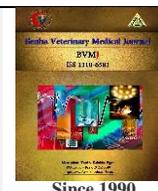




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Impacts of post-hatching feed restriction on growth performance and some physiological parameters in broiler and layer chickens

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ABSTRACT

The current investigation aimed to compare the impacts of delayed feeding post-hatching on the growth performance and hormonal responses of broilers and layers. A total of 180 one-day-old male Ross 308 and Lohmann chicks were used. The chicks were randomly allocated into four groups. Each group was represented in three replicates, with 15 birds per replicate. Group I: Ross308 chicks received feed immediately post-hatching; Group II: Ross308 chicks received feed after 72h post-hatching (FR broilers); Group III: Lohmann chicks were fed immediately post-hatching; Group IV: Lohmann chicks received feed 72h post-hatching (FR layers). All groups were fed ad libitum for the rest of the experiment (42 days). Growth parameters, including live body weight (BW), body weight gain (BWG), feed intake (FI), and feed conversion ratio (FCR), were recorded weekly. Blood samples were collected on the 7th, 28th, and 42nd days of age to determine serum corticosterone, glucose, and insulin levels. FR broilers exhibited a consistent reduction in BW, BWG, and FI, alongside an increased FCR. FR layers showed significantly decreased BW, BWG, and FI and higher FCR for most of the experimental period, although BW showed non-significant differences on the 42nd day of age. Furthermore, feed restriction induced a significant stress response in broilers, marked by elevated corticosterone levels throughout the experiment and transient increases in serum glucose and insulin on day 7 of age. In contrast, layers exhibited a more attenuated response, with only a brief corticosterone increase on day 7 of age. In conclusion, a prolonged delay in feed access post-hatching caused substantial effects on the physiological responses of fast-growing chickens (broilers) and slow-growing breeds (layers). Intriguingly, broiler chickens were more markedly affected by FR than layer chickens.

1. INTRODUCTION

Access to the first feed after hatching is critical in developing poultry's physiological and metabolic patterns (Li et al., 2022). Post-hatching feed restriction, defined as delayed feeding for many hours to days following hatching (Liu et al., 2020), has garnered increasing interest due to its potential long-term impacts on growth performance and metabolic responses in both fast- and slow-growing chickens (Hassan et al., 2023). After hatching from the incubation machine, chicks may lack food and water for up to 24 hours, a period known as the hatching window (Malchow et al., 2025). Furthermore, due to farm transportation, routine vaccinations, sexing, and other circumstances, chicks' feed deprivation time might reach up to 72 hours (Özlu et al., 2022).

Since the interval from the last embryonic days to the first week post-hatching is crucial for the growth and improvement of all body organs (Gawel et al., 2025), prolonged feed delay after hatching can reduce chick weight (Boyner et al., 2025; Lingens et al., 2021), impair the hormonal balance (Wang et al., 2020a; Wang et al., 2020b), delay intestinal development (Wang et al., 2020b; Liu et al., 2020), impact intestinal health (Li et al., 2022), and affect the improvement of the bird's immunity (Madej et al., 2024; Miska et al., 2022). Broilers and layers have been bred for distinct purposes. This leads to significant physiological

differences between fast-growing broiler chicks and slow-growing layer chicks (Buzala and Janicki, 2016). Broilers, selected for rapid growth rate and higher feed intake, have a lower basal metabolic rate (BMR) and use metabolizable energy more efficiently for growth, with lower maintenance energy requirements compared to layers (Swennen et al., 2007; Zhao et al., 2004; Buzala et al., 2015). In terms of growth performance, the initial week following hatching constitutes a significant percentage (20%) of the entire lifespan of broilers (Tona et al., 2004; Ma et al., 2024), where previous research has indicated that body weight increases two to three times during their first week (Wang et al., 2018; Vanhatalo et al., 2021). Thus, delayed feeding post-hatching has been demonstrated to negatively affect body weight gain (BWG) (De Jong et al., 2017), decrease feed intake (FI) (Li et al., 2022), and deteriorate feed-to-gain ratio (Akram et al., 2025). Layer chicks, while selected for their reproductive characteristics instead of rapid growth, may show sensitivity to restricted feeding. Research has indicated that a lack of feed can result in decreased body weight gain (Shinde et al., 2015) and delayed sexual maturity (age at first laying) (Bahry et al., 2023). Considering the hormonal responses, post-hatching FR triggers stress-related mechanisms, particularly the hypothalamic-pituitary-adrenal (HPA) axis (Rajman et al., 2006). A complex neuroendocrine system involving the hypothalamus, pituitary gland, and adrenal glands, which

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together control the body's reaction to stress and maintain homeostasis across multiple physiological systems (Post et al., 2003). Feed-restricted chickens showed high corticosterone levels, an accepted indicator of stress in birds (Romero et al., 2015), reflecting increased physiological stress (Feltenstein et al., 2003). Elevated levels of corticosterone are linked to reduced immune function and changes in energy metabolism, especially in broilers with high metabolic demands (Yang et al., 2015). At the same time, glucose homeostasis was disrupted. FR with subsequent elevated blood corticosterone level increases blood glucose concentrations through hepatic gluconeogenesis (Matos, 2008), while insulin levels in response to stress are variable; some studies report hyperinsulinemia and insulin resistance with increased corticosterone (De Baets et al., 2024), while others do not observe changes in insulin levels during stress (Brugaletta et al., 2022). These hormonal disturbances may continue after the initial FR period, affecting growth and metabolic efficiency in both fast- and slow-growing chickens (Sarjana et al., 2025; Zhao et al., 2014). Significantly, the extent of the impacts differs among strains. Broilers, which are selected for their quick growth, show greater metabolic and hormonal disturbances when feeding is delayed compared to layers, which, although being more robust, are still susceptible to prolonged feed deprivation effects (Simon et al., 2014). In this investigation, the aim was to compare the adaptive physiological responses of fast-growing (broiler) and slow growing (layer) chickens to delayed post-hatch feeding.

2. MATERIALS AND METHODS:

All procedures in this investigation were permitted by the Institutional Animal Care and Use Committee and the Research Ethics Board of the Faculty of Veterinary Medicine, Benha University. Approval number (BUFVTM 15-10-24)

2.1 Experimental birds

One hundred and eighty recently hatched male broiler and layer chicks were obtained from a commercial hatchery (Al-Wadi Poultry Company, El Sadat City, Menoufia Governorate, Egypt). The average body weight was approximately 43.9 g and 41.7 g for Ross 308 and Lohmann chicks, respectively. Wing bands were used to identify birds individually.

2.2 Experimental design and feeding management

Chicks within the same breed were split into four groups; each group contains 45 chicks (3 replicates of 15 birds/replicate):

Group I (Control broilers): Ross 308 chicks receive early nutrition upon hatching (within 4 hours).

Group II (FR broiler): Ross 308 chicks received the first feed 72 hours post-hatching.

Group III (Control layers): Lohmann chicks receive feed immediately post-hatching (within 4 hours).

Group IV (FR layers): Lohmann chicks received their first feed 72h post-hatching. Water was available ad libitum for the four treated groups.

A commercial diet for both breeds was obtained from Pyramids Poultry Company (Teraet El Mansouria, Giza

Governorate). The diet of Ross 308 was scheduled as starter, grower, and finisher phases, according to Ross 308's requirements. Lohmann's diet was scheduled as a starter (days 1-42) according to recommendations for nutrient levels in Lohmann chickens (Table 1).

2.3 Housing, lighting, and ventilation

The birds were kept in hygienic, well-ventilated rooms that had been disinfected using TH4, a synergistic combination of glutaraldehyde and quaternary ammonium compounds, which possess virucidal, bactericidal, fungicidal, and algicidal properties. Following disinfection, fumigation was performed using a mixture of formalin and potassium permanganate to ensure thorough decontamination of the environment. There was sufficient ventilation to eliminate moisture, allowing the litter to dry, as well as to eliminate ammonia from excrement and carbon dioxide that the birds had exhaled. Clean wood shavings were used for covering the floor, creating a 7 cm deep litter that was turned over once a week and replaced every two weeks. During the first two days, the chicks were continuously exposed to light from compressed filament lamps. Then the birds were given a lighting schedule of 23 L/1 D for the rest of the experiment (Khutal et al. 2022).

Table 1: Specifications of starter, grower, finisher broiler diets, and starter layer diet (as-fed basis).

Ingredient	Broiler starter	Broiler grower	Broiler finisher	Layers starter
Crude Protein (CP)	23%	21%	19%	20%
Metabolizable Energy (ME)	2950 Kcal/kg	3100 Kcal/kg	3200 Kcal/kg	2,750 kcal/kg
Crude Fiber	2.65%	2.34%	2.25%	6%
Crude fat	3%	4%	4.50%	7.5%
Digestible lysine	1.25%	1.10%	1%	0.80%
Digestible Methionine + Cystine	0.90%	0.78%	0.78%	0.60%
Total Calcium (Ca)	0.95%	0.75%	0.80%	1.0%
Available Phosphorus (P)	0.50%	0.24%	0.40%	0.45%

2.4. Sampling

2.4.1 Assessment of Growth Performance

Body Weight and Weight Gain

At the starting point of the trial, the initial live body weight of the birds was noted. Subsequently, body weights were monitored at 7-day intervals throughout the experimental period (42 days). Weighing was conducted in the early morning prior to feeding, using a digital electronic balance to ensure accuracy. Weekly live weight gain (g/bird) was determined by calculating the difference in body weight between two consecutive measurements.

Feed Intake and Feed Conversion Efficiency

Chicks were provided with their diets each morning. Daily feed intake was determined by measuring the difference between the amount of feed provided and the residual feed, then dividing by the number of chicks in each group.

Feed conversion ratio (FCR) was computed as the total feed intake (g/bird/week) divided by the corresponding weekly body weight gain (g/bird/week), as follows:

$$FCR = \frac{\text{Feed intake (g) /bird/week}}{\text{Body weight gain (g) /bird/week}} \quad (\text{Alkhalif et al., 2010})$$

2.4.2 Blood samples for Analysis of stress-related indicators in serum

On the 7th, 28th, and 42nd days of age, five birds from each group were chosen at random and humanely slaughtered. Blood samples were collected early in the morning in yellow Vacutainer tubes (clot activator tubes) and then centrifuged

for 15 to 30 minutes at 3000 rpm. Serum corticosterone, insulin, and glucose levels were later determined after the serum was thoroughly separated and stored in Eppendorf tubes at -20 °C.

Measurement of Corticosterone Levels

Corticosterone concentrations were determined using the Cor® assay, which is a competitive immunoassay based on direct chemiluminescent technology. The analysis was conducted with the Atellica® IM Analyzer (Siemens Healthcare GmbH, Henkestraße 127, 91052 Erlangen, Germany), following the procedure outlined by Hawley et al. (2016).

Measurement of Insulin Levels

Serum insulin concentrations were determined using the IRI® assay, a two-site sandwich immunoassay based on direct chemiluminescent technology. The analysis was performed with the Atellica® IM Analyzer (Mianaris Medical Co., Ltd. for Siemens Healthcare GmbH, Henkestraße 127, 91052 Erlangen, Germany), using the technique outlined by Shiraishi et al. (2011).

Measurement of Glucose Levels

Serum glucose concentrations were measured using the glucose oxidase enzymatic approach, as outlined by Brake et al. (1981).

2.5. Statistical analysis

SPSS was used to analyze the data statistically. The independent sample T-test was the procedure used for hypothesis testing. The analysis was done with SPSS software, version 27 (IBM Corp., Armonk, NY, USA). A probability level of $P \leq 0.05$ was considered statistically significant.

3. RESULTS

3.1. Growth performance parameters

The impact of feed restriction on the growth performance, including the live body weight and body weight gain of broilers and layers, is shown in Tables 2 and 3. The results revealed that the FR broiler group showed a significant reduction ($P < 0.05$) in the live body weight and the body weight gain compared to the control group throughout the experimental period (42 days); except on day one of age, there was a non-significant difference ($P > 0.05$) regarding the live BW. Regarding the FR layers, BW was significantly decreased ($P < 0.05$) compared to the control group during the whole experimental period; nevertheless, there was no statistically significant distinction ($P > 0.05$) among layer

Table 2: Effect of feed restriction on live body weight of broiler and layer chickens.

		live body weight (g)						
		Day one	1 st week	2 nd week	3 rd week	4 th week	5 th week	6 th week
Broiler	Control	43.68±0.34	172.52 ^a ±2.19	569.76 ^a ±5.69	1179.64 ^a ±13.03	1758.56 ^a ±17.68	2570.6 ^a ±24.53	2866.2 ^a ±23.29
	FR	43.8±0.28	82.32 ^b ±1.22	359.8 ^b ±5.59	812.04 ^b ±9.88	1225 ^b ±14.79	1955.6 ^b ±44.95	2031.8 ^b ±10.83
	<i>P</i> -value	0.784 ^{NS}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}
Layer	Control	41.32±0.34	65.36 ^a ±1.16	135 ^a ±2.41	240.44 ^a ±3.9	352 ^a ±4.49	546.6 ^a ±5.06	606.68±6.1
	FR	41.76±0.4	48.6 ^b ±0.5	116.04 ^b ±1.23	210.08 ^b ±2.32	314.72 ^b ±3.24	498.36 ^b ±4.32	590.2±3.9
	<i>P</i> -value	0.401 ^{NS}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.016 [*]	0.024 [*]	0.058 ^{NS}

Values are expressed as mean ± S.E.M. Within the same breed, values in the same column carrying different superscripts are significantly different at ($P < 0.05$)

Table 3: Effect of feed restriction on body weight gain of broiler and layer chickens.

		Body Weight Gain (g)						
		W0 - W1	W1 - W2	W2 - W3	W3-W4	W4-W5	W5 -W6	W0 -W6
Broiler	Control	128.84 ^a ±2.21	397.24 ^a ±4.62	609.88 ^a ±10.18	578.92 ^a ±11.39	812.04 ^a ±26.93	295.6 ^a ±12.85	2822.52 ^a ±23.28
	FR	38.52 ^b ±1.2	277.48 ^b ±5.31	452.24 ^b ±8.74	412.96 ^b ±12.18	627.6 ^b ±20.22	178.6 ^b ±12.4	1987 ^b ±10.81
	<i>P</i> -value	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}
Layer	Control	24.04 ^a ±1.1	69.64±1.78	105.44 ^a ±1.84	111.56 ^a ±3.06	194.6 ^a ±4.3	110.08 ^a ±5.37	565.36 ^a ±6.1
	FR	6.84 ^b ±0.34	67.44±1.07	94.04 ^b ±1.43	104.64 ^b ±3.66	183.64 ^b ±3.97	91.84 ^b ±4.22	548.44 ^b ±3.96
	<i>P</i> -value	0.000 ^{**}	0.296 ^{NS}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.000 ^{**}	0.001 ^{**}

Values are expressed as mean ± S.E.M. Within the same breed, values in the same column with different superscripts are significantly different at ($P < 0.05$).

groups on day 42 of age. Also, the FR layers showed a significant reduction in BWG in comparison to the control group throughout all weeks, except during the period of W1-W2, when there was no significant difference.

The impact of feed restriction on broiler and layer feed intake (FI) and feed conversion (FCR) is shown in Tables 4 and 5. The FR broiler group showed a significant decrease ($P < 0.05$) in feed intake throughout the whole experimental period. Regarding FCR, during the whole experimental period, the FR broiler group recorded a significant increase ($P < 0.05$) in FCR in contrast to the control group. When compared to the control group, the FR layer FI significantly decreased ($P < 0.05$) during all weeks except the last week of the experiment, W6, where there was a statistically insignificant difference ($P > 0.05$) between the FR and control groups. Concerning FCR, the FR layer group revealed a significant increase ($P < 0.05$) all over the experimental period compared to the control group, except during the period of W1-W2, no significant difference was observed.

Effect of feed restriction on serum corticosterone, glucose, and insulin levels in broilers and Layers (Table 6).

Serum corticosterone level

Regarding the broiler breed, the FR group showed a significant increase in serum corticosterone levels compared to the control group on d7 ($P > 0.05$), d28 ($P > 0.05$), and d42 of age ($P > 0.05$), whereas the FR layer group showed a significant increase on d7 ($P > 0.05$); however, non-significant differences were observed on d28 ($P > 0.05$) and d42 of age ($P > 0.05$) compared to the control group.

Serum glucose level

The results revealed that the FR broiler group showed a significantly higher serum glucose level on d 7 of age compared to the control group. However, no significant differences ($P > 0.05$) were observed on d 28 and d 42 of age between the broiler FR and control groups. Concerning the layer breed, non-significant differences were observed between the FR and control groups on d 7, d 28, and d 42 of age ($P > 0.05$).

Insulin hormone level

The FR broiler group displayed a significant ($P > 0.05$) increase in serum insulin level compared to the control group on d 7 of age. While it did not differ appreciably from controls on days 28 and 42 of age. With respect to the layer breed, no significant differences were noticed between the FR and control groups on d 7, d 28, and d 42 of age.

Table 4: Effect of delayed feeding on Feed intake in broiler and layer chickens.

		W0-W1	W1-W2	W2-W3	W3-W4	W4-W5	W5-W6	W0-W6	Daily FI
Broiler	Control	112.48 ^a ±1.62	417.44 ^a ±4.43	637.4 ^a ±2.45	886.56 ^a ±2.84	1320.96 ^a ±14.03	1452.48 ^a ±25.75	4827.32 ^a ±45.83	114.94 ^a ±1.09
	FR	38.36 ^b ±0.52	326.28 ^b ±3.81	563.12 ^b ±2.73	806.56 ^b ±9.04	1285.7 ^b ±14.05	1108.6 ^b ±26.96	4128.64 ^b ±29.04	98.3 ^b ±0.69
	P-value	0.000**	0.000**	0.000**	0.000**	0.000**	0.002**	0.000**	0.000**
Layer	Control	38.84 ^a ±0.64	135.52 ^a ±0.95	222.92 ^a ±3.98	229.16 ^a ±6.57	261.2 ^a ±2.38	303.84±1.58	1164.36 ^a ±15	27.72 ^a ±0.36
	FR	20.68 ^b ±0.1	129.16 ^b ±0.59	212.52 ^b ±2.11	213.56 ^b ±0.91	217.44 ^b ±2.75	300.48±2.23	1093.84 ^b ±6.7	26.04 ^b ±0.16
	P-value	0.000**	0.000**	0.025*	0.023*	0.000**	0.118 ^{NS}	0.000**	0.01*

Values are expressed as mean ± S.E.M. Within the same breed, values in the same column with different superscripts are significantly different at (P < 0.05).

Table 5: Effect of delayed feeding on feed conversion ratio in broiler and layer chickens.

		FCR						
		W0-W1	W1-W2	W2-W3	W3-W4	W4-W5	W5-W6	W0-W6
Broiler	Control	0.88 b±0.02	1.05 b±0.02	1.05 b±0.02	1.55 b±0.03	1.68 b±0.07	4.5 b±0.4	1.71b±0.02
	FR	1.02 a±0.03	0.42a±0.77	1.23 a±0.03	2a±0.06	2.76 a±1.03	5.15 a±0.25	1.87a±0.02
	P-value	0.001**	0.001**	0.000**	0.000**	0.000**	0.017*	0.000**
Layer	Control	1.69 b±0.07	1.97±0.05	2.13 b±0.06	2.11 b±0.1	1.24 b±0.03	2.76 b±0.19	1.9 b±0.03
	FR	3.27 a±0.21	1.91±0.03	2.26 a±0.04	2.04a±0.19	1.84 a±0.04	3.27 a±0.38	2.05 a±0.02
	P-value	0.000**	0.407 ^{NS}	0.054*	0.000**	0.008**	0.000**	0.001**

Values are expressed as mean ± S.E.M. Within the same breed, values in the same column with different superscripts are significantly different at (P < 0.05).

Table 6: Effect of feed restriction on serum level of corticosterone, glucose, and insulin in broiler and layer chickens.

		Corticosterone			Glucose			Insulin		
		D7	D28	D42	D7	D28	D7	D28	D42	
Broiler	Control	7.03 ^b ±0.37	13.60 ^b ±0.33	3 ^b ±0.15	174.5 ^b ±5.98	181 ±1.87	190.25 ±7.81	2.93 ^b ±0.34	1.74 ±0.29	8.64 ±0.86
	FR	17.02 ^a ±0.32	21.84 ^a ±1.07	5.73 ^a ±0.15	230.25 ^a ±4.61	209.75 ±12.13	194.25 ±4.59	7.26 ^a ±0.31	1.85 ±0.21	7.38 ±0.29
	P-value	.000**	.002**	.000**	.000**	0.058 ^{NS}	.065 ^{NS}	0.001**	0.768 ^{NS}	0.235 ^{NS}
Layers	Control	13.18 ^b ±0.4	16.53 ±0.34	26.46 ±0.78	261.5 ±14.08	237.5 ±6.24	223.76 ±1.78	1.68 ±0.18	1.48 ±0.41	0.8 ±0.04
	FR	14.68 ^a ±0.2	17.98 ±0.62	26.9 ±1.22	237.5 ±6.24	232 ±4.6	239.75 ±3.33	1.85 ±0.05	1.32 ±0.7	0.71 ±0.08
	P-value	0.028*	0.111 ^{NS}	0.780 ^{NS}	0.396 ^{NS}	0.505 ^{NS}	0.148 ^{NS}	0.44 ^{NS}	0.856 ^{NS}	0.395 ^{NS}

Values are expressed as mean ± S.E.M. Within the same breed, values in the same column carrying different superscripts are significantly different at (P < 0.05).

4. DISCUSSION

The effects of post-hatching feed restriction, particularly during the critical 72-hour window, have been extensively investigated in the context of chicken performance (Akram et al., 2025). A fasting period of seventy-two hours was selected to emulate commercial conditions, wherein the bird may be deprived of feed for up to 72 hours during standard post-hatch processing and transportation (Batal and Parsons, 2002). The obtained results in the current investigation indicate that chicks subjected to post-hatching feed restrictions for up to 72 hours revealed a significant decrease in body weight, body weight gain, and feed intake, and a worse feed conversion efficiency relative to groups that had early feeding. The impaired development of GIT may be the main cause of the observed decline in growth performance. Under normal conditions, the early ingestion of feed plays an important role in stimulating the maturation of the gastrointestinal tract in birds (Gawel et al., 2025). However, when feed intake is delayed, this natural stimulation does not occur at the appropriate time. As a consequence, several structural and functional impairments are observed in the intestine. One of the most significant changes is the reduction in the surface area of the intestinal villi, which directly limits the absorptive capacity of the gut (Holleman et al., 2020). Additionally, a noticeable decline in crypt depth leads to a lower proportion of proliferating cells. Moreover, the overall development of key intestinal segments duodenum, jejunum, and ileum, is also negatively affected (Lopes et al., 2020; Qu et al., 2021). These morphological impairments, in turn, resulted in reduced nutrient absorption and utilization (Hedlund and Jensen

2022), which is a major influence on feed intake and growth (Corduk et al., 2013).

Another point to consider is that adequate feeding in the recent post-hatch phase is essential for skeletal muscle development and satellite cell proliferation, thus important for optimal muscle growth and final body weight (Payne et al., 2020). Consequently, delayed feeding after hatching leads to irreversible growth depression due to impairment in satellite cell activity and delayed muscle maturation (Halevy et al., 2000). The obtained results of the current study demonstrated not only a significant reduction in body weight but also a notable decline in feed intake among the FR groups. This diminution in feed consumption may be closely linked to the physiological stress response, which often triggers a cascade of hormonal changes. One of the key hormones implicated in this process is leptin. In avian species, leptin is a well-known satiety hormone secreted primarily by adipose tissue and has a vital contribution in feed intake regulation and energy homeostasis (Dridi et al., 2000). Under stress conditions, the secretion of leptin can be elevated (Dridi et al., 2008), leading to suppressed appetite and reduced motivation to consume feed (Denbow et al., 2000). Therefore, the elevated leptin levels observed under stress conditions could explain the decreased feed consumption after FR, which in turn contributes to the observed decline in body weight.

Some studies (Peebles et al., 2017; Daşkıran et al., 2012) have shown that chicks with delayed feeding in the initial period of life may experience compensatory growth, a physiological process marked by a high growth rate after restricted feeding or unfavorable conditions (Zhang et al., 2020), and catch up to a similar final weight as chicks that received feed. Without delay if the period of restriction is not

more than 24 hours (Özlü et al. 2022; Gonzales et al. 2003; Vargas et al. 2009). While some compensatory growth may occur after the restriction period, the extent of recovery depends on the timing, duration, and dietary conditions during and after the restriction (Hedlund and Jensen, 2022). As the duration of feed restriction proceeds over 24 hr., the severity of performance loss increases (De Jong et al., 2017). In the current investigation, both broilers and layers were impacted by feed restriction, as reported by Shinde Tamboli et al. (2018). However, in the current study, the detrimental effects of feed restriction on growth performance were more pronounced in the fast-growing (broiler) breed, where the FR group reached a significantly lower live body weight at the conclusion of the trial period in comparison to the control group. This finding confirms the association between the holding time post-hatching and final live body weight in broilers, as Wyatt et al. (1985) reported that chicks that were kept in the hatching window for 14 to 32 hours weighed 5-32% less than chicks maintained in the window for up to 7 hours only. While in layers, compensatory growth was sufficient to eliminate BW differences by day 42 of age, with the delayed-fed layer group recording the same live body weight as groups that received early feeding. These results match those observed in an earlier study reported by Simon et al. (2014).

Along with the negative consequences of feed delays on growth measures, feed restriction has been associated with an increase in physiological stress in poultry. This stress response is commonly indicated by raised levels of plasma corticosterone (CORT) (Wijnen et al., 2022), which is the primary adrenal steroid hormone in birds. Corticosterone is secreted in response to various stressors by means of the stimulation of the hypothalamus-pituitary-adrenal (HPA) axis (Abraham et al., 2025). In the current study, broiler chicks subjected to feed restriction exhibited a sustained elevation in serum corticosterone levels, even after refeeding. However, many studies present inconsistent findings regarding the direct long-term effect of early feed delay on basal corticosterone levels (Wijnen et al., 2022). Earlier research has demonstrated that corticosterone levels increase acutely during periods of food and water deprivation. Nevertheless, these elevations tend to normalize once feed becomes available, with corticosterone concentrations returning to levels similar to those seen in chicks that were fed immediately post-hatch. (Van De Ven et al., 2013; Zulkifli et al., 2016; De Jong et al., 2017). Despite this, there is growing evidence suggesting that early nutritional deprivation may have lasting effects on the endocrine response system. Specifically, it may sensitize the adrenocortical axis, leading to an exaggerated corticosterone response when birds are exposed to acute stressors later in life (Rajman et al., 2006). These stressors may include physical handling during blood sampling or exposure to fear-inducing conditions. Thus, even if baseline levels normalize, the heightened reactivity of the system may persist, indicating long-term physiological alterations due to early feed restriction.

Regarding slow growing (layer) breeds, the impact of early feed restriction on corticosterone levels appears to lessen over time. Although a marked elevation in corticosterone concentration was observed on day 7 post-hatch, the differences in hormone levels on days 28 and 42 of age were not statistically significant in comparison with the control

group. This pattern aligns with the results of Onbaşilar et al. (2009), who stated that the stress response induced by early feed restriction may be temporary. Their research supports the idea that although short-term deprivation can trigger significant physiological stress responses, these effects tend to subside as the layers adapt to the altered nutritional regimen. As a result, the release of corticosterone becomes less pronounced in later stages of development. This adaptation may reduce the long-term physiological burden of early feed restriction, potentially mitigating negative effects on welfare, while still having implications for growth and performance.

In the present study, delayed feeding disrupted blood glucose homeostasis in broiler chicks, causing a compensatory rise within the first two weeks post-refeeding. These results are consistent with the findings of Richards et al. (2010), who reported similar compensatory increases in whole-blood glucose in chicks subjected to a 72-hour feed delay. This glucose rebound is likely supported by the upregulation of SGLT1, a sodium-dependent glucose co-transporter located in the apical membrane of the chicken intestine, which plays a critical role in glucose absorption (Li et al., 2020). This also aligns with the results obtained from our current study on the impacts of post-hatching feed delay on SGLT1 in the chicken intestine, which showed upregulation of SGLT1 in birds subjected to 72 hours of food deprivation. According to Xie et al. (2015), elevated blood glucose may form part of the physiological 'fight-or-flight' response, enhancing survival during stress, as birds that withstand stressors tend to exhibit higher circulating glucose levels. Interestingly, in the slow-growing (layer-type) breed, no significant alterations in blood glucose levels were detected throughout the study period. This discrepancy likely reflects the divergent metabolic demands of broilers and layers during the early post-hatch phase. Broilers, driven by a rapid anabolic growth program, are more metabolically sensitive to delayed feed access. In broilers, such delays in feed suppress hepatic lipogenesis, the liver's conversion of carbohydrates into fatty acids essential for rapid muscle and body mass development, thereby producing transient shifts in glucose levels during the compensatory phase (Shiraishi et al., 2011; Richards et al., 2010).

Compared to layer-type chickens, broiler chickens showed a lower mean blood glucose level in the current investigation. This observation may be linked to genetic selection, as the rapidly developing broiler breed has been reported to have elevated fasting insulin levels and slightly lower fasting glucose levels (Berenjian et al., 2023). Another important finding was that broilers subjected to a 72-hour post-hatch feed delay exhibited a transient increase in serum insulin levels on day 7 of age. This spike is likely a compensatory metabolic adaptation in response to elevated glucose levels, aiming to facilitate glucose uptake and support anabolic processes during the recovery and re-feeding phases. Moreover, elevated levels of stress-related hormones such as corticosterone, glucagon, and catecholamines have been shown to impair insulin sensitivity. According to Berenjian et al. (2023), increased glucocorticoids as a part of the physiological stress response can induce insulin resistance that may result from alterations in key components of the insulin signaling cascade. In contrast, layer-type chickens displayed fewer metabolic disturbances following early feed restriction. This distinction is likely due to the genetic and

physiological differences between the two strains; broilers are selectively bred for rapid growth and exhibit stronger hepatic lipogenesis, while layers possess a more moderate lipogenic and adipogenic response during early development (Lu et al., 2021). Consequently, early post-hatch feed restrictions exert a more significant impact on broilers.

5. CONCLUSIONS

In conclusion, early feed restriction can trigger physiological stress, negatively influencing both growth performance and endocrine function. However, layer-type chicks demonstrated a milder response, likely due to their genetic programming, which prioritizes reproductive capacity and egg production over rapid body mass accumulation. As a result, the metabolic pathways in layers are less affected by early nutritional deprivation compared to broilers.

6. REFERENCES

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