Solutions to the negative impact of heat stress on poultry performance

Heat stress (HS) is one of the most important concerns in poultry production worldwide. Heat stress negatively impacts poultry performance, reducing growth, egg production and product safety. International Poultry Production recently discussed the issue with Muhammad Umar Sohail.

Muhammad carried out research at USDA, USA, and earned his PhD Degree from the University of Veterinary and Animal Sciences, Lahore, Pakistan. He is now working as Assistant Professor of Physiology at Government College University, Faisalabad, Pakistan.

Q: Heat stress is a huge concern for so many countries. Heat stress causes an estimated annual economic loss of $125-165 million in the US poultry industry alone. What can you say about heat stress in Asia?

A: Asia is the biggest producer of poultry meat (more than 30% of the total world production) and eggs. In recent years, the percentage rise in poultry meat and eggs has been higher in developing countries compared to the developed countries.

According to the OECD, this increase will be even greater over the coming years: for instance in India, Pakistan and Bangladesh, poultry meat production will rise by more than 50% between 2015 and 2023. As you know, Asia has a tropical or sub-tropical hot humid climate and modern poultry is generally not well adapted to this climate because the parent lines are mainly developed in Europe and America. We do not have exact statistics regarding economic losses due to heat stress, but it is generally believed that the consequences of HS are more severe in Asian countries.

Q: Heat stress is a common concern for most poultry producers, but how does heat stress impact poultry production?

A: Poultry lack sweat glands and have feathers on their skin. Both these factors negatively affect poultry performance in hot humid climates. In our two published studies with broilers, we reported a 33% drop in broiler weight under chronic heat stress (Table 1) with a dramatic 4.5 points increase in the mortality rate, and a 22% decrease in weight gain in broilers subjected to cyclic heat stress. Similar findings have been reported in laying hens and male breeders.

Q: How is the reduction in metabolic rate during a period of heat stress explained?

A: A drop in metabolic rate is part of physiological adaptation in response to a temperature rise in the environment. Several neuroendocrine mechanisms are activated in chickens in response to heat stress. The basal metabolic rate is determined by plasma T3 (triiodothyronine) in birds. A drop in T3 level, as reported in Sohail et al. (2010), would reduce metabolic heat production to alleviate heat stress.

Likewise, changes in reproductive hormone secretions represent the final sequence in the neuroendocrine pathway leading to the diminished reproductive performance associated with stress.

Rozenboim et al. (2007) reported a reduction in egg production, ovarian weight, plasma progesterone, testosterone and 17 ß-estradiol in laying hens.

Q: Is there any difference in adaptation between chicks submitted to heat stress from the beginning of their lives and poultry submitted to a sudden heat stress wave?

A: It is known that poultry used to high temperatures from day 0 will have better resistance to cope with sudden heat stress compared to others that are not used to high temperatures. This phenomenon is known as acclimatisation.

Moraes et al. (2003), reported that thermal conditioning during embryonic development may possibly induce epigenetic heat adaptation.

Similarly, Lin et al. (2005) reported that the birds' thermotolerance can be enhanced by short run early heat conditioning or feed restriction.

In temperate areas such as Europe and North America, where heat stress waves appear suddenly, consequences are more severe and have more negative impacts on flock performance than in hot areas.

Q: Is there any evidence of an increase in bacterial loads in the gut during a heat stress period?

A: Quite a few studies have reported changes in intestinal microbial ecology under heat stress. It is generally observed that heat stress decreases overall bacterial diversity present in the chicken's gut.

Continued on page 13

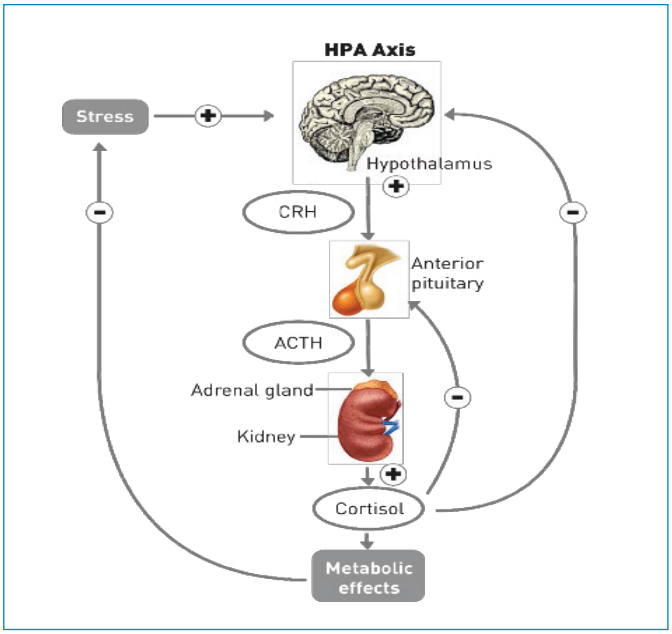

Fig. 1. The endocrine system: an overview (Hiller-Sturmhöfel and Bartke. Alcohol Health Res World: 1998, 22(3);153-64).

Table 1. Effect of heat stress on broilers (Sohail et al).

2012	Days	Control	Heat stress
Body weight	21	825.8^{a} ± 8.77	698.4^{b} ± 6.33
	42	2411.3^{a} ± 30.66	1626.3^{b} ± 143.89
Feed conversion ratio	21	1.31 ± 0.04	1.30 ± 0.02
	42	1.33^{a} ± 0.05	1.67^{b} ± 0.10
Mortality rate (%)	21	4.44	1.11
	42	5.6	10.0
2013	**Days**	**Control**	**Heat stress**
Body weight	21	577.7	564.8
	42	1924.7^{a}	1510.7^{b}
Feed conversion ratio	21	1.71	1.78
	42	1.82^{a}	2.24^{b}

$^{a-c}$Means ± SE within a row lacking a common superscript differ (P<0.05)

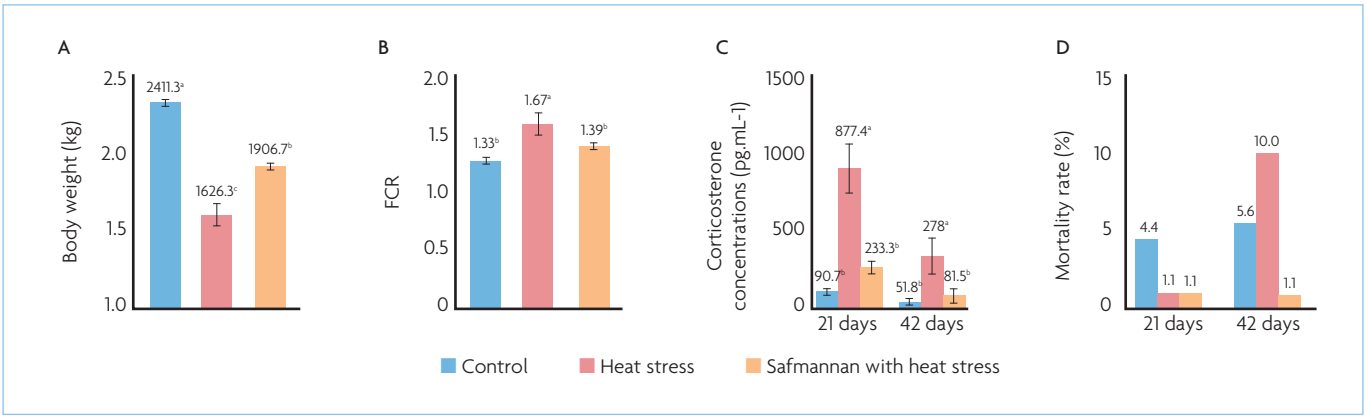

Fig. 1. A: body weight of broilers at 42 days, B: feed conversion rate of broilers at 42 days, C: corticosterone concentrations, D: mortality rate.

Continued from page 11

Songe et al. (2014) reported that intestinal Lactobacillus and Bifidobacterium counts reduced, whereas viable counts of coliforms and clostridium increased under heat stress.

Likewise, Sirsat et al. (2011) reported that the adhesion ability of Salmonella typhimurium with epithelial cells, as observed by pathogenic gene expression levels, increases under heat stress.

Q: What kind of nutritional solutions do we have to cope with heat stress in poultry?

A: Previously, much emphasis was put on vitamin and mineral supplementation during heat stress conditions to improve the bird's electrolyte balance.

Nowadays, research is more focused on balancing a bird's gut microflora under stressful conditions. Our understanding of the hypothalamic-pituitary-adrenal (HPA) axis has shown that a key role is played by microbes. They help to control several body functions via neuroendocrine pathways. As a result, more emphasis is placed on prebiotics and probiotics, either alone or in combination, to modify the gut microbial ecology.

Q: During your PhD, you ran studies on heat stress. What were their objectives?

A: I did two studies with broilers and two pilot studies with broiler breeders and laying hens. The objective of all the studies was very consistent: first we investigated the effect of heat stress on physiological and growth performance of the chickens, and secondly the effect of prebiotic and probiotic supplementations to cope with heat stress.

Q: You said you tested the effect of a yeast parietal fraction on birds subjected to heat stress, why did you make this choice? Among all the different yeast fractions, why did you select Safmannan?

A: Yeast parietal fractions contain mannans and ß-glucan. The literature reports that mannans have dual effects as a prebiotic.

They prevent binding of pathogens with the gut wall by offering their own lectins and expel pathogens out of the gut in the stool. Secondly, mannans, when fermented by microbes, produce short chain fatty acids (SCFA). SCFA have a trophic effect on microflora and immunity.

On the other hand, ß-glucans are known to modulate the immune system of birds, enhancing proliferation of macrophages and amplifying humoral and cell-mediated immune responses.

At that time, Safmannan was a recently launched prebiotic product. Its promised consistency, with at least 20% mannans and 20% beta-glucan, was interesting to test.

Q: What were the zootechnical/physiological results of these experiments?

A: We observed that compared with the heat stress control group, the supplementation of Safmannan improved body weight gain (+17%), feed conversion ratio (-28pts), and reduced mortality (-9pts). It also lowered corticosterone concentration in the heat stress group (Fig. 1).

Q: Could you explain the relationship between the increase in corticosterone levels and heat stress? What could be the mode of action of Safmannan on these parameters?

A: Corticosterone released during a state of stress is part of the body's defence or homeostasis system against stress. The release of corticosterone from the adrenal cortex is mediated by the hypothalamus and the pituitary gland. Under stressing conditions, the hypothalamus produces corticotrophin-releasing-hormone that will stimulate the pituitary gland that, in turn will produce adreno-corticotropic hormone (ACTH).

ACTH activates the adrenal glands to produce cortisol, a major endogenous glucocorticoïd displaying numerous metabolic effects. Moreover, there is now a huge amount of scientific evidence to demonstrate that cortisol is an important inhibitor of several immune functions.

Researchers are not yet able to describe the exact mechanisms involved in a prebiotic/Safmannan-induced corticosterone-lowering effect. It is assumed that the cross-talk between the microbes and the host gut brain axis modulates adrenal gland activity.

Q: You went deeper into your analysis as you studied the gut and tracheal microflora of birds subjected to heat stress. Why?

A: Heat stress changes the physiology of the respiratory tract by inducing panting and acidosis. These mechanisms may influence respiratory tract microbial ecology. Secondly, probiotics have been reported to positively affect respiratory tract infections and immunity. So these beliefs prompted me to investigate the effect of stress and supplementations on the tracheal ecology.

Q: What were your results?

A: We observed significant differences between microbial diversity, species richness between the two mucosal sites (trachea vs caecum) (Fig. 2.).

Q: Based on your experience, what are the most promising solutions to cope with heat stress?

A: The modern broiler industry is continuously expanding to hot climate developing countries where climatic control of broiler houses is lagging behind because of high installation and operational costs and unreliable electricity supplies.

Researchers are urged to explore alternative approaches to manage heat stress.

To my understanding, dietary management and genetics are the key factors for the control of heat stress. Therefore breeding for adaptation to heat should become a strategic goal.

It is generally believed that heat stress can be alleviated by using the naked-neck (Na) and the featherless gene (sc).

Good feeding practices can help to cope with heat stress. As an example, feeding in the early morning and late evening is recommended during the summer season to lower heat production by the birds during the day. ■

References are available from the author on request

Fig. 2. Percentage of tracheal microflora.