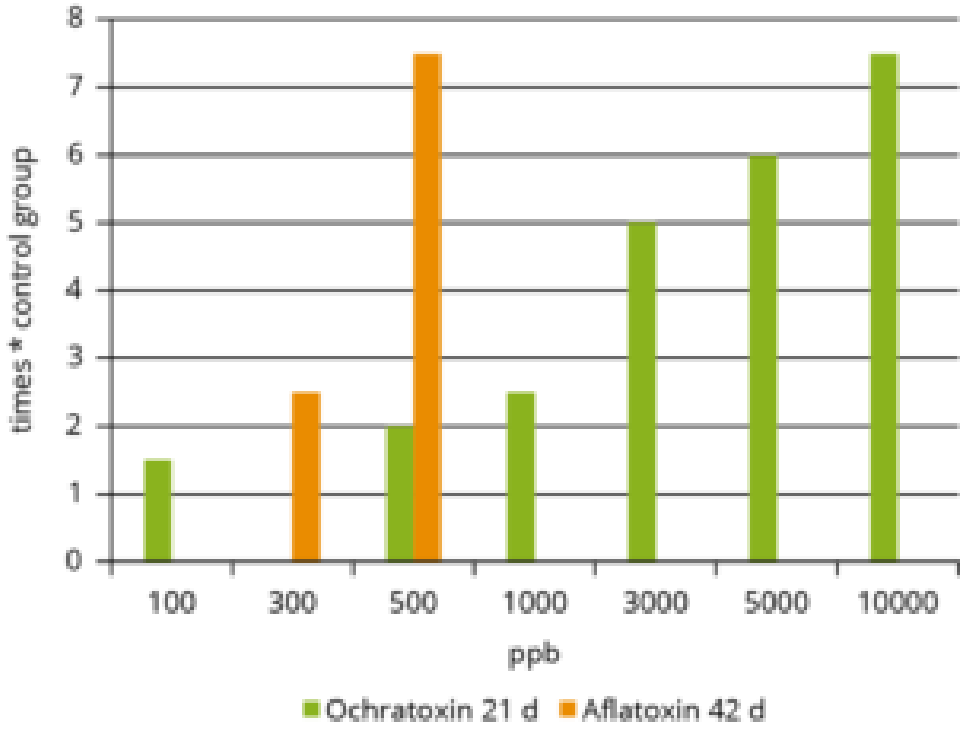

Figure 3 – Effects of mycotoxins on embryonic mortality

Figure 3 shows the effect of different mycotoxins on embryonic mortality. Incremental levels of ochratoxin or aflatoxin heighten embryonic mortality in a range from 1.5 to 7.5 times the embryonic mortality of the control group. In some cases, embryos are affected even when the hens received feed contaminated with mycotoxin levels that are within the guidelines suggested by the EFSA.

For example, an exposure to 4900ppb of DON for ten weeks increases the number of embryos with abnormalities. The causes are not entirely clear, as only traces of DON can be found in the egg. However, we do know that this mycotoxin can affect the protein synthesis at the level of the hen's liver and therefore compromise the deposition of nutrients into the egg.

Mycotoxins' effects on the progeny may cause long-term damage

Ochratoxin and aflatoxin can be transferred into the egg, where they exert toxicity on the embryos. This does not necessarily result in mortality. However, the chicks can suffer from a compromised immune function due to two reasons: lower transmission of antibodies from the hen and lower viability of the chickens' immune cells, accompanied by a lower relative weight of the bursa of Fabricio and the thymus.

When both aflatoxin and ochratoxin are present in the feed, the effect on these parameters is synergistic. As a consequence of mycotoxin contamination, the animals' immune response is impaired, which makes them more susceptible to infection. The final result could be increased early chick mortality due to a higher incidence of bacterial and viral infections.

The transmission of other mycotoxins into the egg is minimal. While this means that a direct effect on the progeny is unlikely to occur, mycotoxin contamination still has a snowball effect: we have to consider the indirect effect of a lower deposition of nutrients on chick quality.

Prevention is key: mycotoxin risk management

for breeder hens

The best approach to manage mycotoxin risk is to implement an integrated strategy that includes good crop and grain storing practices, regular raw material sampling and mycotoxin evaluation and analysis. Management tools (such as MasterRisk) can help to evaluate mycotoxin interactions and to choose the best strategy for dealing with specific mycotoxin challenges.

The results of mycotoxin analyses can be used to take decisions regarding the inclusion levels of raw materials and in choosing feed additives that counteract mycotoxins. Products based on plant extracts, yeast cell walls, and clay minerals can help to stabilize a digestive system challenged by mycotoxins. They support the barrier function in the intestine, preventing the passage of mycotoxins into the bloodstream.

Phytomolecules are another piece of the puzzle: thanks to their antimicrobial, anti-inflammatory and antioxidant properties, they support liver function. This is particularly important for long-living animals prone to accumulating mycotoxins in their body tissues.

For a long time the "deleterious effects" of mycotoxins on breeder hens and "their repercussions on progeny health status and performance have not received from a scientific point of view as much attention"(Calini and Sirri, 2007) as they ought to have. However, now that the dangers of mycotoxins for breeder hens' welfare, health and performance are better understood, it is clear that mycotoxin risk evaluation and management is central to successful poultry production.

*This article first appeared in All About Feed on 31 October 2018

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Photo: Hans Prinsen.

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Beyond AGPs: Controlling necrotic enteritis through gut health optimization

Antibiotic growth promoters (AGPs) have routinely been used in intensive poultry production for improving birds' performance. However, in recent years, reducing the use of antibiotics in animal production has become a top priority, due to concerns about the development of antibiotic-resistant bacteria and mounting consumer pressure. Multiple countries have introduced bans or severe restrictions on the non-

therapeutic use of antibiotics, including in the US, where the Food and Drug Administration has implemented measures to curb the use of antibiotics since 2017.

However, the removal of AGPs poses challenges for poultry performance, including reduced feed efficiency, decreased daily weight gain, as well as higher mortality. Moreover, the withdrawal of AGPs in feed is widely recognized as one of the predisposing factors for necrotic enteritis (NE). NE is one of the most common and economically important poultry diseases, with an estimated global impact of US$ 5 to 6 billion per year. As a result of withdrawing AGPs, the usage of therapeutic antibiotics to treat NE has increased. To break out of this vicious cycle and to secure the efficiency of poultry production, alternatives are needed that combat NE where it starts: in the gut.

Necrotic enteritis: a complex disease

NE is caused by pathogenic strains of *Clostridium perfringens* (CP): ubiquitous, gram-positive, spore-forming anaerobic bacteria. The spores of CP can be found in poultry litter, feces, soil, dust, and contaminated feed. Low levels of different CP strains are naturally present in the intestines of healthy birds, kept in check by a balanced microbiome. However, when gut health is compromised, pathogenic strains can proliferate at the expense of unproblematic strains, resulting in clinical or sub-clinical NE.

Animals suffering from the clinical form show symptoms such as general depression, reluctance to move, and diarrhea, with mortality rates of up to 50%. Infected birds suffer from degenerated mucosa lesions in the small intestines. Even in its “mild”, subclinical form, which often goes unnoticed, the damage to the animals’ intestinal mucosa can result in permanently reduced performance and consequent economic losses for the producer.

Certain predisposing factors have been found to enable the proliferation of pathogenic strains in the gastrointestinal tract. Diet is a key example: the composition of the gut flora is directly linked to feed composition. High inclusion rates of cereals (barley, rye, oats, and wheat) that contain high levels of non-starch polysaccharides (NSPs), high levels of indigestible protein, and inclusion of proteins of animal origin (e.g. fishmeal) have been shown to predispose birds to NE.

A range of diseases (e.g. chicken infectious anemia, Gumboro, and Marek’s disease), but also other factors that have immunosuppressive effects, such as heat or cold stress, mycotoxins, feed changes, or high stocking density, render birds more susceptible to intestinal infections. The single most prominent predisposing factor for the occurrence of NE is the mucosal damage caused by coccidiosis.

Gut health is key to combating necrotic enteritis

To control NE, a holistic approach to optimizing the intestinal health of poultry is needed. It should take into account not only parameters such as diet, hygiene, and stress, but should also make use of innovative tools.

Phytomolecules, also known as secondary plant compounds, are essentially plants’ defense mechanisms against pathogens such as moulds, yeasts, and bacteria. Studies have demonstrated the antimicrobial effects of certain phytomolecules, including against antibiotic-resistant pathogens. Phytomolecules have also been found to boost the production of digestive enzymes, to suppress pro-inflammatory prostaglandins and have antioxidant properties. These features make them a potent tool for optimizing gut health, potentially to the point of replacing AGPs.

Can phytomolecules mitigate the impact of necrotic enteritis?

To study the impact of phytomolecules on the performance of broilers challenged with a NE-causing CP strain, a trial was conducted at a US-based research facility. In this 42-day study, 1050 male day-old Cobb 500 broiler chicks were divided into 3 groups, with 7 replicates of 50 chicks each.

On the first day, all animals were vaccinated against coccidiosis through a live oocyst spray vaccination. The experimental diets met or exceeded the National Research Council requirements, and were fed as crumbles/pellets. On days 19, 20, and 21, all pens, except the negative control group, were challenged with a broth culture of *C. perfringens*. A field isolate of CP known to cause NE (originating from a commercial broiler operation) was utilized as the challenge organism. On day 21, three birds from each pen were selected, sacrificed, group weighed, and examined for the degree of present NE lesions.

The positive control group received no supplements. The trial group received a synergistic combination of two phytogenic products containing standardized amounts of selected, microencapsulated phytomolecules: an in-feed phytogenic premix (Activo, EW Nutrition GmbH) and a liquid complementary feed supplied via the drinking water (Activo Liquid, EW Nutrition GmbH). The products were given at inclusion rates corresponding to the manufacturer's baseline antibiotic reduction program recommendations (Figure 1):

Figure 1: Trial design

Trial Groups	Challenge with NE-causing CP strain	Supplements
Negative control	No	No
Positive control	Yes	No
Activo + Activo Liquid	Yes	Activo 100g/MT + Activo Liquid at 250ml per 1000l on days 12-14 for 24 hrs per day, on days 19-21 for 16 hrs per day

The trial results indicate that the addition of phytomolecules helps to mitigate the impact of NE on broilers' performance. The group receiving Activo and Activo Liquid showed a better feed conversion (Figure 2) compared to the positive control group (NE challenge, no supplement). Also, better lesion scores were noted for animals receiving phytomolecules (0.7 and 1) than for the positive control group (1.6).

The most significant effect was observed concerning mortality: the group receiving Activo and Activo Liquid showed a 50% lower mortality rate than the positive control group (Figure 3). These results clearly indicate that phytomolecules can play an important role in mitigating losses due to NE.

Figure 2: Adjusted FCR

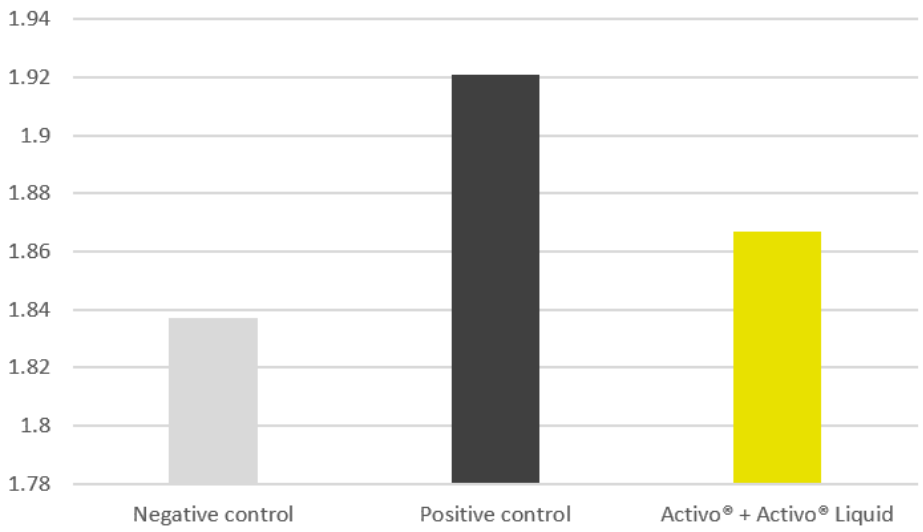
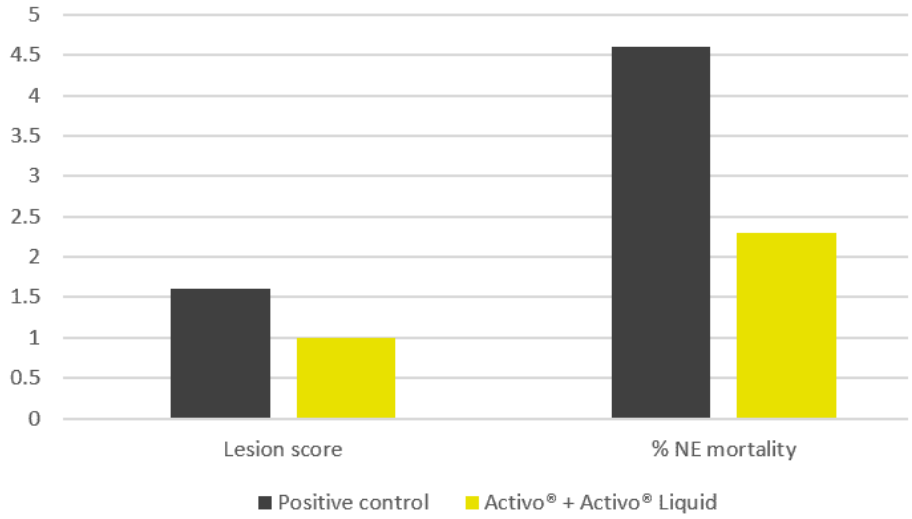


Figure 3: Lesion scores and mortality



Tackling necrotic enteritis in a sustainable way

In an age of AGP-free poultry production, a concerted focus on fostering animals' gut health is key to achieving optimal performance. This study strongly demonstrates that, thanks to their antimicrobial, digestive, anti-inflammatory and antioxidant properties, phytomolecules effectively support birds' intestinal health when challenged with NE. The inclusion of Activo and Activo Liquid, two phytogenic products designed to synergistically support birds during critical periods, resulted in improved feed conversion, better lesion scores, and 50% lower mortality.

In combination with good dietary, hygiene, and management practices, phytomolecules are therefore a potent tool for reducing the use of antibiotics: including Activo and Activo Liquid in their animals' diets allows poultry producers to reduce the incidence of NE, to mitigate its economic impact in case of outbreaks, and therefore to control NE in a sustainable way.

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