Environmental Stress and Mycotoxins in Breeders: Hidden Losses in Fertility, Egg Quality & Chick Output

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In Tropical countries like India, Bangladesh & Sri Lanka, high temperatures and humidity significantly increase the risk of mycotoxin contamination in animal feed. FAO surveys report mycotoxin contamination in over 70% of cereals and oilseeds used for animal feed, making them a major threat to poultry health and productivity.

The problem becomes particularly severe during the summer & monsoon when:

- The new maize crop with high moisture enters the market
- Drying is often inadequate
- Feed mill humidity remains high

The Science of Mycotoxins & Environmental Risk

- Summer temperatures (28–38°C) and high humidity promote fungal growth and mycotoxin production in feed ingredients.
- Freshly harvested “new maize” often enters feed mills with unsafe moisture levels (13–15%), exceeding the safe storage limit of 11–12%.
- High-moisture grain undergoes self-heating during storage, creating ideal conditions for fungal proliferation.

- Poor aeration, condensation, insect damage, and humid storage environments further accelerate contamination.
- Common summer mycotoxins include Aflatoxins, Ochratoxins, and T-2 toxins, which may develop during both pre- and post-harvest stages.
- Although pelleting and heat treatment may destroy molds, mycotoxins are generally heat-stable and remain toxic to poultry.

Regional Case Studies: South Asia

India

Coastal Humidity Crisis – Andhra Pradesh, Telangana, Tamil Nadu, Odisha & West Bengal

- Pre-monsoon humidity promotes *Aspergillus flavus* growth in stored maize.
- Aflatoxin contamination commonly reduces shell quality and egg production.
- Fatty Liver Hemorrhagic Syndrome (FLHS) is frequently observed.

Short-Storage Trap – Punjab & Haryana

- Wet maize stored during summer heat retains internal moisture, favoring T-2 toxin and Ochratoxin formation.
- Broilers commonly show oral lesions, feed refusal, and poor FCR.

Mixed Toxin Challenge – Maharashtra

- Co-contamination with Aflatoxin and Fumonisin is common.
- Combined toxicity causes immunosuppression, poor vaccine response, and increased mortality during disease outbreaks.

Bangladesh

High Humidity Feed Risk

- Persistent humidity and post-flood harvesting increase moisture retention in maize and rice by-products.
- Aflatoxin frequently co-occurs with Ochratoxin A.
- Causes immunosuppression, uneven flock uniformity, and poor hatchability in breeders.

Sri Lanka

Tropical Storage Challenge

- Tropical coastal humidity and prolonged ingredient storage favor fungal proliferation.
- Aflatoxin and Fumonisin commonly develop during humid transit and storage.
- Causes thin shells, liver damage, and poor FCR.

Nepal

Mountain Moisture Variability

- Humid Terai grains stored in cool hill regions favor mixed mycotoxin contamination.
- Aflatoxins commonly co-occur with DON and Zearalenone.
- DON causes feed refusal , while Zearalenone induces prolapse and false layers.

Mycotoxins and High Temperature Humidity Index (THI): Synergistic effects on poultry health, immunity & productivity

The Immunological “Blackout”

- Aflatoxins, Trichothecenes, and Ochratoxins inhibit protein synthesis, reducing the formation of antibodies and immune cells.
- Mycotoxins cause atrophy of immune organs (bursa of Fabricius, thymus, and spleen)
- Macrophage activity and phagocytosis are reduced, weakening bacterial clearance.
- Cytokine signaling is disrupted, delaying immune activation against infections.
- Enhanced oxidative stress: Mycotoxins increase the occurrence of reactive oxygen species (ROS), and heat stress weakens antioxidant defenses, resulting in severe cellular and liver damage.
- Oxidative stress caused by aflatoxins and trichothecenes leads to immune cell apoptosis and tissue damage.

Gut Health & Barrier Damage

The gut is the first line of defense. Mycotoxins and heat stress act like a “chemical and physical abrasive” on the intestinal lining.

- Villi Destruction: T-2 and Aflatoxins cause necrosis (cell death) of the intestinal villi. This reduces the surface area for nutrient absorption, leading to poor FCR.

- The “Leaky Gut” Phenomenon: Heat stress causes blood to be diverted from the internal organs to the skin for cooling (vasodilation). As a result, the gut receives less oxygen, causing the tight junctions (the “glue” between intestinal cells) to break down and become permeable. Mycotoxins also have a direct effect, inhibiting tight junctions proteins.
- Pathogen Entry: Mycotoxins further erode the protective mucus layer. With the “gates” (tight junctions) open and the “walls” (mucus) gone, bacteria can freely enter the bloodstream.

Disease Susceptibility

Because the immune system is “blind” and the gut is “leaky,” the bird becomes a target for opportunistic infections.

- Secondary Bacterial Infections: Normal gut bacteria like *E. coli* and Salmonella transition from harmless to fatal, causing systemic septicemia.
- Viral Synergism: Small viral loads, such as Inclusion Body Hepatitis (IBH) that a healthy bird would normally survive, become highly fatal.
- Coccidiosis Flare-ups: Damaged gut linings are more easily colonized by Eimeria, making standard anti-coccidial programs less effective.

Vaccine Failure:

- Mycotoxins suppress B-cell and T-cell maturation, reducing vaccine effectiveness.
- Low immunoglobulin (IgG, IgA, IgM) production results in poor antibody titers.
- Memory immune cells fail to develop properly, causing weak long-term immunity.
- Common field outcomes:
    - Poor seroconversion
    - Breakthrough infections
    - Uneven flock protection
    - Failure of ND/IBD/IBV vaccination programs
- Maternal toxin exposure reduces immunity transfer to chicks, increasing early-age disease vulnerability.

POULTRY STRESS PATHWAYS

Endocrine & structural effects of Heat Stress and Mycotoxins in poultry.

1. PRIMARY STRESSOR	2. CELLULAR PATHWAY	3. RESULTING DAMAGE
HEAT STRESS High ambient heat ($>30^\circ\text{C}$) triggers thermoregulatory adaptation and circulatory shunting.	**MUCOSAL ISCHEMIA** Peripheral vasodilation diverts blood flow. Intestinal tissue starves of oxygen, spawning oxidative ROS.	**LEAKY GUT & CORTICOSTERONE SURGE** Junction failure breaches barrier. Adrenal axis triggers hormone-induced immune suppression.
MYCOTOXINS Fungal contaminants (Aflatoxin, DON) ingested via primary grain feed supplies.	**RIBOSOMAL INTERCEPT** Mycotoxins bind to peptidyl ribosomes, shutting down protein synthesis & mucosal cell repair cycles.	**BURSAL ATROPHY & ENZYME LOSS** Severe lymphoblast necrosis in the Bursa of Fabricius cuts antibody output & digestive efficiency.
COMBINED STATE Co-exposure compounds physical vulnerabilities, amplifying systemic organ stress.	**TOXIC SYNERGISM** Compromised intestinal lining accelerates mycotoxin absorption while ribosomal block halts barrier repair.	**VACCINE FAILURE & SEPSIS** Humoral antibodies fail to form protecting titers. Pathogens (*E. coli*) pass directly into blood channels.

EPITHELIAL BARRIER DEGRADATION ("LEAKY GUT")

How stress disrupts paracellular boundaries in the avian small intestine:

1. **ISCHEMIA & ROS ACCUMULATION**
   Shunted blood restricts oxygen supply, releasing superoxide radicals that attack lipid cell membranes.
2. **JUNCTION DISASSEMBLY**
   Occludin and Claudin proteins degrade, dissolving the physical locks between adjacent enterocytes and opening paracellular channels.

IMMUNE BREAKDOWN SUMMARY

The combined impact of high glucocorticoids and physical toxins disables standard defenses:

BURSAL ATROPHY
Toxins induce selective cell necrosis in the primary immune center (Bursa of Fabricius), blocking maturation of critical B-lymphocytes.

ADRENAL GLAND INTERFERENCE
Activated endocrine axis releases a constant Corticosterone surge, down-regulating global leukocyte activation rates.

Physiology Note: Unlike mammals, birds lack *17-alpha-hydroxylase* in the adrenal cortex, utilizing **Corticosterone** rather than Cortisol as their primary stress-axis marker.

NECROPSY MARKERS	LAB DIAGNOSTICS	REMEDIATION ACTIONS
• Watery, translucent gut content • Severe shrinking of Bursal folds	• Serum Corticosterone ELISA profiling • Histology of lymphoid follicles	• Active bentonite / mycotoxin binders • Hydro-electrolyte support (Betaine)

Figure 1: How mycotoxins and heat stress cause damage in poultry

Breeder Reproductive Dysfunction and Transgenerational Effects of Mycotoxins

In breeder operations, mycotoxins represent a catastrophic economic threat because they are vertically transmitted. Unlike commercial layers, where the loss is limited to the individual bird's production, breeder contamination compromises the viability of the entire next generation.

1. Impact on the Reproductive Systems (Male & Female)

Mycotoxins hit both sides of the fertility equation, often exacerbated by summer heat.

Female Reproductive System

- Mode of action: Mycotoxins (especially Zearalenone) mimic estrogen. This disrupts the hypothalamic-pituitary-ovarian axis.
- Impact: inflammation of the oviduct, cystic ovaries, and reduced synthesis of yolk precursors in the liver, resulting in a sharp drop in egg production and poor internal egg quality.

Male Fertility

- Mode of action: Toxins like T-2 and Aflatoxin induce oxidative stress that damages the phospholipid membrane of sperm cells.
- Impact: Under heat stress, rooster semen quality already declines; mycotoxins accelerate this by reducing sperm motility, concentration, and increasing morphological abnormalities. This leads to a massive spike in infertility rates.

2. Hatchability & Embryonic Mortality

For breeders, mycotoxins represent a "generational loss" via vertical transmission.

- Mode of action (the yolk bridge): Some mycotoxins are highly lipophilic. As the liver assembles the yolk, it deposits toxins directly into the egg.
- The "three-wave" mortality:
    1. Early (Days 1–7): Toxins interfere with mitosis (cell division), leading to early deaths often mistaken for "infertility."
    2. Mid-Term (Days 8–18): As the embryo begins intensive absorption of the toxic yolk, its developing liver and kidneys are compromised. This is the classic "Toxin Fingerprint."
    3. Late (Days 19–21): Ochratoxins impair the embryo's ability to mobilize calcium from the eggshell. As a result, the chick becomes too weak to pip and dies fully developed inside the shell ("dead-in-shell").

3. Chick Quality, Grading, and Settability

The "Chick Quality" starts in the breeder's gut and kidney health.

- Mode of action (nutrient malabsorption): Mycotoxins reduce pancreatic lipase and bile salts. This prevents the mother from absorbing fat-soluble vitamins (A, D, E, K) and pigments. They also lead to lower intestinal adsorption due to a reduced absorption area and lower transporter efficacy.

- Impact on chick quality:
    - "Pale Bird Syndrome": Chicks lack vital carotenoids for early-stage defense.
    - Skeletal weakness: Interference with Vitamin D3 metabolism results in weak legs and

"rubbery beaks" in Day-Old-Chicks (DOCs).
    - High first week mortality (FWM): Chicks hatch immunosuppressed, leading to high mortality during the first week.
- Impact on egg grading & settability: Mycotoxins (Ochratoxin) are nephrotoxic, damaging the kidneys and disrupting the blood calcium-carbonate balance. This leads to "Sandpaper" shells, misshapen eggs, and a 5–10% drop in the number of settable eggs fit for the incubator.

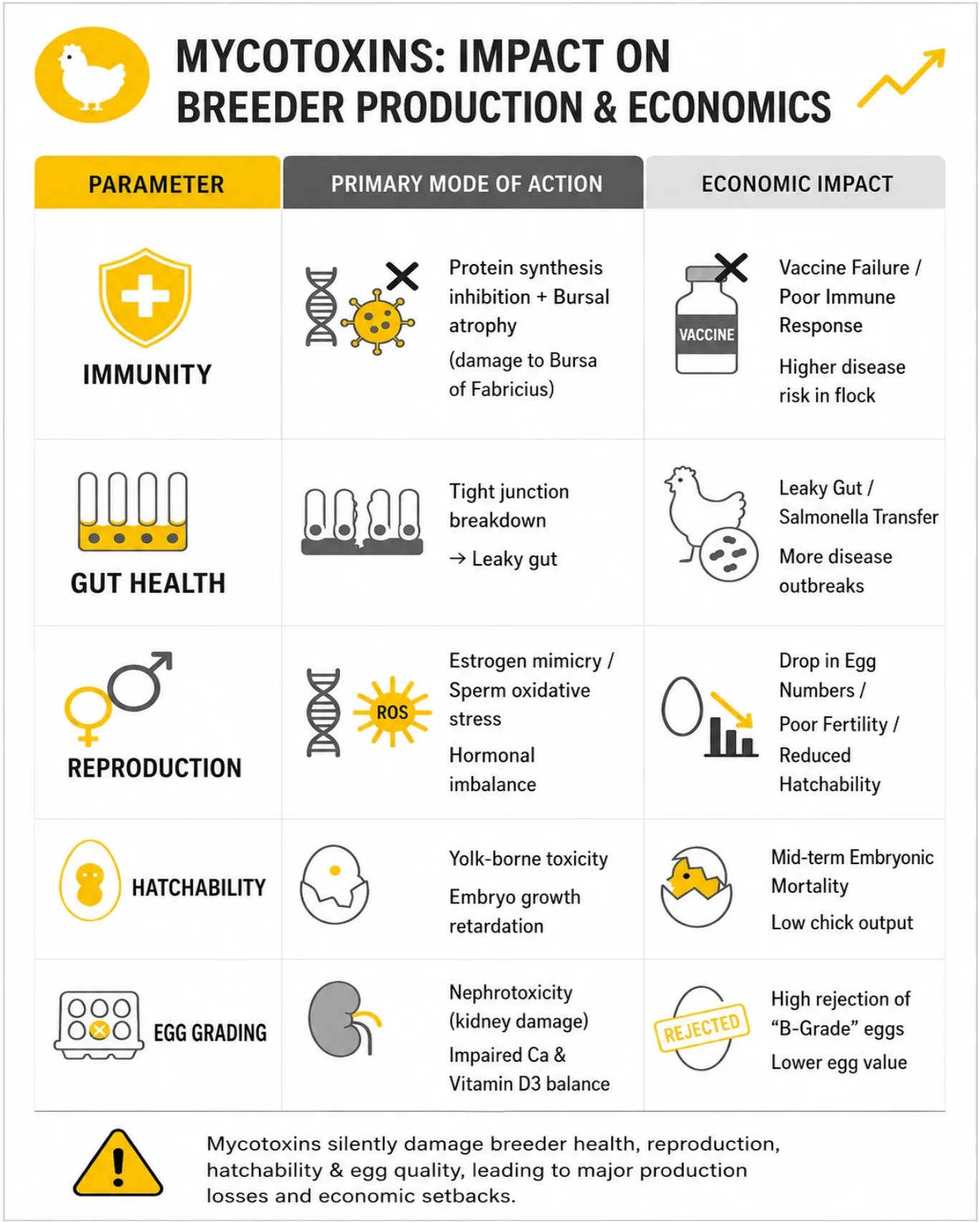

Figure 2: The impact of mycotoxins on breeder production and economics

Pro tip for breeders: In summers, high-moisture new maize triggers a mycotoxin surge that synergistically destroys the breeder's kidneys and shell gland, crippling egg settability. A 5% spike in "Dead-in-Shell" embryos during breakout analysis is a definitive indicator of feed toxicity rather than incubator failure.

Integrated Mycotoxin Mitigation Strategies for Poultry Production

To pursue an effective mycotoxin mitigation strategy, it is essential first to identify which mycotoxins are relevant to a given region before implementing measures.

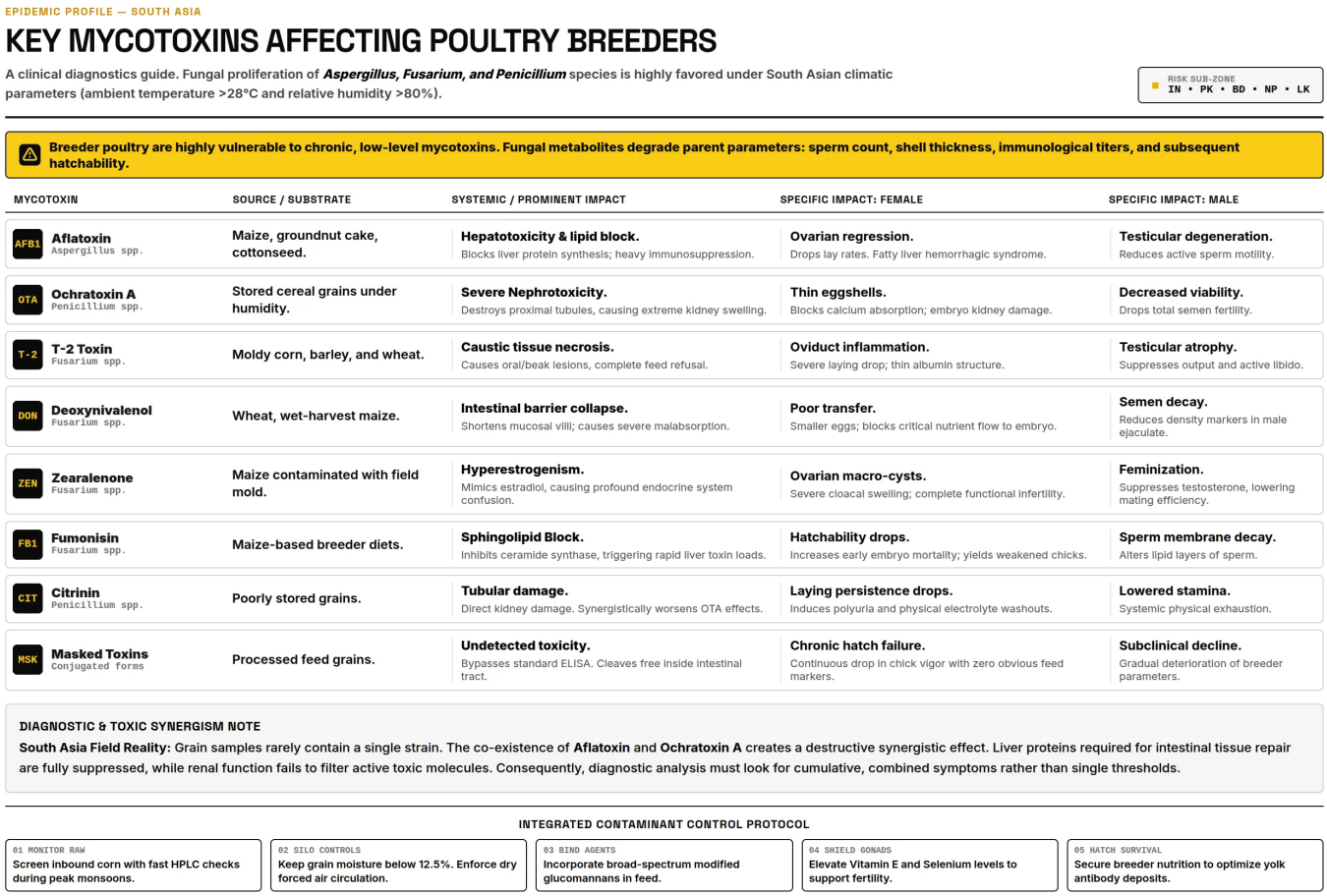

Figure 3: Key mycotoxins affecting poultry breeders in South Asia

1. Raw Material Management

- Strict moisture control: Reject any maize arriving with >14% moisture.
- Rapid screening: Perform rapid mycotoxin screening before unloading raw materials.
- Mechanical grain driers: To maintain a safe storage moisture level (<12%)

2. Feed Plant & Storage Hygiene

- First-In, First-Out (FIFO): Ensure strict inventory rotation to prevent "pockets" of old, moldy feed from contaminating new batches.
- Frequently clean silos and elevators: High temperature and humidity cause moisture condensation on silo walls, leading to localized mold growth.
- Antifungal Treatment: Use buffered organic acids (propionic and formic acid) to limit mold proliferation in feed.

3. Broad-Spectrum binders:

Bentonites (for Aflatoxins) and Yeast Cell Walls: These components help bind pathogenic bacteria like *E. coli* that capitalize on the “leaky gut” caused by toxins and heat stress.

4. Physiological & Gut Health Support

- Water acidification: Lower the drinking water pH to 4.5–5.5. This prevents bacterial blooms in the water lines when birds increase water intake by 3 times during heat stress.
- Liver & kidney tonics: Supplemental hepatic (milk thistle/silymarin) and renal support to help the bird metabolize and export toxins more efficiently.
- Metabolite supplementation: Use 25-hydroxyvitamin D3 in breeder diets to bypass the liver/kidney damage and ensure shell quality remains intact.
- Antioxidant boost: Increase levels of Vitamin E, C, and Selenium to counter the oxidative stress caused by the heat-toxin synergy.

5. Monitoring & Diagnostics

- Hatchery breakout analysis: Monitor “dead-in-shell” embryos. A spike in mid-term mortality is an immediate indicator that the breeder feed toxin binder needs a dosage increase.
- Frequent lab testing: Mycotoxin testing at least weekly during the new maize transition to identify the specific toxin profile.

Solutions are available to support toxin risk management

In the challenging climate where high-moisture “new maize” and summer humidity create a complex cocktail of mycotoxins, endotoxins, and pesticide residues, traditional, single-ingredient binders often fall short. Modern poultry production requires a proactive solution that does more than just “bind”; it must protect the bird’s internal integrity.

Solis Max – The effective myco- and endotoxin solution for sustained profitability

Solis Max is engineered to meet customers’ demand for an effective solution, offering a multi-pronged defense mechanism that targets the root causes of performance collapse. Solis Max uses a synergistic blend of five key components to ensure the flock’s safety.

Trials prove the effectiveness of Solis Max

Solis Max shows dose-dependent adsorbing capacity against multiple mycotoxins:

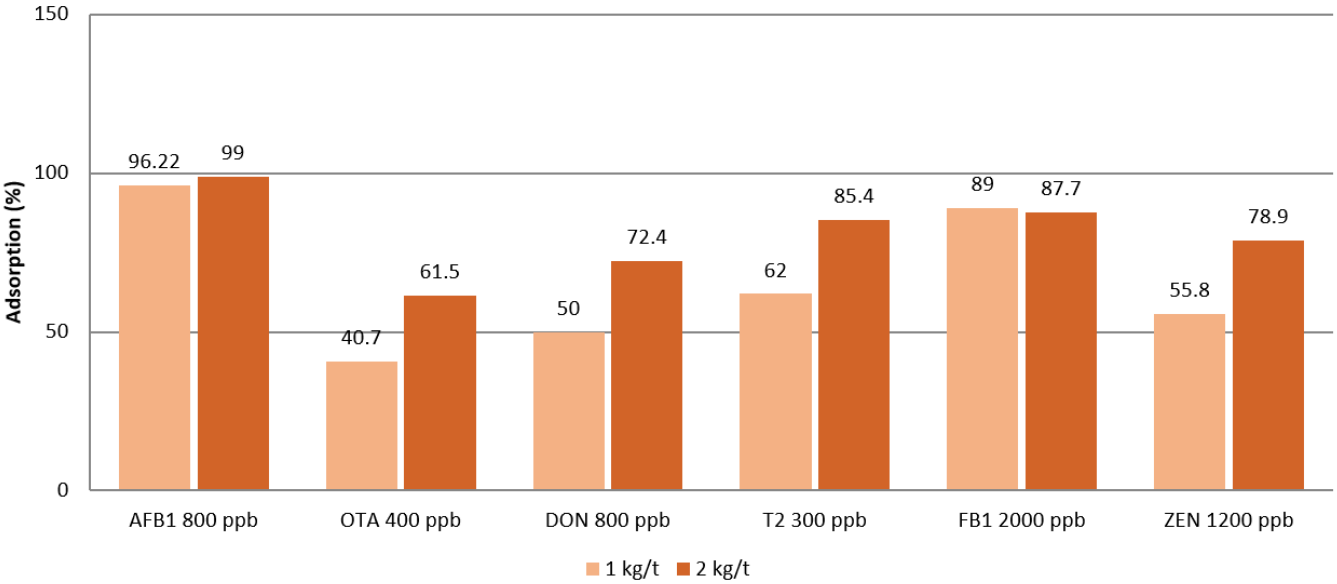

Figure 4: Mycotoxin Binding Capacity Of Solis Max

SOLIS MAX shows endotoxin adsorbing capacity – 1mg of SOLIS MAX absorbs 20 endotoxin units (EU) of E. coli endotoxin (80% adsorption rate):

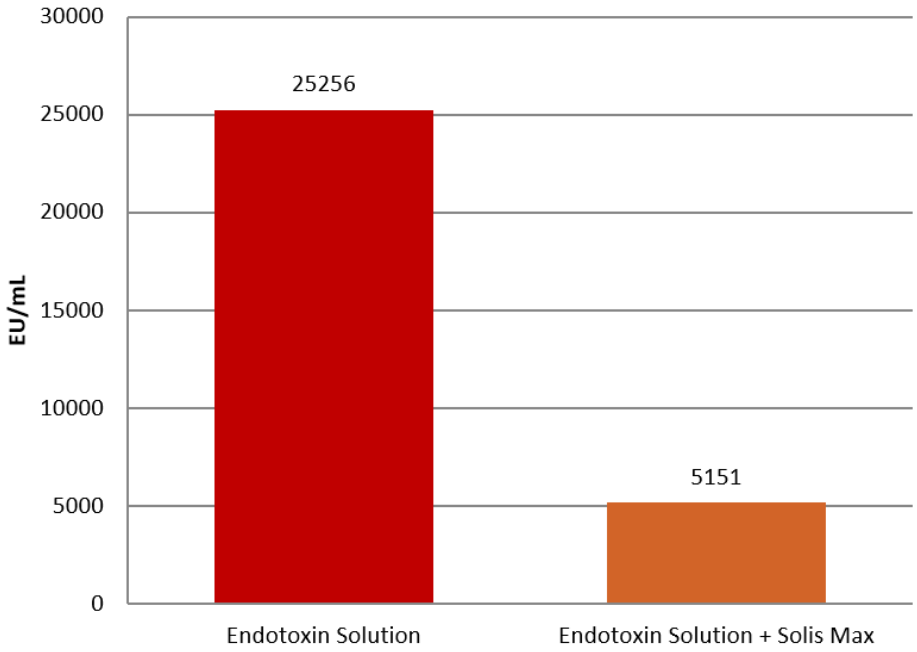

Figure 5: Endotoxin Binding Capacity Of Solis Max

Solis Max demonstrates high pesticide binding efficiency across multiple compounds:

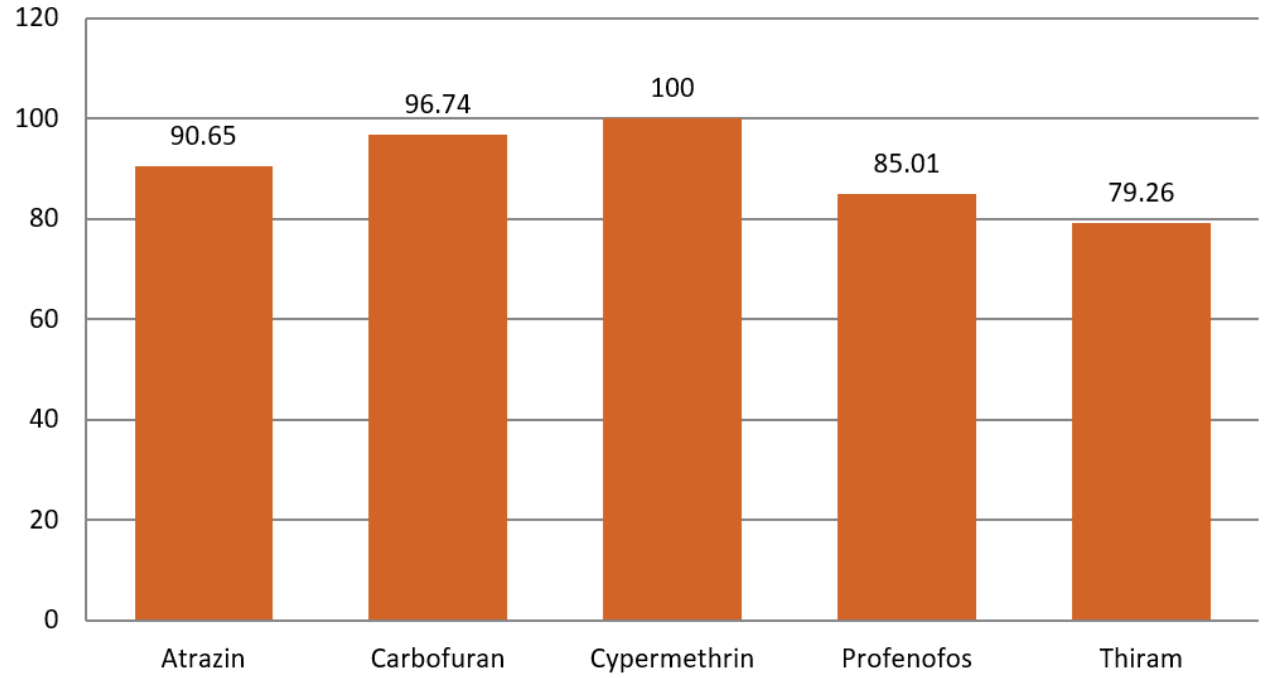

Figure 6: Pesticide Net Binding Capacity Of Solis Max (%)

Conclusion:

The convergence of a high Temperature–Humidity Index (THI) and mycotoxicosis represents a critical, multisystem challenge in poultry production, precipitating severe pathology across the hepatic, renal, and gastrointestinal systems. In breeding operations, this crisis exhibits a transgenerational impact: lipophilic mycotoxins are vertically transmitted to the yolk, inducing mid-term embryonic mortality and compromising post-hatch progeny immunity.

Mitigation demands stringent control of raw material moisture alongside advanced, broad-spectrum interventions. Utilizing an advanced multi-pronged solution like Solis Max counters this synergy by providing physicochemical adsorption and targeted organ protection. By neutralizing the concurrent threats of mycotoxins, endotoxins, and pesticides, it preserves cellular integrity, mitigates systemic pathology, and maintains optimal performance under extreme environmental stress.

References available upon request.

Managing heat stress in pigs in Asia

Conference Report

Heat stress poses a significant challenge to pig production, particularly in Asia, due to the region's warm and humid climate. In the following, Dr. Merideth Parke, Global Application Manager Swine at EW Nutrition, discusses effective management strategies to mitigate the adverse effects of heat stress on pig health and productivity.

Understanding Heat Stress

Pigs are particularly vulnerable to heat stress due to their limited ability to dissipate heat. "This is because they lack functional sweat glands, have relatively small lungs, a thick subcutaneous fat layer, and a narrow thermoneutral zone. The pigs' thermoneutral or 'comfort' zone varies by age and weight. For instance, sows require 18-22°C, grow-finish pigs less than 25°C, while newborn piglets need a much warmer 35°C," she explained.

Furthermore, today's lean and efficient pigs have higher metabolic demands and produce more body heat, making them more susceptible to heat stress than pigs from the 1980s.

Symptoms of heat stress include:

- Increased respiration rates (>50/minute)
- Elevated rectal temperature (>39.5 oC)
- Decreased feed intake
- Reduced growth rates
- Lower reproductive performance
- Lower reproductive performance

Pigs naturally reduce their feed intake as a response to heat stress, which is a mechanism to decrease metabolic heat production from digestion. For example, research on sows has shown that for each 10°C increase between 25-27°C at 50-60% relative humidity, they reduce their feed intake by 214 g/day.

Managing Heat Stress

Managing heat stress is complex. It requires a combination of solutions specific to each production system. Additionally, it must be considered that heat stress is not only about temperature. Its impact can be exacerbated by relative humidity, which hinders heat dissipation through evaporation. The heat index chart below demonstrates the relationship between temperature, humidity, and comfort levels for a grow-finish pig. Pigs require an environment where the heat index is within the thermoneutral zone, enabling them to shed heat and maintain efficient feed utilization and growth.

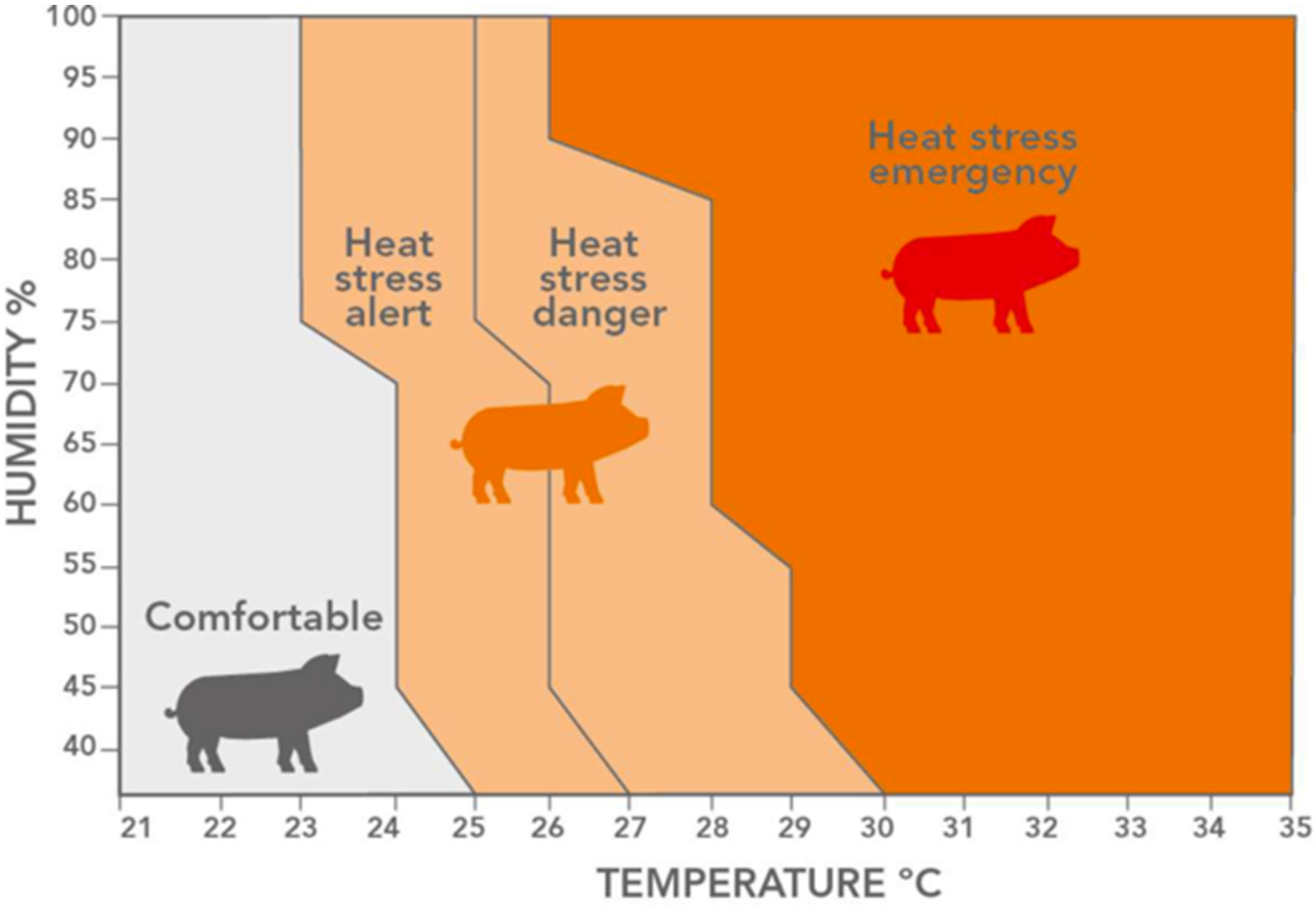

Figure 1: Heat stress index chart *(kepro.nl)*

While we often initially look to nutritional interventions, such as reducing dietary crude protein levels, increasing fats, or adding feed additives such as betaine, the effectiveness of these heat mitigation strategies is limited if the pigs are not eating well. Therefore, we must first focus on environmental management to reduce external heat absorption and increase heat load shedding. Pigs with the highest metabolic demands – lactating and gestating sows and finisher pigs – are especially susceptible to heat stress and should be given priority.

Several strategies to effectively manage heat stress can be used:

1. ## Misters and sprinklers

   Misters or sprinklers can help cool pigs through evaporation. However, these should be used strategically – running them for short periods followed by breaks – to maximize cooling effects without creating excessive moisture and wet conditions that could lead to other health issues, such as skin lesions or respiratory problems.

   However, water-based cooling systems can inadvertently raise the heat index in humid environments. When water is sprayed into a humid environment, it will further increase the moisture levels in the air, exacerbating the heat stress situation. If humidity is too high, alternative cooling methods, such as evaporative cooling pads or high-pressure fogging systems,

may be more effective.

Snout and flank drip systems deliver water directly onto the pig's body, mainly targeting areas more sensitive to heat. This localized approach enables heat dissipation without excessively increasing humidity in the surrounding environment.

2. Ventilation and airflow

Increased air movement, combined with misting or sprinkling (in low-humidity environments), can enhance the cooling effect by enhancing evaporative and convective heat loss. This combination helps reduce the temperature the pigs 'feel', making them more comfortable.

Producers should assess their ventilation systems and consider modifications to improve air circulation. This can be achieved by installing additional fans. However, the fans must be maintained – clean fan blades and louvers can increase efficiency by 30%. Furthermore, it must be evaluated if there are dead spots and drafts at the pig level, not along the walkways.

Using suspended ceilings can effectively reduce the airspace that needs cooling and can lead to lower energy costs for cooling systems.

3. Housing and surroundings

Adding insulation to roofs and walls can help reduce heat transfer inside the pig housing. Applying reflective coatings (such as white paint) to rooves and walls can help deflect solar radiation, reducing heat accumulation inside the shed by several degrees.

Dense vegetation surrounding a piggery can provide shade and reduce reflective heat. However, it can also obstruct airflow and trap moisture, increasing local humidity and exacerbating the pigs' heat index and heat stress.

4. Drinking water

Providing fresh, chilled drinking water (10°C) is a highly effective method for mitigating heat stress in pigs and increasing feed intake to improve overall performance. Insulating header tanks and water pipes can help to maintain cool temperatures.

Regular checks on water supply systems are essential to ensure they function correctly and provide adequate flow rates to the end of the line. For example, lactating sows need a flow rate of 4 L/minute.

5. Stocking density and body condition

Higher stocking densities can exacerbate heat stress in pigs. Increased animal density leads to higher ambient temperatures due to the combined metabolic heat produced by the animals and reduced airflow at the pig level. Lower stocking densities can allow pigs to manage their body temperature better.

Pigs with higher body condition scores (more body fat) may be more susceptible to heat stress. Excess fat can hinder effective heat dissipation, making it more difficult for these pigs to regulate their body temperature during hot weather.

6. Monitoring and evaluation

Continuous monitoring of temperature, humidity levels, and airflow is vital to adjust cooling strategies as necessary. A common mistake when monitoring the pigs' thermal environment is placing sensors in walkways at head height for workers because they are easier to read than at pig level in the pens. Sensors should be positioned in several locations throughout the shed. Regardless of sensor readings, stockpersons need to observe behavioral changes that provide immediate insights into the welfare and comfort of pigs during high-temperature periods.

7. ## Husbandry

   Pigs must be regularly observed for signs of heat stress, such as rapid breathing, reduced activity and feeding, lateral recumbency, and changes in vocalization. Aggressive behaviors may increase among pigs during heat stress as they compete for cooler spaces and water. Early detection of behavioral changes allows for timely interventions.

   “Schedule feeding during cooler parts of the day, such as early mornings or late evenings. This practice helps minimize additional heat production from digestion during peak temperatures”, according to Dr. Parke.

   “When moving pigs, especially pregnant sows, to the farrowing room, do so during the coolest times of the day and allow them to walk at their own pace.”

Conclusion

In conclusion, in the first run, each aspect of a production system must be critically evaluated, and existing housing or husbandry procedures must be modified to reduce the severity of the adverse effects of high temperatures on pig health and performance.

EW Nutrition’s Swine Academies took place in Ho Chi Minh City and Bangkok in October 2024. Dr. Merideth Parke, Global Application Manager, Swine, was one of the highly experienced speakers of EW Nutrition. She is a veterinarian who strongly focuses on swine health and preventive medicine.