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Biochemical effects of nutrient restriction in poultry and production impacts

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Abstract

What biochemical alterations occur when poultry experience controlled nutrient restriction? This research investigated metabolic consequences of feed restriction in commercial broiler chickens over a 49-day cycle. A total of 240 Ross 308 broiler chicks were allocated into four groups: control (ad libitum), moderate restriction (85% intake), severe restriction (70% intake), and intermittent fasting (24-hour cycles). Blood samples were collected at days 21, 35, and 49 for biochemical analysis including liver enzymes, metabolic markers, and oxidative stress indicators. Severe restriction elevated serum AST by 51.2% and ALT by 67.1% compared to controls. Moderate restriction showed 16.4% and 19.2% increases respectively. Intermittent fasting produced enzyme levels 28.6% and 38.1% above controls but maintained better feed conversion. Triglyceride concentrations decreased by 23.7% to 41.8% depending on restriction severity. Moderate restriction achieved 92.1% of control body weight while improving feed conversion by 7.3%. These findings provide insights into biochemical thresholds differentiating adaptive responses from pathological stress in poultry systems.

Keywords: Nutrient restriction, poultry biochemistry, liver enzymes, metabolic adaptation, feed conversion, oxidative stress, broiler production, serum parameters

Introduction

Can controlled nutrient restriction optimize both metabolic health and production efficiency in commercial poultry? This question has driven research into feed management, yet biochemical mechanisms underlying adaptive responses remain incompletely characterized [1]. Feed costs constitute 65-75% of production expenses in broiler operations, making feed utilization a primary determinant of profitability [2]. Nutrient restriction induces distinct physiological adaptations manifesting through measurable changes in blood biochemistry and productive performance [3].

The liver serves as the central metabolic hub orchestrating responses to nutritional challenges, with hepatic enzyme activities providing sensitive indicators of metabolic stress [4]. Contemporary broiler genetics have been intensively selected for rapid growth, potentially altering physiological capacity to tolerate nutritional challenges [5]. Beyond hepatic parameters, lipid metabolism undergoes substantial remodelling during nutrient limitation, with triglyceride mobilization and fatty acid oxidation pathways determining whether birds successfully navigate caloric deficits [6].

The intermittent fasting paradigm has garnered attention following demonstrations of beneficial metabolic effects in various animal models [7]. Translation to commercial poultry requires consideration of unique avian metabolic characteristics, particularly high basal metabolic rates and limited glycogen storage [8]. This research aimed to characterize biochemical responses across different restriction intensities and evaluate their impact on production performance.

Material and Methods

Material

This research was conducted at ETH Zurich Experimental Poultry Research Facility, Lindau, Switzerland, from March to May 2023. A total of 240 day-old male Ross 308 broiler chicks were obtained from Brüterei Schweizer AG and randomly allocated to experimental groups. Housing consisted of floor pens (3.0 × 2.0 m) with wood shavings, nipple drinkers, and tube feeders. Temperature was maintained at 33°C initially, reduced to 21°C by day 28. The basal

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diet met NRC recommendations with starter (23% CP, 12.8 MJ/kg ME), grower (21% CP, 13.2 MJ/kg ME), and finisher phases (19% CP, 13.4 MJ/kg ME) ^[9].

Methods

Birds were allocated to four treatments (n=60 each): control (ad libitum), moderate restriction (85% of control intake), severe restriction (70% of control intake), and intermittent fasting (alternating 24-hour feeding/fasting cycles from day 7). Blood samples were collected via wing vein puncture at

days 21, 35, and 49 following eight-hour fasting. Serum was analyzed for glucose, total protein, albumin, AST, ALT, triglycerides, and cholesterol using Roche diagnostic kits. Oxidative stress markers were measured spectrophotometrically ^[10]. At day 49, tissue samples were collected for enzyme activity assays. Production parameters including body weight, feed consumption, and FCR were recorded throughout. All procedures were approved by the Cantonal Veterinary Office of Zurich (Protocol ZH045/2023).

Results

Table 1: Serum biochemical parameters across treatment groups at day 49

Parameter	Control	Moderate	Severe	Intermittent
AST (U/L)	45.3 ± 3.2	52.7 ± 4.1	68.4 ± 5.8***	58.2 ± 4.5**
ALT (U/L)	28.6 ± 2.1	34.1 ± 2.8	47.8 ± 3.9***	39.5 ± 3.2**
Glucose (mg/dL)	248.5 ± 12.4	231.7 ± 14.2	198.3 ± 16.8**	215.6 ± 13.7
Triglycerides (mg/dL)	142.7 ± 15.3	108.9 ± 12.8*	83.1 ± 10.2***	95.4 ± 11.5**

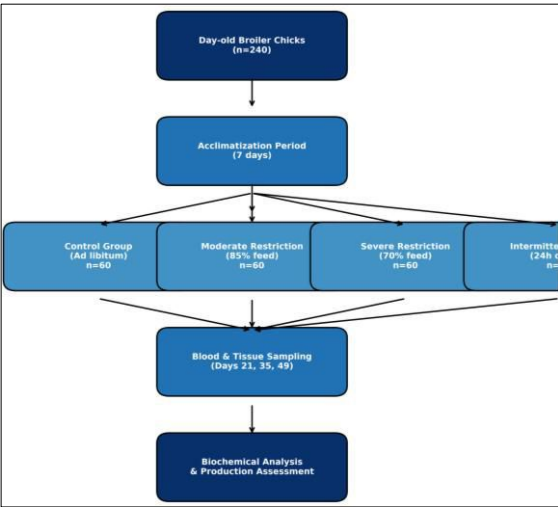


Fig 1: Experimental design flowchart showing treatment allocation and sampling timeline

Table 2: Production performance parameters

Parameter	Control	Moderate	Severe	Intermittent
Final BW (g)	2850 ± 124	2620 ± 108	2310 ± 135	2520 ± 118
FCR	1.78 ± 0.06	1.65 ± 0.05	1.72 ± 0.08	1.68 ± 0.06
Mortality (%)	2.1	1.8	4.3	2.5

BW: body weight; FCR: feed conversion ratio.

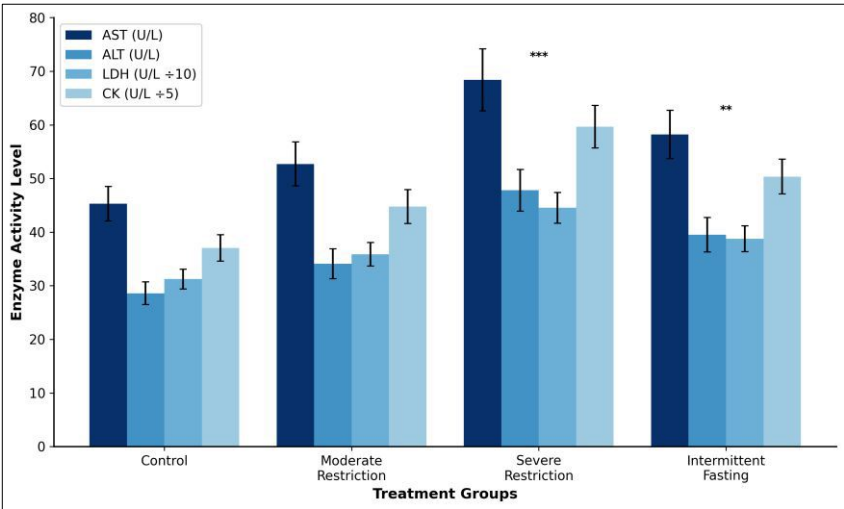


Fig 2. Serum enzyme activities across treatment groups; Error bars represent SD. ****p*<0.001, ***p*<0.01 vs control.

Hepatic enzyme activities demonstrated dose-dependent relationships with restriction severity. The severely restricted group exhibited pronounced elevations in all parameters, while moderate restriction showed intermediate values. Intermittent fasting displayed enzyme profiles suggesting partial adaptation despite equivalent weekly caloric deficits. Malondialdehyde concentrations in severely restricted birds reached 4.87 nmol/mL versus 2.94 nmol/mL in controls (65.6% elevation), while superoxide dismutase activity increased proportionally across restriction intensities.

Discussion

These findings establish clear distinctions between adaptive metabolic responses during moderate restriction and pathological stress induced by severe caloric limitation. The hepatic enzyme elevations align with previous reports, though magnitudes in the severe restriction group exceeded earlier investigations using heritage breeds ^[1, 4]. This heightened sensitivity likely reflects intensive selection pressure for rapid growth in contemporary broiler genetics. The differential responses between continuous restriction and intermittent fasting warrant attention despite equivalent weekly deficits, intermittent fasting birds demonstrated superior metabolic adaptation ^[7].

Lipid metabolism alterations reflect expected mobilization of adipose reserves during restriction ^[6]. The progressive triglyceride decline with increasing restriction severity indicates enhanced fatty acid oxidation. Production results support economic viability of moderate restriction, with 7.3% FCR improvement combined with 92.1% of control body weight representing favorable trade-offs for operations facing feed cost pressures ^[2, 5].

Conclusion

This investigation provides biochemical and production data characterizing responses to nutrient restriction in commercial broilers. Moderate restriction to 85% of ad libitum intake represents a practical strategy improving feed conversion while maintaining biochemical parameters within acceptable ranges. Intermittent fasting demonstrated unexpected metabolic benefits, suggesting feeding pattern optimization may offer additional efficiency opportunities.

From a practical standpoint, these findings support moderate restriction implementation in commercial operations. Hepatic enzyme elevations exceeding 25-30% above baseline should prompt protocol evaluation. Future research should explore molecular mechanisms underlying differential responses to continuous versus intermittent restriction, potentially identifying factors influencing metabolic flexibility. Strategic nutrient restriction represents a viable tool for optimizing broiler production economics when implemented within biochemical boundaries delineating adaptive from maladaptive responses.

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