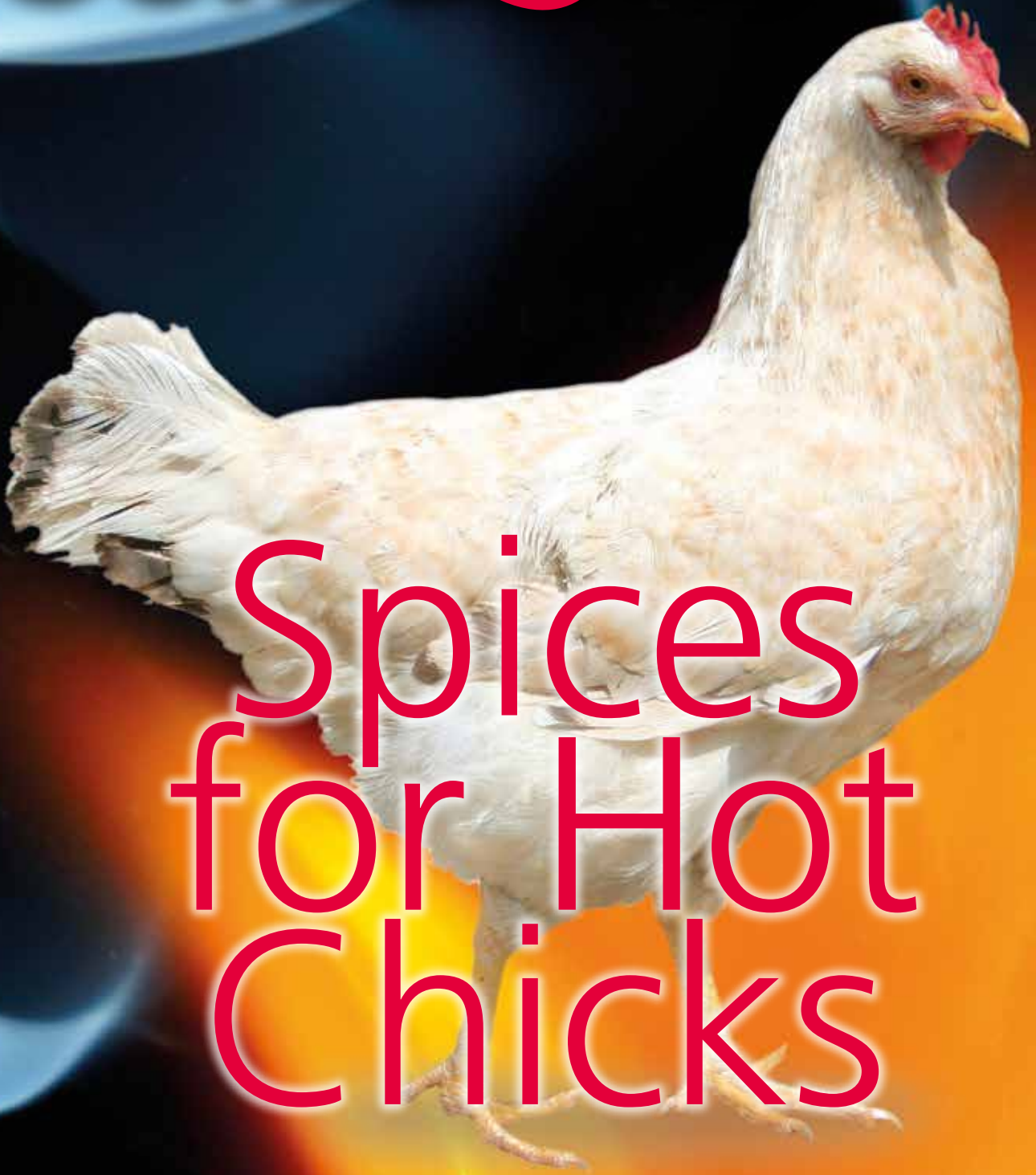


Science & Solutions



Spices for Hot Chicks

Photos: Ievgeniia Mukhomet, silverjohn



**The Harm of
Endotoxins**



**What's Wrong
with My Birds?**

Part 7: Avian Gout

Photo: fotostorm

Editorial

Looking forward to summer

With spring blossoming in the northern hemisphere thoughts will be turning not only to the summer holidays but to ways of dealing with excessive heat in the poultry house during summer. Dry heat alone can be dealt with, but the higher the humidity, then the more difficult this becomes. Even in countries we assume are not humid in hot weather, the humidity can be higher than suspected and exacerbate the situation.

The outcome of heat stress can range from reductions in feed intake and performance in breeders, broilers and layers through to immune suppression and eventually death. In this issue Dr. Tan looks at the problem of heat stress and ways to alleviate it through management and nutritional factors.

The poultry industry is becoming more aware of the effects on performance caused by naturally occurring endotoxins; produced from the breakdown of the cell walls of Gram-negative bacteria found in the intestine. Another aspect of heat stress is that the increase of internal body temperature by as little as 1-2°C in some species increases the permeability of the intestinal membrane; possibly through weakening of the tight junctions. This can lead to more endotoxins reaching the blood system. The subject of endotoxins is discussed in the second article by Dr. Schaumberger.

Finally in the “What’s wrong with my birds” section we discuss the nutritional factors which can influence an increase in avian gout which can be of particular importance in the early lay period of commercial layers.



Andrew ROBERTSON

Poultry Technical Manager



Contents



Photo: AndreyGonulko

Spices for Hot Chicks: Phytochemicals for Heat-Stressed Birds

2

In both warmer and cooler climates, phytochemical feed additives in poultry diets help alleviate the negative impacts of heat stress.

By **Justin TAN, DVM**



Endotoxins and Their Negative Impact on Poultry

6

Lipopolysaccharides can impair birds' immune system and performance. Adsorption can be a viable solution.

By **Simone Schaumberger, PhD**



What's Wrong with My Birds? Part 7: Avian Gout

9

A handy diagnostic checklist of symptoms, causes and remedies.

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Editor: Ryan Hines
Contributors: Andrew Robertson, Simone Schaumberger, Justin Tan, Luca Vandl
Marketing: Herbert Kneissl, Karin Nährer
Graphics: Reinhold Gallbrunner, Michaela Hössinger
Research: Franz Waxenecker, Ursula Hofstetter, Paolo Doncecchi
Publisher: BIOMIN Holding GmbH
Erber Campus, 3131 Getzersdorf, Austria
Tel: +43 2782 8030
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Spices for Hot Chicks

Phytogenics for Heat-Stressed Birds

By **Justin TAN**, Regional Sales & Marketing Director, Asia Pacific

High ambient temperatures mean detrimental performance and reduced profits for producers. Extreme cases cause suffering and death in all poultry breeds. Phytogenic feed additives in poultry diets help alleviate the negative impacts of heat stress by exerting an anti-inflammatory and antioxidant effect in birds.



P

oultry producers commonly face the challenge of heat stress either seasonally or year-round. Poultry farmed in hot and humid countries are genetically derived from strains originally bred in, and selected for, the cool climates of Europe and North America. Rearing birds outside of their thermal comfort zone could mean failing to achieve full genetic potential. Producers in warmer climates or those in cooler zones who adjust their shed temperatures to their own comfort levels, not to that of their birds, should consider the impact of heat stress on flocks.

Passing the upper critical bound

For any stage and any species, heat stress occurs when birds have difficulty balancing body heat production and body heat loss. Within the thermoneutral zone illustrated

Figure 1. The thermoneutral zone and consequences of heat stress.

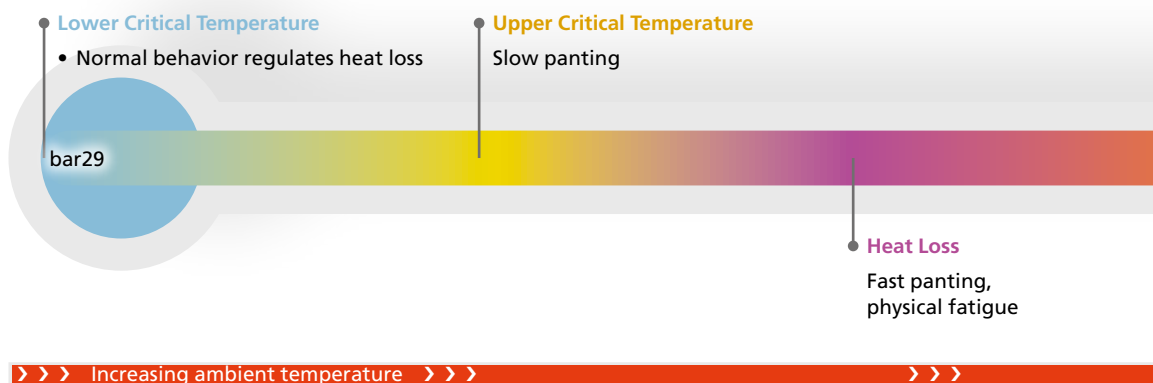
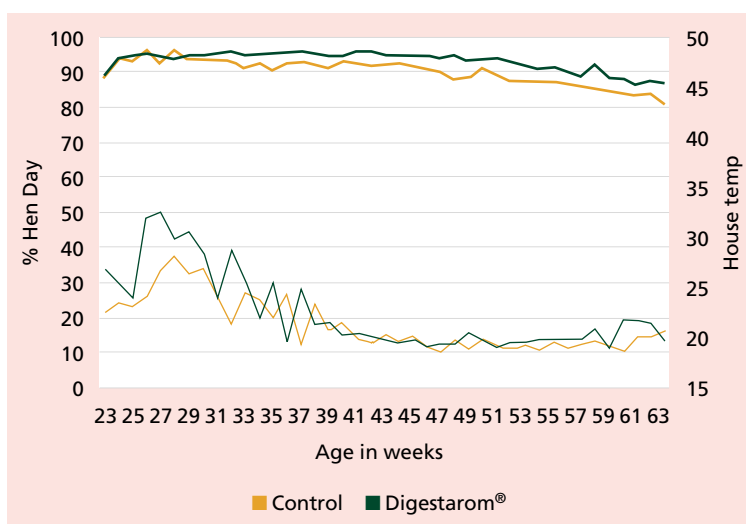


Figure 2. Hen day performance and house temperatures.



Source: BIOMIN

in Figure 1, birds are able to lose heat at a controlled rate by modifying their behavior. There is no heat stress at this point and their body temperature remains constant.

At or above the upper critical temperature birds begin to pant to actively lose body heat. Panting is considered a normal response to heat, but as temperatures continue to increase, the rate of panting increases concurrently. If heat production becomes greater than

maximum heat loss either in intensity (acute heat stress) or over prolonged periods (chronic heat stress), birds very quickly begin to die. The body temperature of the broiler must remain very close to 41°C. If body temperature rises above 45°C, death starts to occur rapidly.

Strategies to overcome heat stress

Various measures can be taken to prevent heat stress (Table 1). A number of options exist for nutrition and feeding strategy approaches.

Heat stress induces unfavorable changes in indigenous bacterial microbiota. Supplementation of multi-strain probiotics may enrich the diversity of microbiota in the bird’s jejunum and caecum, restoring the microbial balance and maintaining their natural stability.

Temporary feed restriction before heat exposure can enhance thermal resistance of broilers. Feed withdrawal reduces heat production, increment speed of body temperature and mortality of broiler chickens. However, this strategy may also result in reduced growth rate, a longer growing period and a delay in slaughter age.

The dual feeding program is another strategy used for broilers, which includes a protein diet during the cooler phase and an energy-rich diet during the warmer phase of each day. This maintains a nutritional balance by adequate proportion of the two diets. During a heat stress challenge, dual feeding reduces body temperature and mortality.

In laying hens, partial feed restriction or a controlled feeding regime alleviates the harmful effect of heat stress on laying performance. Changing the feeding time from twice to just once daily is also favorable to the performance of laying hens. The best time to feed the flock would be in the afternoon near sunset.

Layer diets are normally provided in mash form, but consider pellets during summer. Although feed consumption might not be affected by pelleting the ration, egg production, feed efficiency and water intake can be significantly increased in laying hens. Increased water consumption and improved digestibility of the

Table 1. Measures to counteract heat stress.

Provide adequate ventilation for number of birds housed
Reduce stocking densities
Insulate sheds sufficiently to avoid solar heat gain
Position fans to optimize wind speed and air circulation
Use evaporative cooling pads or atomizing nozzle
Maintain water-electrolyte balance
Vitamin supplementation

Source: BIOMIN

Maximum Heat Loss

Birds cannot control body temperature

Death From Heat

Figure 3. Trial parameters and results of the heat stress study in commercial layers.

		Control	Digestarom®	Absolute Difference
Trial weeks	n	41	41	
Av. Laying Rate	%	86.9	89.2	+ 2.3
Eggs produced	n	249.5	256.0	+ 6.5
Av. Dirty eggs	%	9.88	9.80	- 0.08
Av. Cracked eggs	%	0.64	0.65	+ 0.01
Av. Feed intake	g/bird/day	114	107	- 7
Av. Egg weight	g	62.71	63.01	+ 0.3
Produced egg mass	kg/hen/287d	15.65	16.13	+ 0.48
Feed conversion rate (FCR)		2.10	1.91	- 0.19

Source: BIOMIN

diet is probably responsible for the advantageous effect of pelleting. Broiler chickens, however, prefer to eat more feed with larger particle size in hot environments. When corn is fed as whole grains, broilers consume more protein and have an improved feed efficiency.


Phytogenics to combat heat stress

Phytogetic feed additives (PFAs, or botanicals) are able to alleviate the negative consequences of heat stress. A recent study was conducted in Germany with a flock of Lohmann Brown Classic birds that came to lay during the early summer months at a time of high temperatures recorded in the hen houses. The birds were divided into two groups, with the control group being fed with a basal diet, while the treatment group received the same basal diet but with the inclusion of Digestarom® Poultry (a phytogetic feed additive) at an inclusion rate of 150 g per ton of feed. The trial ran from the flock age of 23 to 63 weeks.

The effects of the phytogetic feed additive during the heat stress period are displayed in *Figure 2* below. The group of hens supplemented with Digestarom® in their diets consistently outperformed the control group throughout the 41 weeks in terms of hen day production, maintaining a steady peak amid the heat

stress period, despite recording higher house temperatures, due to less efficient insulation and ventilation in the older building compared to that of the control group. The average hen day production of the Digestarom® group was 89.2% compared to 86.9% in the control group, representing a 2.3% increase in laying rate. There was also an improvement of feed conversion of 19 points in the birds fed diets containing the phytogetic feed additive, together with higher egg mass and average egg weights (*Figure 3*). The return on investment for the egg producer was 1 : 7.

Conclusion

Digestarom® positively helped the flock of commercial layers to maintain their peak production throughout a period of heat stress. The birds receiving Digestarom® had better laying performance, better feed efficiency, improved feed conversion ratio, higher profitability, higher return-on-investment and better health status, with no reports of any disease outbreak. Due to its scientifically proven mode of action and unique benefits for the birds, Digestarom® successfully helped to combat heat stress, proving once again its potential as a next generation feed additive for innovative poultry nutrition and contributing to increased profits for the producer. 



Endotoxins and their negative impact on poultry

By **Simone Schaumberger**, Mycotoxin Risk Management Product Manager

Endotoxins, present everywhere in a broiler's environment, pose a genuine threat that can impair immune system and performance. A multi-strategy (myco)toxin risk management solution can counteract both endotoxins and mycotoxins.

Endotoxins are part of the outer membrane of the cell wall of all Gram-negative bacteria (e.g. *E. coli*, *Salmonella*, *Shigella*, *Pseudomonas*) that are released from bacterial cell walls by shedding or through bacterial lysis. These toxins, also called lipopolysaccharides (LPS) because of their structure, consist of a lipid and a polysaccharide (Figure 1). While there are many natural sources of endotoxins, for example, air, dust, food, water, and feces, the major source is the gastrointestinal tract.

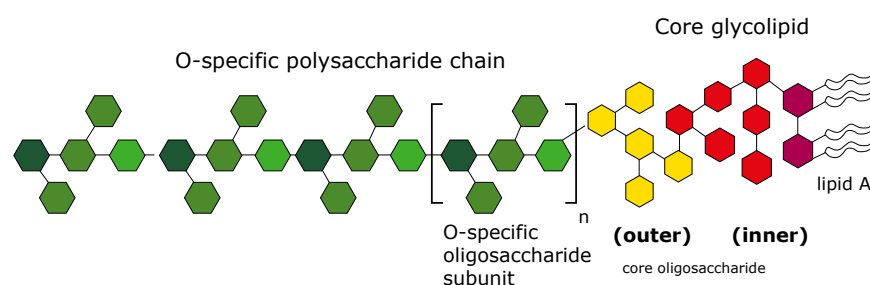
Endotoxin contamination – why we should care

Poultry are exposed to lipopolysaccharides throughout their lives. In healthy birds, the intestinal and other epitheliums such as skin or lungs, represent an effective barrier that prevents the passage of lipopolysaccharides into the bloodstream.

Once there, however, endotoxins can elicit strong immune responses, weakening birds' immune systems and impairing performance. Severely pronounced immune response can lead to septic shock.

Recent research shows that exposure to lipopolysaccharides through dust in the environment impairs the immune response in chickens and can lower resistance against pathogenic insults. For these reasons, greater care regarding endotoxin exposure is crucial for proper animal health and performance.

Figure 1. Gram-negative bacterial endotoxin (lipopolysaccharide, LPS).



Flock exposure

In recent years, new concerns have emerged about nutritional, environmental, and social factors that may disrupt the barrier function and/or increase exposure to lipopolysaccharides. Such exposure may result in clinical or sub-clinical signs that ultimately affect poultry production. Septic shock is very often related to infection by pathogenic Gram-negative bacteria that produce lipopolysaccharides.

However, the presence of lipopolysaccharides in blood can also be due to metabolic or gut barrier problems along with issues related to the intestinal microflora. Given that many of the factors vary from bird to bird, a combination of factors as a whole will result in disparate impacts within a flock.

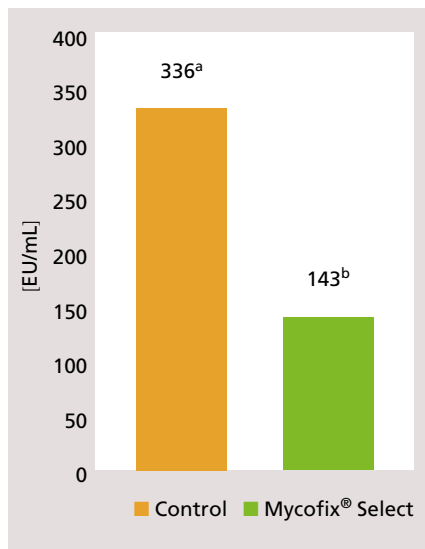
Dietary changes play a clear role. For example, moving birds from a corn-based diet to a rye-wheat-barley diet was shown



Photo: Sebastian Kautitzki

Endotoxin exposure may result in clinical or sub-clinical signs that ultimately affect poultry production.

Figure 2. Endotoxin content in gizzard (EU/mL).



Source: BIOMIN

to increase lipopolysaccharide levels in blood serum along with inflammatory markers.

Other factors can significantly contribute to endotoxin-associated problems. For instance, in summer high temperatures inside poultry units can induce heat stress. In other livestock species, it is well established that a 1-2° C increase in inner body temperature causes the intestinal tight junction proteins to be affected, thereby increasing intestinal permeability and allowing more lipopolysaccharides to enter the blood stream. Whether the same mechanism applies to poultry is currently under investigation.

Subtherapeutic use of antibiotics in some countries not only raises concerns

about antibiotic resistance but also about the change in gut microflora and the plausible release of lipopolysaccharides in the gut lumen that results from killing Gram-negative bacteria.

In addition, mycotoxins such as deoxynivalenol are also very well known to disrupt the intestinal barrier. The multitude of factors relating to lipopolysaccharide exposure helps explain the disparate impact on birds.

Reducing LPS in birds

A trial was conducted with the aim to investigate the influence of a multi-component mycotoxin deactivator (Mycofix® Select) on performance and health status of broiler chickens fed diets naturally contaminated with mycotoxins in an environment with high pathogen pressure (*E. coli*).

Over 600,000 day-old broiler chickens (Ross or Hubbard) were investigated in this field study. The animals were allocated on three different farms. On each farm two houses were compared at the same time (control vs Mycofix®). In each production cycle the birds were kept until 35 days of age. Feed contained a mixture of mycotoxins consisting of B-trichothecenes such as deoxynivalenol (200 ppb), fumonisins (470 ppb) and zearalenone (75 ppb).

Besides performance parameters, intestinal content samples were taken for surveying the endotoxin load. Figure 2 displays the results for the endotoxin concentration in stomach content of broilers at the end of the trial.

Conclusion

The addition of a multi-component mycotoxin deactivator proved to be effective counteracting low level of mycotoxin challenges in combination with *E. coli* pressure. Overall performance of broiler chickens was enhanced; the endotoxin load in the gut lowered and the negative effects of *E. coli* were reduced. These results reinforce the importance of counteracting the effects of endotoxins in order to protect birds' health and improve performance. 🌿

Environment and endotoxin exposure



Photo: r. drewek

While the main route for lipopolysaccharide exposure in poultry is the gastrointestinal tract, the concentration of endotoxins in the air and dust should not be overlooked - endotoxins are a major component of biological dust. Air endotoxin levels are an important issue not only for the animals, but also for workers in poultry houses. The highest concentrations of airborne endotoxins among livestock production facilities have been recorded on poultry farms at 310 to 1090 ng/m³ air.

This reinforces the importance of good management regarding hygiene and dust levels on farms, and specific measures to protect workers such as wearing a fine dust mask.

What's Wrong with My Birds?

Part 7: Avian Gout/Kidney Failure

Avian gout is a consequence of kidney damage which can occur from a number of potential causes leading to the accumulation of uric acid/urates in the renal tubules and serous coats of the heart, the liver, the mesentery, the air sacs or the peritoneum.

Due to its complex aetiology, it is difficult to diagnose; however, the most common signs are dehydration, pale combs, depression and swelling and reddening of the feet which impair bird movement. In layers, where it is mainly observed, avian gout can lead to mortalities up to 50%, with 19-35 week-old hens mostly affected.

The causes for this condition are varied (see table right), ranging from management and/or nutrition-related, to pathogens and/or the presence of mycotoxins in feed. In terms of nutrition, special attention must be paid to the calcium/phosphorus balance, sodium and vitamin D₃.

In general, any condition favouring an increase of uric acid in blood favours precipitation in tissue and, as a consequence, development of gout. Excess dietary calcium with low available phosphorus results in the precipitation of sodium-urate crystals and calcium pyrophosphate (pseudogout). In younger birds, gout due to sodium intoxication may be observed at sodium levels exceeding 0.4% in water and 0.8% in feed.

Likewise, high levels of vitamin D₃ can increase calcium absorption from the intestine favouring the formation and deposition of urate crystals. Also nutrition-related is the protein level in feed which in excess of 30% causes uric acid production leading to excretory loads in kidneys. Concurrently, sulphates decrease calcium resorption causing excess calcium secretion through the urine. This favors gout, as well as any other factor contributing to urine alkalinity. Water deprivation falls in this category as it leads to increased concentrations of uric acid and other minerals in the blood and later on in the kidneys and urine.

Viruses such as infectious bursal disease (IBDV) and/or infectious bronchitis can enhance mortalities in the presence of preexisting kidney damage.

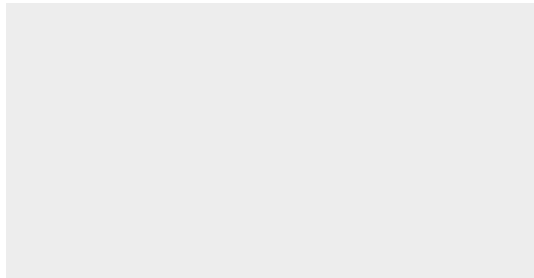
In terms of mycotoxin contamination of feeds, the nephrotoxic aflatoxins (Afla), ochratoxin A (OTA) and citrinin are of major concern. The impairment of the kidney function which results from the action of these mycotoxins reduces uric acid excretion and results in the accumulation of uric acid in the body.

Check list	Corrective action
Potential cause: MYCOTOXINS: Ochratoxin A (OTA), citrinin, aflatoxins (Afla)	
<ul style="list-style-type: none"> • Positive for Afla, citrinin and/or OTA in raw materials (ELISA) or feed (HPLC) • Raw materials originating from supplier/ region with history of mycotoxin contamination • Histopathology: Check other target organs of these mycotoxins (e.g. kidneys, liver) • Decline in overall flock performance 	<ul style="list-style-type: none"> • Check average contamination levels • Use Mycofix® at the correct dosage level • Avoid feed bins or feed/water lines becoming contaminated by stale, wet or mouldy feed
Potential cause: NUTRITION: Calcium, sodium, vitamin D₃	
<ul style="list-style-type: none"> • Level of minerals and vitamins in diets 	<ul style="list-style-type: none"> • Correct level of minerals and vitamin D₃ • Control fish meal usage (rich in salt) • Control total sodium chloride content in feed (<0.3%)
Potential cause: NUTRITION: Protein	
<ul style="list-style-type: none"> • Protein level in feeds 	<ul style="list-style-type: none"> • Correct protein level in feeds
Potential cause: MANAGEMENT: Water deprivation	
<ul style="list-style-type: none"> • Observe animal behavior to understand the cause of water deprivation • Transportation and vaccination procedures • Drinkers in terms of number, position and blockages that may impede access. • Chemicals added to water (disinfectants, copper sulphate, etc) may result in water refusal, dehydration and gout 	<ul style="list-style-type: none"> • Improve transportation condition of birds (access to water) • Adjust number, position and access to drinkers • Avoid overcrowding • Correct blockages in nipples
Potential cause: PATHOGENS: Infectious bursal disease (IBDV/Gumboro)	
<ul style="list-style-type: none"> • Maternal antibody titres are very low in day-old chicks 	<ul style="list-style-type: none"> • Adapt vaccination program to the demands of the field situation in each particular area/epidemiology • Increase biosecurity level
Potential cause: PATHOGENS: Infectious bronchitis (IB)	
<ul style="list-style-type: none"> • Laboratory tests to confirm the presence of the coronavirus in a swab or tissue sample 	<ul style="list-style-type: none"> • Adapt vaccination program to the demands of the field situation in each particular area/epidemiology

References are available on request

For more information, visit www.mycotoxins.info

DISCLAIMER: This table contains general advice on poultry-related matters which most commonly affect poultry and may be related to the presence of mycotoxins in feed. Poultry diseases and problems include, but are not confined to the ones present in the table. BIOMIN accepts no responsibility or liability whatsoever arising from or in any way connected with the use of this table or its content. Before acting on the basis of the contents of this table, advice should be obtained directly from your veterinarian.



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