Heat stress remediation with polyphenols from botanicals

By Juan Javierre | April 19, 2022

Broilers suffer more than mammals from heat stress due to their lack of sweat glands. (Courtesy Layn Natural Ingredients)

Functional botanical solutions help alleviate symptoms of heat stress

The heat of summer is upon us, and at a broader level, temperatures continue to climb globally. Heat stress is a multisystem syndrome associated with cycling high temperature that greatly affects production animals, including broilers, layers, pigs and ruminants.

While the precise manifestation of heat stress varies depending on severity or the affected species, the outcomes can be very serious, including reduced animal welfare and reduction of performance and economic losses.

A common approach of using reduced feed intake methods to help combat heat stress often results in less weight gain per unit of feed energy, reduced growth rate, impacts on reproductive efficiency, reduced egg and milk production, and worsening meat quality.

Natural, polyphenol-rich botanicals have been shown to provide deep, metabolic support for heat stress, while maintaining the health and productivity of animals.

Animal susceptibility

The root cause of heat stress is the inability of the animal to evacuate the internal heat created by metabolic processes. This may be due to failure of ventilation and cooling systems in the house, high environment temperature, lack of cooling systems in the animal body, or all these factors together. When heat is not removed, body temperature increases, electrolyte balance is disrupted, metabolism is altered, and productivity parameters worsen. Researchers have reported mortality in broilers over 20% in cases of severe heat stress in traditional poultry houses in the tropics.

Mammals can cope with heat stress better than poultry due to sweat gland function. When environment humidity is low, sweat evaporates, cooling the skin, and with it, the capillary blood, that in its turn cools the body. Broilers lack sweat glands and can only evacuate heat by panting, or radiation out of the un-feathered sections of the skin: shanks, crest and wattles. Panting causes loss of water and CO_2, which results in respiratory alkalosis with increase of blood pH; feed intake decreases and water intake increases up to seven times, resulting in higher urine flow and moist litter.

The primary effects of heat stress have been traditionally associated with hormonal and electrolyte imbalances. More recently, other metabolic systems have been implicated in its pathogeny: metabolic oxidation, and alterations of gut permeability and barrier status.

Chemical changes

Superoxide is a reactive oxygen species (ROS) byproduct of the energetic transduction at mitochondrial level, where up to 4% of all oxygen consumed ends as superoxide due to electron leak. Superoxide dismutase (SOD) in the mitochondrial matrix and intermembranes transform superoxide into hydrogen peroxide, also an ROS.

Hydrogen peroxide can be reduced to the very active hydroxyl radical in presence of copper and iron ions. Hydroxyl radical can diffuse into cytosol via membranes, exerting deleterious effects far from the originating site.

Catalase (CAT) and glutathione complex (GSH-Px) enzymes are the antioxidant systems the body uses to avoid hydroxyl production by splitting hydrogen peroxide down into oxygen and water.

Another built-in mechanism to limit superoxide production in physiological conditions is the activity of membrane uncoupling proteins (UCP), which create a proton backflow that reduces local oxygen concentration at complex level, and limits ROS production.

Heat stress mechanisms

Heat stress pathogenicity relies on three mechanisms: the increase of cell energy demand, the reduction of uncoupling proteins, and the alteration of intestine barrier function.

The increased energy generation to cover the cell demand is always associated to higher ROS production. Additionally, heat reduces the amount of uncoupling proteins, allowing for the buildup of superoxide and hydrogen peroxide. And the increased oxidative status alters the intestinal tight junction integrity, leading to leaky gut syndrome.

The role of polyphenol-rich botanicals

Polyphenols contained in botanical extracts constitute a tool providing relief to animals reared under heat stress. Polyphenols are recognized as powerful metabolic antioxidants, and in this role they can contribute to the antioxidant defense needed when the oxidative stress in the animal caused by high temperatures rises.

Research shows that polyphenols contained in green tea extracts reduced malondialdehyde (MDA, a marker for cell lipid peroxidation), and increased antioxidant enzymes (CAT, SOD, GSH-Px) in the liver of heat stressed quails, and improved feed intake and growth rates previously diminished by heat stress. Feeding tea polyphenols to chickens, researchers have shown SOD, GSH-Px and CAT increase, and MDA and TBARS decrease in blood serum. Moreover, feeding tea polyphenols to heat-stressed chickens improved feed intake and overall performance.

Polyphenol-rich botanical extracts from grapes have also shown to be able to reduce the oxidative status of the chicken, as demonstrated by increasing the content of liver SOD and GSH-Px. Interestingly, heat stress may impair the reproductive capacity of male broilers, and reduce fertility and hatchability in breeders. These conditions were reversed feeding polyphenols from botanical extracts of grapes. At the same time, performance parameters also improved.

Other mechanisms by which polyphenol-rich botanical extracts alleviate the consequences of heat stress relate to heat shock protein (HSP) expression. HSP are biological markers for heat stress and functional protectors of cell proteins. Ginkgo extracts reduce the expression of HSP, but increase the nuclear translocation of such proteins in cells from heart, liver and intestine, reducing the severity of heat stress effects on critical systems. Impact on HSP during heat stress have been also described when feeding grape, ginger, tea, olive or *Polygonum* extracts to chickens.

Polyphenol-rich botanical extracts interact with intestinal cells during heat stress, improving the barrier function, avoiding microbial translocation, and preventing the appearance of immunitary or inflammatory reactions within the lamina propria. Research exists reporting improvement of barrier function, gut morphometry, or tight junction proteins gene expression degraded by heat stress, and reverted by the feeding of pomegranate, tea, *Polygonum* or apple extracts.

Conclusion

Heat stress is a multi-factorial syndrome that involves many critical metabolic processes. Recent research reports the involvement of recently uncovered metabolic pathways and mechanisms disrupted by heat. Increasing the oxidative status of chickens has important repercussions on cell membrane integrity and metabolism, DNA replication and overall homeostasis. Oxidation in the intestinal cell causes alterations in tight junction protein synthesis, disrupting the barrier function, allowing microbial translocation, and creating the basis for inflammatory processes in the lamina propria. Polyphenol-rich botanical extracts can alleviate these symptoms due to their metabolic antioxidant properties and the protection of critical intestinal structures.

References available upon request.

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