Effects of Heat Stress on Intestinal Mucosal Structure in Poultry and Possible Causes: Post-print

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Abstract

Heat stress is one of the critical factors affecting poultry growth and health. The intestine serves not only as the site for digestion and nutrient absorption in poultry, but also as the first line of defense against external pathogenic microorganisms. Heat stress impacts the intestinal structure of poultry, disrupts intestinal microbial balance, affects intestinal immune function, compromises intestinal mucosal integrity, increases the likelihood of bacterial translocation, and ultimately influences poultry growth and health. This article summarizes research findings regarding the effects of heat stress on poultry intestinal structure, and preliminarily proposes potential mechanisms underlying these effects, thereby providing a basis for further elucidating the impact of heat stress on poultry intestinal health.

Full Text

Title and Authors

Effects of Heat Stress on Poultry Intestinal Mucosal Structure and Its Possible Mechanisms

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Abstract

Heat stress is a critical factor affecting poultry growth and health. The intestinal tract serves not only as the site for nutrient digestion and absorption but also as the first line of defense against pathogenic microorganisms. Heat stress impairs

intestinal structure, disrupts microbial balance, compromises immune function, damages mucosal integrity, increases bacterial translocation, and ultimately affects poultry growth and health. This paper reviews research findings on heat stress effects on poultry intestinal structure and proposes potential mechanisms to further elucidate its impact on intestinal health.

Key words: Heat stress; Poultry; Intestinal structure; Mucosal integrity; Intestinal microflora

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Introduction

The intestinal tract of poultry comprises the duodenum, jejunum, ileum, cecum, and rectum, serving as the primary site for nutrient digestion and absorption. Although accounting for only approximately 1.5% of body weight, the intestine is about six times the body length. Circular folds and intestinal villi expand the small intestinal surface area by 20-30 fold, effectively enhancing absorptive function [1]. The intestine also represents a major energy-consuming tissue, utilizing 6.0%-8.0% of dietary energy [2]. Furthermore, the poultry intestine harbors numerous microorganisms, including many pathogenic microbes entering through feed and water, making the intestinal epithelium a critical barrier against pathogens [3]. Maintaining normal intestinal structure and function is essential for poultry growth and health.

Heat stress affects poultry health and productive performance, significantly reducing broiler growth performance [4] and decreasing avian uncoupling protein mRNA expression [5]. It also impairs immune organ development, damages small intestinal morphology [6], and alters cecal microflora diversity [7]. Heat stress represents a significant factor influencing poultry intestinal structure and function, affecting both morphology and mucosal integrity through multiple mechanisms such as reduced feed intake and decreased intestinal blood flow. This paper summarizes research on heat stress effects on poultry intestinal structure and explores underlying mechanisms to enhance understanding of its impact on intestinal health.

Effects on Intestinal Structure and Function

1.1 Intestinal Morphology

Ambient high temperature affects intestinal villus height in poultry. Deng et al. [8] found that 12 days of heat stress at 34°C shortened villus height in the ileum and cecum of laying hens. Song et al. [9] observed that exposing broilers to 33°C for 10 hours daily over 20 days reduced jejunal villus height. Zhang et al. [6] from our research group demonstrated that 14 days of heat stress at 31°C significantly decreased villus height in both jejunum and ileum. Similar findings have been reported in broilers [10-11] and pigs [12-14]. However, Quinteriro-Filho et al. [15] reported that 10 hours of heat stress at 31°C did

not significantly affect jejunal villus height in broilers. Burkholder et al. [16] found that 24 hours of heat stress at 30°C had no significant effect on villus height in the ileum and cecum. These discrepancies may be attributed to lower environmental temperatures or shorter stress durations. Hao et al. [17] observed that 5 hours of heat stress at 36°C did not significantly affect jejunal villus height, but the villus height-to-crypt depth ratio decreased significantly after 10 hours. Additionally, different intestinal segments may exhibit varying sensitivity to high temperature, as 30°C for 14 days significantly reduced villus height in the distal jejunum but not in the proximal segment [18].

Reports on the effects of high temperature on intestinal crypt depth have been inconsistent. Song et al. [9] observed increased jejunal crypt depth in broilers, while Quinteriro-Filho et al. [15] found no significant effect. Conversely, Burkholder et al. [16] and Al-Fataftah et al. [10] reported decreased crypt depth in broilers. Similar results have been observed in pigs [12-13]. These differential effects may be related to stress intensity and duration, as Pearce et al. [14] demonstrated that crypt depth initially increased then gradually decreased with prolonged heat stress in pigs.

Villus height is jointly regulated by apoptosis and shedding of mature epithelial cells, migration of immature epithelial cells, and proliferation of crypt stem cells. Reduced villus height may be associated with enhanced apoptosis of mature epithelial cells, as Yu et al. [13] observed ultrastructural damage to jejunal villus tips and epithelial cell shedding under high temperature. In vitro studies by Yu et al. [19] also found that elevated culture temperature increased apoptosis in IEC-6 intestinal epithelial cells. Crypt depth may be related to stem cell proliferative activity, which Yamauchi et al. [20] suggested may be more sensitive to high temperature. During initial heat stress, poultry may compensatorily enhance stem cell proliferation to restore epithelial cell loss at villus tips. As adaptation occurs, crypt depth may recover, but persistent heat stress may suppress stem cell proliferation due to reduced nutrient intake, leading to shallow crypts.

1.2 Mucosal Integrity

High temperature affects both villus height and crypt depth, impairing nutrient digestion and absorption [21] while compromising mucosal integrity and increasing susceptibility to pathogenic infection. Huang et al. [22] reported significantly elevated plasma endotoxin levels in broilers after 10 hours at 38°C, with similar findings in pigs [14] and mice [23-24]. Endotoxin entry stimulates immune responses, increasing blood cytokines such as IL-1 and TNF- [25-26]. Deng et al. [8] found significantly elevated plasma TNF- and IL-1 in laying hens at 34°C, with comparable results in humans [27].

Endotoxin, a lipopolysaccharide complex from Gram-negative bacteria (molecular mass ~1×10 -20×10), normally enters the body only in trace amounts through tight junctions or receptor-mediated transcytosis [26]. Therefore, ele-

vated blood endotoxin and cytokine levels indirectly indicate compromised intestinal integrity. Using Ussing chambers, Song et al. [9] directly demonstrated that 20 days at 33°C significantly increased jejunal mucosal permeability, manifested by decreased transepithelial electrical resistance (TER) and enhanced macromolecule permeability. Similar findings have been reported in mice and pigs [14,28-29]. In vitro studies showed elevated temperature damaged tight junctions between Caco-2 cells [30] and altered tight junction morphology in porcine jejunum [13], collectively demonstrating that high temperature reduces mucosal integrity.

Compromised integrity may involve mast cells, as Deng et al. [8] found increased mast cells in the intestinal epithelium of heat-stressed laying hens. Mast cell-derived tryptase and histamine enhance epithelial permeability [31-32]. High temperature also affects tight junction protein expression and distribution. Ikari et al. [33] reported suppressed ZO-1 expression and its translocation from membrane to cytoplasm. Dokladny et al. [30] similarly found decreased ZO-1 expression, but observed upregulated Occludin expression [34], speculating this represents a compensatory protective response. Pearce et al. [14] found heat stress affected mRNA expression of ZO-1, Occludin, and Claudin-3 in porcine intestine, showing initial decrease followed by increase, supporting this hypothesis. Additionally, high temperature elevates cytokines [8], affecting myosin light chain kinase expression [14] and causing myosin light chain phosphorylation. This regulates actin cytoskeleton contraction, opening tight junctions and increasing permeability [35-38], representing another mechanism of mucosal damage.

Impaired mucosal integrity increases pathogenic infection risk. Burkholder et al. [16] observed increased Salmonella attachment in broiler intestine after 24 hours at 30°C. Quinteiro-Filho et al. [39-40] found heat stress enhanced Salmonella colonization in the cecum and crop and promoted translocation to spleen, liver, and bone marrow, indicating reduced resistance to infection. Compromised epithelial integrity allows endotoxin entry into circulation, activating local and systemic immunity and diverting energy and nutrients from growth to acute-phase protein production, thereby inhibiting growth [41-42]. This may be a key mechanism by which high temperature affects poultry health.

Possible Mechanisms

2.1 Reduced Feed Intake

The effects of high temperature on intestinal structure may be partially mediated by reduced feed intake. Zhang et al. [43] found that 70% feed restriction significantly decreased duodenal villus height in broilers. However, Liu et al. [44] reported no significant effects on villus height or crypt depth in yellow-feathered broilers with the same restriction, possibly due to their slower growth rate and reduced sensitivity to short-term restriction. Yamauchi et al. [45] observed decreased duodenal and jejunal villus height in fasted laying hens. Nuñez et

al. [46] found 60% feed restriction significantly reduced small intestinal villus height and crypt depth in piglets. Ferraris et al. [47] concluded that feed restriction severely compromises mucosal structure and transport function, increases macromolecule permeability, and causes villus atrophy.

Garriga et al. [18] investigated high temperature effects on broiler intestinal structure while using pair-feeding to isolate reduced feed intake effects. The pair-fed group also showed reduced distal jejunal villus height. Pearce et al. [14] reported similar results in pigs, with pair-fed animals showing increased lipopolysaccharide permeability. These findings indicate that heat stress effects on intestinal structure are partially mediated by reduced feed intake.

2.2 Elevated Body Temperature

High temperature effects on intestinal structure may be related to elevated body temperature. In vitro studies show increased culture temperature inhibits IEC-6 epithelial cell proliferation, induces apoptosis [19], and affects Caco-2 tight junctions [27]. Heat stress increases rectal temperature in pigs [13] and rats [19], potentially affecting epithelial proliferation, tight junctions, and apoptosis. Bouchama et al. [27] observed that heatstroke patients with elevated rectal temperature showed significantly increased TNF, IL-1, and endotoxin, which decreased with temperature reduction, indirectly linking elevated body temperature to intestinal structural alterations.

2.3 Reduced Intestinal Blood Flow

Under high temperature, animals increase peripheral blood flow for heat dissipation, reducing intestinal blood flow [48-49]. Prolonged reduction may cause epithelial hypoxia [23], ATP depletion, lactate accumulation, and cellular dysfunction, ultimately causing necrosis and shedding, reduced villus height, and compromised epithelial integrity [38,48].

2.4 Intestinal Microorganisms

High temperature effects may be mediated through altered intestinal microflora. Peng et al. [7] used 16S rDNA PCR-DGGE to demonstrate that sustained 31°C affected cecal microflora structure and diversity in broilers. As heat stress duration extended, impacts on dominant bacterial communities in the duodenum, jejunum, and ileum of laying hens became more pronounced, coinciding with greater reductions in villus height and crypt depth [50]. Heat stress decreased Enterobacteriaceae while increasing Streptococcus and Staphylococcus [51], and enhanced Salmonella colonization [16,39-40]. Pathogens stimulate secretion of inflammatory cytokines (TNF, IL-1), inducing myosin light chain phosphorylation, opening tight junctions, and increasing permeability [35-36].

Song et al. [9] found high temperature reduced Lactobacillus and Bifidobacterium while increasing E. coli and Clostridium. Probiotic supplementation under heat stress increased Lactobacillus and Bifidobacterium, improved villus

height, and promoted tight junction protein expression, suggesting heat stress effects are associated with microbial composition changes. However, research on high temperature effects on microflora remains limited, and mechanisms remain unclear.

Summary

In summary, heat stress affects poultry intestinal structure, reduces mucosal integrity, and suppresses immune function. These effects may be associated with reduced feed intake, elevated body temperature, decreased intestinal blood flow, and altered microflora. By compromising intestinal structure and function, heat stress ultimately impairs poultry growth and health.

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