Effects of Dietary Sodium Alginate on Growth Performance and Renal Gene Expression in Broiler Chickens

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Introduction

Poultry became the world's most consumed meat in 2019, and the top consumption trend is expected to continue across the livestock sectors (Jacobs, 2022; FAOStat, 2020). The increased consumption is primarily attributed to consumers' perceptions of poultry as affordable, high in protein, tasty, and healthy with low-fat (National Chicken Council, 2022). To meet this growing demand, the poultry industry has successfully implemented breeding programs, resulting in a 400% increase in bird growth rate and a doubling of body weight over the past 50 years (Zuidhof et al., 2014; Barbut et al., 2008). However, accelerated growth of body weight results in elevated metabolic oxygen consumption, increase in reactive oxygen species (ROS), and excessive nitrogen turnover, which lead to oxidative, inflammatory, and hypoxic stress at organ levels (Coudert et al., 2023; Oke et al., 2024). In case of broilers, high-protein diets accelerate uric acid production and impose significant oxidative stress during the early stage of growth. This stress triggers transcriptional activation of Adenosine Deaminase (ADA) and Xanthine Dehydrogenase (XDH), promoting purine degradation and ROS generation.

Prebiotic sodium alginate, an extract of seaweed, is a naturally occurring acidic linear polysaccharide. In previous studies, sodium alginate has shown improvements in immune functions and antibacterial effects by enhancing phagocytic activity in humans, rats, and chickens (Fujihara and Nagumo, 1993; Yan et al., 2011). Those beneficial effects were proposed through two complementary mechanisms. First, it enhances the generation of short-chain fatty acids (SCFA) by reshaping the gut microbiota —particularly butyrate—which serves as a systemic anti-inflammatory and antioxidant metabolite. Second, it may directly modulate host gene expression through improved mineral binding, intestinal viscosity regulation, and reduction of postprandial ammonia absorption. These physiological adjustments can collectively reduce purine flux and oxidative stress at the renal level (Zhang et al., 2022; Rongshuang Han., 2025). However, no study has investigated the effects of sodium alginate on broiler growth performance and stress-related gene expressions.

Purpose

The purpose of this study was to evaluate dietary effects of sodium alginates on broiler growth and 12 stress-related renal gene expressions

Methods

All experimental procedures were reviewed and approved by the California Polytechnic State University Institutional Animal Care and Use Committee (Protocol #1613, 1908).

Bird diets, feeding, and body weight

A total of 270 chicks were obtained from a local hatchery after vaccination. The one-day old chicks were weighted and distributed across 18 floor pens (3 treatments; 6 pens per treatment; 15 birds per pen) to have a similar initial body weight per pen. Corn and soybean meal (SBM) based diet was formulated as the primary ingredients and adjusted according to the nutrient requirements recommended by the National Research Council (NRC, 1994). Birds were fed ad libitum for 6.5 weeks using three levels of sodium alginate at 0%, 0.05%, and 0.1% in 3 feeding phases - starter (0-2 weeks), grower (2-4 weeks), and finisher (4-6 weeks). Bird growth conditions were monitored, and mortality rate was recorded daily. Body weight gain and feed consumption were recorded weekly.

Sample Collection and Tissue Sampling

In 14, 28, and 42 days, birds were randomly selected from each treatment group for sample collection after euthanizing, and blood and tissue samples were immediately collected. Approximately 5 mL of blood was drawn aseptically from the wing vein and allowed to clot at room temperature for 30 min. The samples were centrifuged at 3,000 × g for 10 min to separate the serum, which was then aliquoted into 1.5 mL microtubes and stored at −80 °C until analysis. Tissue samples were collected from the kidney and cecum. The kidney tissues were divided for RNA and DNA analyses, and each portion was immediately immersed in liquid nitrogen for flash-freezing. Frozen samples were stored at −80 °C, and approximately 25 mg of tissue was used for nucleic acid extraction after partial thawing. To prevent cross-contamination, all dissection instruments were sterilized between samples, and all procedures were performed under aseptic conditions within a biosafety cabinet.

Serum Butyric Acid Quantification by ELISA

Serum butyric acid concentrations were determined using a commercial Butyric Acid ELISA Kit (Cat. No. ELK8174) according to the manufacturer's instructions. Before analysis, serum samples were thawed at room temperature and centrifuged at 10,000 × g for 10 min to remove lipids and debris, and the resulting supernatants were used for quantification. All samples were analyzed under identical conditions to minimize inter-assay variation. Absorbance was measured at 450 nm using a microplate reader (Bio-Rad, USA), and butyric acid concentrations were calculated based on a standard calibration curve.

RNA Extraction and qRT-PCR

Total RNA was extracted from 30 mg of kidney tissue using the RNeasy Plus Mini Kit (QIAGEN, Germany) according to the manufacturer's protocol. RNA concentration and purity were assessed using a NanoDrop 2000 spectrophotometer (Thermo Fisher Scientific, USA) before subsequent analysis. Complementary DNA (cDNA) was synthesized using the AccuPower RT PreMix Kit (Bioneer, Korea) following the manufacturer's instructions, and the resulting cDNA was stored at −20 °C until quantitative PCR analysis. Quantitative real-time PCR (qRT-PCR) was performed using a Takara Real-Time PCR System (Takara, Japan). The amplification program consisted of an initial denaturation at 95 °C for 5 min, followed by 40 cycles of 95 °C for 5 s and 59 °C for 30 s in a two-step protocol. Gene expression levels were calculated using the ΔCt method, with RPL13 serving as the housekeeping gene.

Results

Dring the period of 6 weeks, one-day old broiler chicks (42.1 g) grew and became market size birds (3094.1 g) with a linear increase of body weight up to 5 weeks and level off, thereafter (Fig. 1 a, b). No significant difference in body weight and body weight gain was observed between control birds and sodium alginate birds ($P > 0.05$) (Fig. 1 a, b). Similarly, no significant difference was found in feed intake and feed conversion ratio ($P > 0.05$) (Fig. 2 a, b). Based on the results of no difference in body weight gain and feed intake, the sodium alginate is expected to contribute to the balance of physiological adaptation rather than weight gain during the high-energy turnover period up to 5 weeks. At the end of 6 weeks, both feed intake and feed conversion ration were significantly lower in the diet of 0.1% sodium alginate over the control and 0.05% sodium alginate diets while body weight increased continuously (Fig.1, 2 a, b). These results indicated that 0.1% of sodium alginate supplements improved feed efficiency without adverse effects on broiler performance.

To identify molecular mechanisms under high-protein diets, the expressions of twelve renal stress-related genes were profiled, six of which were further analyzed: *ADA*, *XDH*, *HIF1A*, *TGFB1*, *ABCG2*, and *SCNN1A* (Fig. 3). Results indicated that broilers fed control diet or sodium alginate supplement at 0.05% resulted in progressive upregulation of *TGFB1, XDH, HIF1A,* and *SCNN1A* with body growth, reflecting fibrotic activation, oxidative burden, hypoxic adaptation, and ionic stress, respectively. Alginate supplement diet at 0.1%, however, significantly reduced these expressions, ADA and TGFBI at day 14, XDH, HIFIA, SCNNIA, and ABCG2 at day 42, respectively, suggesting enhanced transcriptional stability. Subsequently, these transcriptional markers reveal that broilers under control feeding experienced oxidative, hypoxic, and fibrotic stress, whereas alginate supplementation at 0.5% alleviated these burdens and restored renal homeostasis — a molecular demonstration of stress resolution not measurable by traditional phenotypic indices.

Conclusions and Recommendations

Our study indicated that no significant difference was observed in live bird weight for 6 weeks, regardless of dietary levels, with no difference seen for feed consumption, except week 6. However, the consistent down-modulation of ADA, XDH, and HIF1A highlights attenuation of early oxidative and purine-derived stress, while transient suppression of TGFB1 reflects controlled fibrotic remodeling. The stabilization of ABCG2 and SCNN1A suggests reduced renal excretory strain and osmotic load, collectively indicating an improved balance between oxidative metabolism, oxygen utilization, and structural adaptation. Yan et al. (2011) also reported that no significant difference in body weight was found when sodium alginate was fed 0.2%. However, the dietary sodium alginate induced a robust mucosal immune response, which may lead to a recline in mortality and salmonella infection. Improving immune response and antibacterial agents is important for enhancing broiler performance and meat safety. Prebiotics have been known to improve host's defensive mechanisms, while probiotics help maintain microbial balance and decrease pathogen load. So, it will be interesting to evaluate any synergistic effects on broiler performance and feed conversion ration when sodium alginate (prebiotic) and potential prebiotic agents are co-fed.

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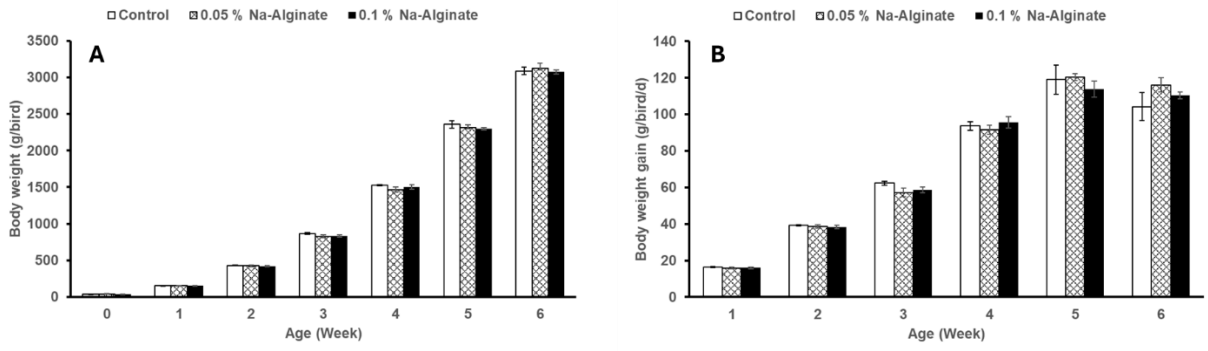

Figure 1. Effect of dietary sodium alginate on body weight and body weight gain of broiler chickens over the course of 6 weeks (n = 6 pens/dietary treatment).

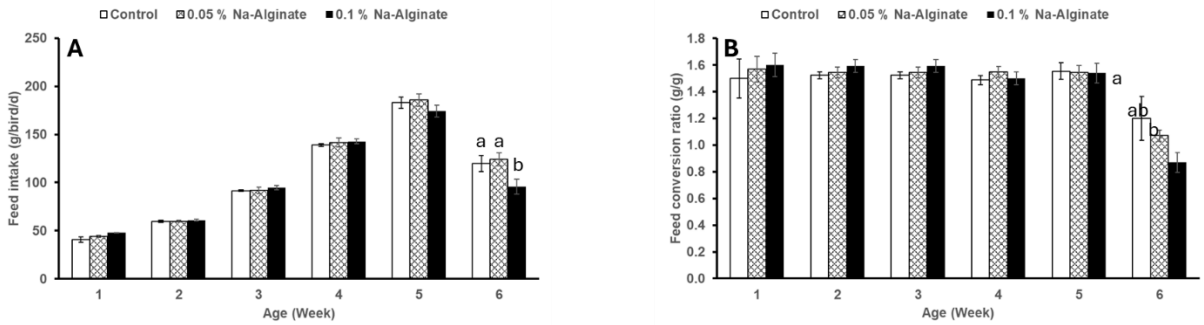

Figure 2. Effect of dietary sodium alginate on feed intake and feed conversion ratio of broiler chickens over the course of 6 weeks (n = 6 pens/dietary treatment).

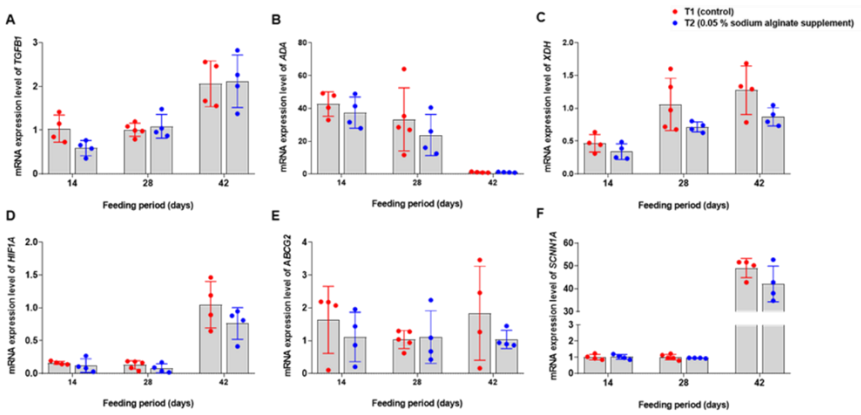

Figure 3. Time-course transcriptional profiles of five renal stress–related genes (*ADA*, *XDH*, *HIF1A*, *TGFB1*, ABCG2, and *SCNN1A*) showing differential modulation by 0.05% sodium alginate supplementation during the 42-day broiler rearing period.

(a) *TGFB1* expression representing delayed fibrotic activation and stable ECM remodeling. (b) *ADA* expression indicating attenuation of inflammatory purine metabolism. (c) *XDH* expression reflecting reduced oxidative load and uric acid synthesis. (d) *HIF1A* expression showing decreased hypoxic stress under alginate feeding. (e) *ABCG2* expression demonstrating lowered demand for metabolic waste efflux and improved renal metabolic efficiency. (f) *SCNN1A* expression indicating enhanced sodium reabsorption and epithelial barrier integrity, contributing to improved renal ion homeostasis and fluid balance.