The Impact of Heat Stress on Intestinal Health in Broiler Chickens – Phyto-therapeutic Perspectives

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Abstract
One of the most significant challenges facing modern poultry farming is heat stress, a phenomenon that is becoming increasingly prevalent due to climate change. Exposure to high temperatures in broiler chickens disrupts intestinal homeostasis, leading to microbiological, structural, and functional changes that affect both growth performance and welfare. Oxidative stress induced by heat stress increases intestinal permeability and alters the morphology of intestinal villi, thereby compromising the integrity of the intestinal barrier and promoting the onset of an inflammatory response mediated by pro-inflammatory cytokines. A complementary strategy to mitigate the effects of high temperatures involves the use of phytogenic compounds, as they are credited with antioxidant, anti-inflammatory, antimicrobial, and immunomodulatory properties. Phyto-therapeutic interventions contribute to improving the antioxidant system, restoring intestinal morphology, and stabilizing the gut microbiota, with the ultimate effect being improved nutrient absorption. This paper aims to synthesize current information regarding the impact of heat stress on intestinal physiology and to highlight the role of phytotherapy as an effective strategy for maintaining intestinal health.

Keywords: broiler chickens, gut health phytogenic additives,

1. Introduction

In modern broiler chicken production systems, heat stress represents one of the greatest environmental challenges, resulting from an imbalance between internal heat production and the body's ability to dissipate it [1,2]. Thermal insulation (feather-covered body), the absence of sweat glands [3], and a high metabolic rate (due to genetic selection for accelerated growth) make broiler chickens extremely susceptible to heat stress [4,5]. Physiologically, the broiler chicken has a thermal comfort zone (thermoneutral zone) ranging from 18 – 24°C, within which homeothermy is maintained with minimal energy expenditure, without the activation of compensatory thermoregulatory mechanisms. When the ambient temperature exceeds 24 – 26°C, the birds begin to experience the first signs of heat stress, and at temperatures above 26 – 29°C, they are forced to resort to energy (intensive evaporative cooling mechanisms, such as increased respiratory rate, panting) [5,6]. In addition to panting, which is the primary method of evaporative cooling (respiratory rate can increase to 250 – 260 breaths/ min), broilers also exhibit other behavioural adaptation strategies, such as: reduced activity, lethargy [7]; raising/spreading the wings, a behaviour that thus promotes heat loss through convection by exposing areas with less feather coverage [8]; seeking cool areas [1]; decreased feed intake and increased water intake [9]; aggression, tonic immobility, and ruffling of feathers [6,8].

2. Materials and methods

This paper presents a narrative review of scientific literature on the evaluation of the effects of phytotherapy incorporated into the diet of broiler

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chickens subjected to heat stress. The scientific review aimed to identify medicinal plants used in the poultry industry to maintain digestive tract health under high ambient temperature conditions by consulting the PubMed, Web of Science, Google Scholar, and Scopus databases. Original articles and reviews published in English were included, which evaluated the effects of supplementing feed with phyto-therapeutic products, using search strategies combining the terms "broiler chicken," "heat stress," "gut health," "oxidative stress," "phytotherapy," and "poultry nutrition." Studies conducted on other species, studies in which broiler chickens were not subjected to experimental heat stress, and papers without full-text access were excluded. The data used were organized according to the plant product used, hybrid, administered dose, and observed effects.

3. Results and discussion

The Effects of Heat Stress on Broiler Chickens. In birds, behavioural thermoregulatory mechanisms are effective against excessive heat only when exposure is short-term; in the case of prolonged exposure to high temperatures, homeostasis can no longer be maintained through such mechanisms, leading to heat stress, characterized by metabolic imbalances and the activation of complex mechanisms, such as:

- the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-medullary adrenal axis - leading to the secretion of corticosterone (a stress marker with a potent immunosuppressive effect) and catecholamines (norepinephrine, epinephrine), which mobilize energy reserves [1,6] and the thyroid axis, where levels of triiodothyronine (T3) and thyroxine (T4) decrease to reduce internal heat production and metabolic rate, a process that significantly slows growth [7,8];
- cardiovascular mechanisms - peripheral vasodilation, redistribution of blood flow from organs to the periphery [6];
- oxidative mechanisms - which disrupt redox homeostasis due to an imbalance between the production of reactive oxygen species (ROS) and the antioxidant system's capacity to neutralize them [10,11];
- cellular damage - excess free radicals cause lipid peroxidation (increased malondialdehyde, MDA), loss of function in essential proteins (carbonylation) [5], and DNA oxidation [3];
- immunological mechanisms - lymphoid organs (spleen, thymus, bursa of Fabricius) undergo regression due to a decrease in the number of T and B lymphocytes, as well as a reduced capacity to generate an effective immune response [8].

The impact of heat stress on broiler chickens is profound and multidimensional, triggering a cascade of physiological, immunological, and morphological reactions that affect intestinal health and the microbiota, thereby impairing performance, systemic health, and the well-being of the chicks. Leaky gut syndrome (impairment of the morphology and integrity of the intestinal barrier) is caused by severe ischemia and hypoxia in the gastrointestinal tract, due to the cardiovascular system redirecting a large volume of blood from internal organs to the periphery to dissipate heat [12], a phenomenon accompanied by the following effects:

- oxidative stress - is caused by the massive production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), which overwhelm the antioxidant system and damage epithelial cell membranes [13,14];
- morphological microarchitectural changes - are due to a decrease in the height of intestinal villi and an increase in crypt depth, leading to a reduction in the villus - to - crypt ratio (VH/CD). A decrease in this ratio results in villous atrophy (reduction in the nutrient absorption surface area), oedema in the lamina propria (as a result of increased intestinal permeability and disruption of tissue homeostasis), crypt hyperplasia, and a shortened lifespan of mature enterocytes [15,16];
- increased paracellular permeability - inflammation and oxidative stress destabilize the proteins in tight junctions (occludin and claudin) and cause contraction of the actomyosin ring, thereby increasing intestinal permeability and allowing endotoxins and pathogens to translocate from the intestinal lumen into the lamina propria and into the portal and systemic blood circulation [6].

The onset of intestinal dysbiosis (disruption of the gut microbiota due to an imbalance between beneficial and potentially pathogenic microorganisms) leads to impaired digestive function, altered immune response, and increased susceptibility to infections and systemic inflammation, through:

- a reduction in protective commensal bacterial populations, such as *Lactobacillus*, *Bifidobacterium*, *Faecalibacterium*, *Bacteroides*, and species from the *Ruminococcaceae* family - essential bacteria that produce short-chain fatty acids (SCFAs) such as butyrate and acetate, which serve as energy sources for epithelial cells and play a fundamental role in maintaining mucosal integrity and preventing inflammation [13];
- proliferation of opportunistic and pathogenic bacteria – under conditions of thermal stress, an expansion of facultative anaerobic bacteria and certain potentially pathogenic microorganisms has been observed, such as *Salmonella*, *Shigella*, *Clostridium*, *Escherichia coli*, a change due to alterations in oxygen levels within the intestinal lumen (caused by mucosal lesions), which favours the proliferation of strictly anaerobic bacteria, particularly those in the phylum *Proteobacteria*, which are better adapted to environments with increased oxygen availability [17].

Investigations conducted using the pair-feeding technique by Nano - Hara and colleagues (2020) revealed that these morphological lesions as well as the increase in intestinal permeability are not merely a consequence of reduced feed intake exhibited by birds exposed to high temperatures, but direct effects of heat stress [18].

Table 1. The Effects of certain herbal remedies used in poultry farming

Plant	Broiler/ HS (◦C)	Dose	Effect	Source
Macleaya cordata	Ross 308 (33 – 38°C)	60 / 100 mg/kg	- improves body weight; - improves resting heart rate; - reduces inflammation (↓TNF-α, NF-κB); - ↓ corticosterone levels; - strengthens tight junctions	[23]
Macleaya cordata (extract)	Yellow feathered (35 ± 2°C)	1000 mg/kg	- improves body weight (BW), daily weight gain (ADG), and feed conversion ratio (FCR); - modulates the cecal flora; - ↓ liver toxicity (AST, ALT, LDH).	[24]
Steam exploded pine particles (PSPP)	Indian River (31°C – 6h/day)	0.5% / 1% (diet)	- ↓rectal temperature; - ↓expression of HSP-70 and HSP-90 genes; - strengthens intestinal integrity (↑ expression of MUC2 and ZO1).	[25]
Lippia origanoides (EO) *+ betainǎ (extract)*	Cobb 500 (35°C)	45 ppm EO + 150 ppm betaine	- ↑ BW, ADG; - improved intestinal morphology (↑ VH); - ↑ bone mineralization; - ↓ intestinal permeability; - ↓ chest myopathies.	[26]
Origanum vulgaris + *Salvia rosmarinus*	Broiler (31.9°C)	50 / 100 mg/kg / 50 + 50 mg/kg / 100 +100 mg/kg	50 mg oregano extract: - significantly improved growth, feed utilization, and amylase/protease activity; - positively regulates tight junction genes (CLDN1, ZO-1, ZO-2, MUC2); - ↓ NOX4 and NOS2; - ↓ IgG, IgM.	[27]
Thymus vulgaris (extract)	Ross 308 (25-30°C) (starter)	2 kg/t	- improves body weight; - improves DWG (starter); - no effect: economic performance, intestinal morphology, nutrient content in feces.	[28]
Thymus vulgaris (powder)	Arbor Acres (33,6°C)	10 – 30 g/kg	- improved FCR; - ↑ gizzard weight; - increased IBD antibody titer; - ↓MDA.	[29]
Thymus vulgaris (oil)	Arbor Acres (35°C)	1.0 / 1.5 /2.0 g/kg	- dose 1.0: improved FCR; - doses 1.5/2.0: ↑ total plasma protein; - all doses: ↓ reduced liver enzymes (AST).	[30]
Thymus vulgaris	Ross 308	0.5% /	- thyme: did not significantly affect performance;	[31]

Plant	Broiler/ HS (◦C)	Dose	Effect	Source
+ lemon juice	(34 °C)	1% thyme+ 2ml/L lemon juice (water)	- mix: ↑ feed intake; ↑ weight gain; ↓ H/L stress ratio; ↓ triglyceride concentration.	
Thymus vulgaris + *Mentha pulegium* (powder)	Ross 308 (32°C)	0.5% thyme/ 0.5% mint/ 0.5% mix	- thyme/mint: ↑moisture; ↑meat brightness. - mix: ↑pH; ↓MDA.	[32]
Thymus vulgaris + Vitamins (A, C, E) (oil)	Ross 308 (34°C)	300 mg/kg thyme oil + vitamins	- thyme oil + vitamins A and C mixture: no significant effects - thyme oil + vitamin E mixture: partial reduction of the effects caused by heat stress.	[33]
Camellia sinensis	Arbor Acres (35°C)	300 / 600 mg/kg (EGCG)	- ↑ antioxidant enzyme activity; - ↓ MDA; - ↓ UA, CHOL, TG, LDH, CK, ASL; - ↑ BW, FI; - ↑ activity TP, GLU, ALP.	[34]
Punica granatum	Ross (37°C)	4 % - 10% (pulp)	Doses >7%: - ↓ cholesterol/LDL; - ↓ MDA.	[35]
Spirulina platensis	Ross 308 (35 ± 1°C)	1 g/kg	- mitigates the effects of HS on the EPEF index; - improve survival rates; - ↑ *Lactobacillus spp*; - ↓ *E coli* population; - ↑ weight of lymphoid organs.	[36]
Spirulina platensis	Cobb 500 (32 - 33°C)	3%	- ↑FBW; - ↑ GPx; - ↑ IL4; - ↑ CLDN2.	[37]
Spirulina platensis	Cobb 500 (34 ± 1°C)	0.5% / 1% / 1.5%	- improve ADG, BW, and FCR; - ↑ hemoglobin and hematocrit levels; - ↓ cholesterol and LDL levels; - ↑ HDL levels; - ↑ SOD and GPx; - ↓ MDA.	[38]
Allium sativum (powder)	Ross 308 (35 ± 1°C)	200 mg/kg	- improvement in intestinal morphology; - improvement in survival rate; - ↑ in the number of lactobacilli; - ↑ in coliforms.	[36]

[1]ADG – average daily gain; [2]ALT - alanine aminotransferase; [3]ALP- alkaline phosphatase; [4]AST - aspartate aminotransferase; [5]ATP - adenosine triphosphate; [6]BW - body weight; [7]BWG - body weight gain; [8]CAT - catalase; [9]CHOL – cholesterol; [10]CK - creatin kinase; [11]CKMB - creatine kinase; MB; [12]CLDN - claudins; [13]DWG – daily weight gains; [14]EO - essential oil; [15]EGCG – epigallocatechin gallate; [16]EPEF – European production efficiency factor; [17]FCR - feed conversion ratio; [18]FI - feed intake; [19]GLU – glucose; [20]GPx - glutathione peroxidase; [21]H/L - heterophil/lymphocyte ratio; [22]HSP - heat shock protein; [23]IBD- infectious bursal disease; [24]Ig - immunoglobulin; [25]IL - interleukin; [26]LDL - low density lipoprotein; [27]LDH – lactate dehydrogenase; [28]MDA - malondialdehyde; [29]MUC-2 - mucin; [30]NF-kB - nuclear factor kappa-B; [31]NOX4 – NADPH oxidase 4; [32]NOS2 – inducible nitric oxide synthase; [33]SOD - superoxide dismutase; [34]T3 - triiodothyronine; [35]TAC - total antioxidant capacity; [36]TG – triglycerides; [37]TNF - α- tumour necrosis factor alpha; [38]UA – uric acid; [39]VH – villus hight; [40]ZO – zonula occludens.

Another important response is impaired immune function, the onset of local inflammation, and the development of malabsorption [19]. The activation of toll - like receptors (TLR4), due to

the translocation of endotoxins (LPS), triggers an NF-κB intracellular signalling cascade, thereby releasing major pro-inflammatory cytokines TNF-α, IL-1β, IL-6, creating a vicious cycle by exacerbating inflammation, which in turn leads to the deterioration of tight junctions, thereby worsening intestinal permeability [13]. Systemic immunosuppression is associated with a sudden increase in corticosterone levels, a hormonal release driven by the hypothalamic - pituitary - adrenal axis, which has a potent immunosuppressive effect. This effect is due to a reduction in the phagocytic capacity of macrophages, causing atrophy of lymphoid tissues, thereby decreasing immunoglobulin A (IgA) secretion, which compromises the protection of the intestinal mucosa [6,20]. Due to the decrease in the absorption surface area (reduction in the height of intestinal villi) and changes in the microbiota, the activity of digestive enzymes (amylase, lipase) and nutrient transporters, sodium - glucose linked transporter 1 and glucose transporter 2 (SGLT- 1, GLUT- 2) is reduced [6].

Wang et al. (2022) found that extracts of *Macleaya cordata* are rich in isoquinolone alkaloids, particularly sanguinarine and chelerythrine [24], while Khongthong and colleagues (2023) demonstrated that such extracts are capable of regulating the expression of genes encoding tight junction proteins, such as occludin, claudin-1, and mucin-2, directly in the jejunum, thereby limiting intestinal permeability during periods of heat stress [23].

The study conducted by Goel et al. (2025) demonstrated that pine (steam-processed pine wood particles, PSPP) could protect the mucus layer lining the intestine, maintaining the structure of the villi and blocking the adhesion of pathogens [4]. Plants such as oregano (*Origanum vulgare*) and thyme (*Thymus vulgaris*) exhibit remarkable efficacy due to their essential oils, rich in thymol, carvacrol, and monoterpene phenols, which can alter the cell walls of Gram-negative bacteria, strongly inhibiting the proliferation of pathogens such as *Escherichia coli* and *Salmonella*. *Clostridium perfringens* (responsible for necrotic enteritis) [39]. Green tea *(Camellia sinensis)* and pomegranate (*Punica granatum*) are major sources of catechins and tannins, capable of restoring the balance of the intestinal flora by promoting beneficial bacterial families [34,35]. The microalgae *Spirulina platensis* and *Chlorella vulgaris* (often used in conjunction with phytosupplements) are rich in astaxanthin, phycocyanin, lutein, and beta-carotene, which directly support intestinal integrity by increasing the proportion of beneficial bacteria that secrete short-chain fatty acids from the genera *Ruminococcus*, *Oscillospira,* and *Lactobacillus* [40].

4. Conclusions

High ambient temperatures represent one of the most significant challenges facing modern poultry farming, as they pose a risk to the integrity of the gastrointestinal tract and have a direct impact on the welfare, health, and production performance of broiler chickens. Genetic selection focused on accelerated growth rates and superior feed conversion, as well as limited thermoregulatory capacity, combined with intensifying climate phenomena, has increased birds' susceptibility to high ambient temperatures. In this context, heat stress should not be viewed as a factor of environmental discomfort but as a complex pathogenic agent capable of inducing systemic disturbances with major digestive manifestations; the gastrointestinal tract, particularly the small intestine, stands out as a target organ of heat stress.

Oxidative stress plays a central role in the pathogenesis of intestinal lesions, driven by increased production of reactive oxygen species alongside the depletion of endogenous antioxidant systems, thereby causing membrane lipid peroxidation, deterioration of structural proteins, and cellular dysfunction in enterocytes, compromising the integrity of the intestinal epithelium and facilitating the passage of endotoxins into the circulation due to increased intestinal barrier permeability; simultaneously, intestinal dysbiosis, characterized by a reduction in beneficial bacterial populations and the proliferation of pathogenic ones, amplifies local inflammation and reduces digestive efficiency.

In this context, phytotherapy emerges as a promising nutritional intervention, as it aligns with the global trend toward reducing synthetic additives and growth-promoting antibiotics.

According to the scientific literature, numerous medicinal plants, extracts, and essential oils possess anti-inflammatory, antimicrobial, and

immunomodulatory properties, and their inclusion in poultry diets (especially oregano, thyme, green tea, garlic, mint, rosemary, or seaweed) contributes to improving intestinal mucosal integrity and maintaining normal villus morphology, ultimately improving the overall physiological status of birds subjected to heat stress. Specialized studies have shown that the mechanisms by which phytogenic additives support the body's resilience (reduction of corticosterone, improved digestion through increased enzymatic secretion, modulation of the immune response, and stimulation of digestion) translate into better weight gains, reduced mortality rates, and optimized feed conversion.

Given the high variability in the composition of plant-based raw materials (which is influenced by soil and climate conditions, origin, harvest time, and processing technology), future research should focus on identifying optimal doses, microencapsulation methods, and synergistic combinations of extracts to optimize the stability and bioavailability of active compounds.

Looking ahead, integrating phytotherapy into a holistic heat stress management system, by combining nutritional measures with the optimization of the microclimate in housing, may represent a new strategic direction that significantly enhances the broiler chicken's ability to adapt to unfavorable environmental conditions.

In conclusion, phytotherapy represents an effective and sustainable alternative for mitigating the effects of heat stress on the intestinal integrity of broiler chickens, but requires the development of experimentally validated, standardized phytotherapeutic formulations adapted to modern rearing systems, in the context of future climate challenges.

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