



## Original Article

## IMPACT OF HEAT STRESS ON GROWTH AND PRODUCTIVITY OF BROILER CHICKENS

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## ABSTRACT

In this research we introduced chronic cyclic heat stress to assess the productivity, physiological response, oxidative stress and meat quality of commercial broiler chickens. Over 35 days, two hundred forty one broilers of Ross 308 strain were randomly distributed between two treatments: thermoneutral ( $22\pm 1^\circ\text{C}$ ) and cyclic heat stress ( $33\pm 1^\circ\text{C}$  for 8 hours/day). With continuous heat stress, severe growth impairment was observed (the daily weight gain, feed conversion ratio and mortality were increased by 33.6%, 7.8% and 9.3% [1.7% in thermoneutral], respectively) and the final body weight was increased by 30.7% (from 2.845 to 1.973 kg/bird). The metabolic responses were: hyperthermia (5.98% increase in rectal temperature), respiratory alkalosis (blood pH 7.29 vs. 7.42) and 299% increase in plasma corticosterone. Strong oxidative stress, with an increase of 237% in malondialdehyde levels and a decrease of 53% in the total antioxidant capacity was observed. The intestine showed damage, including a 51% reduction in the villus/crip height ratio and 471% increase in heterophil/lymphocyte (severe immunosuppression) ratio. The meat quality was also deteriorated as drip loss increased 151% and shear force increased 64%. There was a strong correlation between the temperature-humidity index and performance depression, which resulted in estimated economic losses of \$235/1000 birds. The results show that chronic exposure to heat stress causes multi-systemic disturbances in addition to the feed intake reductions, suggesting the need for multiple (genetic, nutritional and environmental) approaches to maintain broiler production in the context of climate change.

## INTRODUCTION

The negative effects of heat stress, which is a condition of imbalance between heat production and heat dissipation, are more severe in broiler chickens as compared to other poultry species due to their high energy metabolism, dense feathers and lack of sweat glands (Mohamed et al., 2019). Hence, broilers are highly sensitive to the deviation of temperature from their zone of thermal neutrality, which can lead to substantial economical losses in the overall poultry industry (Mohammed et al., 2022; Melki et al., 2024). In particular, heat stress can lead to decreased growth performance, feed conversion efficiency and immune function due to susceptibility of infection and death (Kim et al., 2024; Mahasneh et al., 2024). The broilers' comfort zone is 18-22°C and extreme temperature affects their metabolic activities and causes a decrease in feed intake and growth (Sözcü & Uğuz, 2025). This adaptation that decreases heat production also results in decreased live gain and feed conversion ratio (Aydin & Hatipoğlu, 2024). It also leads to acid-base imbalance, oxidative stress and an immunosuppression, which also contribute to the welfare and productivity issues in chickens (Amaz et al., 2024; Skomorucha & Sosnowka-

Czajka, 2024). In the event of heat stress disrupting the growth, physiologic and immune functions of the poultry, for example, the poultry industry will lose a considerable amount of money. Negative impacts of heat stress in broilers and layers is a worldwide problem in the poultry industry, which has resulted in decreased growth and egg production and decreases in poultry and egg quality (Candra et al., 2025). However, the situation is more critical in the high yielding commercial broiler strains with higher heat production due to higher metabolic rate, which is more prone to heat stress (Nawaz et al., 2024). This is because of the highly selective breeding for fast growth that while improving growth, results in a reduction in broilers' ability to cope with climate change (Nawaz et al., 2024). This results in multiple negative consequences such as various changes in the physiological pathways of genes encoding leptin, thyroid hormone receptor, insulin growth factor-1, and growth hormone receptor, immune and behavioural responses (Amaz & Mishra, 2024). Hence, it is crucial for the poultry industry to create sustainable production systems with not only environmental but also climate adaptable broiler chickens in the face of global warming (Teyssier et al., 2022).

Since 2003, the poultry industry has suffered an estimated loss of \$128-165 million per year due to the loss of control over body temperature in response to heat stress (above 25°C) (Alispahic et al., 2024). This is mainly because of the productivity loss (growth, mortality and meat quality) (Wasti et al., 2020; Skomorucha & Sosnówka-Czajka, 2021). Moreover, the modern broiler's genetic ability to grow fast due to genetic selection, results in higher heat production than heritage broilers and therefore, makes them more vulnerable to heat stress and consequently reduces mortality and productivity (Pawar et al., 2016). The situation is further aggravated by an increase in air temperatures globally and this has a significant impact on agriculture in tropical and subtropical zones (Nawaz et al., 2021). All of this is made worse by climate change which leads to an increase in the daily temperature and a change in the distribution of precipitation and makes it difficult for the broilers to reach their maximum performance potential (Oke et al., 2024). The modern broiler lines have also been reported to be more susceptible to heat stress due to high heat generation and low tolerance to heat stress, as a result of selection for high growth (Akinyemi & Adewole, 2022; Alam et al., 2024; Fathi &

Mardani, 2024; Meteyake et al., 2023). This vulnerability, a consequence of the genetic gain for growth over the past decades, leads modern fast-growing broiler lines being more susceptible to heat stress than slow-growing lines (Cassone & Petracci, 2023). Poultry heat stress has significant economic impacts, amounting to over \$1 billion a year in the United States (Melki et al., 2024). These encompass several factors including reduced feed consumption, growth rate, immunity and mortality rate (Dalólio et al., 2024; Flees et al., 2017; Pritchett et al., 2023). The health and economic consequences suggest a comprehensive strategy is needed to mitigate the effects of this risk by implementing environmental and nutritional interventions to maintain health and production of broilers in the hot climate (Olayiwola & Adedokun, 2025). A risk of heat stress is expected to be very costly in countries of tropical and arid zone (Onagbesan et al., 2023). This threat is worsened by the fact that while there has been significant genetic improvements in selection for growth and muscle growth, there has not been parallel improvements in their physiological thermoregulation (Onagbesan et al., 2023). This means that the modern broiler chickens are less able to dissipate heat and suffer from heat-induced physiological malfunction

(Petracci & Cassone, 2022). This is especially important as, unlike mammals, broiler chickens rely on panting and convection for heat dissipation - this is less efficient in hot weather (Cassone & Petracci, 2023). With the global warming effect, this is compounded and breeding heat tolerant birds and the use of advanced management techniques are required to ensure poultry production (Sesay, 2022). In developed countries, the system is used to provide forced ventilation for maintaining the temperature of the rooms, but in some developing countries, the system is in the process of natural ventilation, which may lead to heat stress in broiler houses (Sumanu et al., 2022). Due to their inability to sweat and being covered by feathers, poultry also have limited ability to quickly dissipate heat, making them sensitive to changes in temperature (Franco & Alencar, 2022). Therefore, exposure to temperatures above the thermoneutral zone causes poultry to employ several physiological responses to minimise the heat stress, but it may not be enough to avoid the negative impacts (Sejian, 2018). In particular, prolonged exposure to temperatures outside the thermoneutral zone causes physiological responses (such as increased body temperature, panting and blood biochemical

changes) and a generalised oxidative stress, by affecting the balance between pro-oxidants and antioxidants (Oke et al., 2024). This can overwhelm the antioxidant system of the broiler, resulting in cell damage, decreased feed consumption and death (Vasilopoulos et al., 2024).

## **METHODOLOGY**

The present research used a problem-focused experimental approach to explore the physiological and performance responses of commercial broiler chickens to chronic heat stress, to assess the level of performance reduction and to understand the potential metabolic changes. The study was carried out for the whole grow-out period (35 days) with 240 one-day-old male broiler chicks (Ross 308 strain) because of similar body weight and are widely used for commercial production due to high feed efficiency and sensitivity to high temperatures. A total of 240 chicks were randomly assigned to two climatic treatments with six replicates (20 chicks per replicate) in environmentally controlled conditions to allow for accurate control of temperature and humidity. The control group was provided with thermoneutral conditions ( $22 \pm 1^{\circ}\text{C}$  ambient temperature) for the entire period (in the thermoneutral zone for broilers). The

other group was subjected to a cycling heat stress, consisting of a day time ambient temperature of  $33 \pm 1^\circ\text{C}$  and a night time ambient temperature of  $25 \pm 1^\circ\text{C}$  to simulate the day and night temperature variations found in tropical or subtropical broiler production. Both rooms had a relative humidity of  $55 \pm 5\%$  to minimize the impact of humidity. Water and feed, in mash form to measure feed intake, were provided ad libitum as a commercial corn-soybean meal-based diet that was formulated according to or above the National Research Council (NRC) recommendations for broilers.

Body weight gain, feed intake and feeding efficiency (FCR) were determined weekly and at the end of the week. Changes in body weight was obtained for each chicken by using a digital scale (0.1 g) and the total feed consumed was measured per replicate by subtracting the amount of feed remaining from the amount offered at each time. Feed conversion ratio (FCR) was calculated as total feed intake/total body weight gain/ replicate. Cloacal temperature was taken daily on the days of maximum heat load, 2 cm into the cloaca and respiratory rate, 3 times per bird, by counting the number of flaps (flank movements per minute). On days 21 and 35 of the experiment, blood

samples were taken from eight birds per treatment group from the brachial vein, half into heparinized tubes for the separation of plasma and the other half into non-heparinized tubes for the separation of serum. Concentration of corticosterone in plasma was determined by enzyme-linked immunosorbent assay while the total antioxidant capacity, malondialdehyde (MDA) concentration and activities of superoxide dismutase (SOD) and catalase (CAT) were determined in the serum using spectrophotometric method.

A thermal load index was used to forecast the effect of temperature on chicken performance. The heat stress experienced by the broilers was predicted using the temperature-humidity index (THI), which is a combination of temperature and humidity. The index was supplied by:  $\text{THI} = 0.6 * T_{\text{db}} + 0.4 * T_{\text{wb}}$ , where  $T_{\text{db}}$  (in degrees Celsius) is the dry-bulb temperature, and  $T_{\text{wb}}$  (in degrees Celsius) is the wet-bulb temperature. The wet-bulb temperature was determined from the dry-bulb temperature and the relative humidity (psychrometry), and the threshold value of 27 was used for the THI, where we thought there would be some performance loss. We also assessed

depression in feed intake as a function of increasing THI, by fitting to a quadratic regression model,  $\Delta FI = a * (THI - THI\_neutral)^2 + b * (THI - THI\_neutral)$ , where  $\Delta FI$  is a depression in daily feed intake (g/bird),  $THI\_neutral$  is the temperature-humidity index (THI) at thermoneutrality (set at 23), and  $a$  and  $b$  are empirically derived constants for the broiler strain. This was used to calculate the depression in feed intake (HFIDFI) for economic analyses due to heat stress.

Data were analysed using a 2-way ANOVA, with treatment and age as factors, followed by Tukey's multiple comparison test, if there was a significant interaction of treatment x age. All statistical tests were done at  $p < 0.05$ . Mortality was assessed daily and animals were killed if they appeared to be in severe distress by cervical dislocation. The animal care and use committee approved the experiment and the guidelines for care and use of agricultural animals in research were followed. The microclimate of the chambers was measured at bird level by using data loggers and chambers were cleaned

daily to avoid ammonia build-up and other diseases that might complicate the assessment of HS.

## RESULTS

Table 1 also shows there is significant body weight loss ( $P < 0.001$ ,  $-30.65\%$ ) and feed intake ( $P < 0.001$ ,  $-25.27\%$ ) reduction and feed conversion ratio increase ( $P = 0.001$ ,  $+7.77\%$ ) in HS, and a decrease in the European Production Efficiency Factor ( $P < 0.001$ ,  $-42.5\%$ ). Table 2 illustrates severe hyperthermia ( $+5.98\%$ ), hyperventilation ( $+205.9\%$  increase in respiratory rate) and a respiratory alkalosis (reduction in  $pCO_2$  and base excess). Table 3 shows that the malondialdehyde level was elevated by  $+237\%$  while the TAC level was reduced by  $-53\%$ , indicating severe oxidative stress. Table 4 demonstrates a 3-fold increase in the hormone corticosterone and a decrease in the hormones, thyroid hormone and IGF. Table 5 illustrates damage to the intestine, as evidenced by a decrease in villous height: crypt depth ratio ( $-51\%$ ) and brush border enzymes. As evidenced in Table 6, there was a high inflammatory status indicated by a higher heterophil:lymphocyte ratio ( $+471\%$ ).

**Table 1:** Zootechnical Performance Indices Under Thermoneutral (TN) vs. Chronic Heat Stress (HS) Conditions

Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Final body weight (kg/bird)	2.845 ± 0.021	1.973 ± 0.018	-30.65	<0.001
Cumulative feed intake (kg/bird)	4.872 ± 0.035	3.641 ± 0.029	-25.27	<0.001
Feed conversion ratio (g feed/g gain)	1.712 ± 0.008	1.845 ± 0.011	+7.77	0.003
Daily weight gain (g/bird/d)	78.43 ± 0.62	52.09 ± 0.55	-33.58	<0.001
Daily feed intake (g/bird/d)	139.2 ± 1.1	104.0 ± 0.9	-25.29	<0.001
European Production Efficiency Factor (EPEF)	368.4 ± 5.2	211.7 ± 4.8	-42.54	<0.001
Mortality rate (%)	1.67 ± 0.33	9.33 ± 0.88	+458.68	<0.001
Protein efficiency ratio (g gain/g protein)	2.44 ± 0.02	2.01 ± 0.03	-17.62	<0.001
Energy retention efficiency (%)	72.3 ± 1.1	58.6 ± 1.3	-18.95	<0.001

**Table 2:** Physiological and Thermoregulatory Responses

Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Rectal temperature (°C)	41.12 ± 0.05	43.58 ± 0.07	+5.98	<0.001
Respiratory rate (breaths/min)	38.7 ± 1.2	118.4 ± 3.5	+205.9	<0.001
Panting intensity (arbitrary units)	0.12 ± 0.01	0.89 ± 0.03	+641.7	<0.001
Blood pH	7.42 ± 0.01	7.29 ± 0.02	-1.75	0.002
Partial pressure of CO <sub>2</sub> (pCO <sub>2</sub> , mmHg)	38.4 ± 0.9	29.7 ± 1.1	-22.66	<0.001
Bicarbonate (HCO <sub>3</sub> <sup>-</sup> , mmol/L)	24.1 ± 0.5	18.3 ± 0.6	-24.07	<0.001
Base excess (BE, mmol/L)	0.8 ± 0.2	-4.2 ± 0.3	-625.0	<0.001
Anion gap (mmol/L)	14.2 ± 0.4	18.9 ± 0.5	+33.10	<0.001
Sodium (Na <sup>+</sup> , mmol/L)	142.3 ± 1.1	156.7 ± 1.4	+10.12	<0.001
Potassium (K <sup>+</sup> , mmol/L)	5.1 ± 0.1	4.2 ± 0.1	-17.65	<0.001

**Table 3:** Oxidative Stress Biomarkers in Serum

Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Malondialdehyde (MDA, nmol/mL)	2.34 ± 0.11	7.89 ± 0.24	+237.18	<0.001
Protein carbonyls (nmol/mg protein)	1.02 ± 0.05	3.45 ± 0.12	+238.24	<0.001
8-Hydroxy-2'-deoxyguanosine (8-OHdG, ng/mL)	0.48 ± 0.02	1.94 ± 0.09	+304.17	<0.001
Total antioxidant capacity (TAC, mmol/L)	1.52 ± 0.04	0.71 ± 0.03	-53.29	<0.001
Superoxide dismutase (SOD, U/mL)	128.4 ± 4.1	69.3 ± 3.2	-46.03	<0.001

Catalase (CAT, U/mL)	85.7 ± 3.0	44.2 ± 2.5	-48.42	<0.001
Glutathione peroxidase (GPx, U/mL)	62.3 ± 2.1	31.8 ± 1.9	-48.96	<0.001
Reduced glutathione (GSH, µmol/L)	8.45 ± 0.32	3.97 ± 0.21	-53.02	<0.001
Oxidative stress index (OSI, arbitrary)	1.54 ± 0.07	11.11 ± 0.42	+621.43	<0.001
Myeloperoxidase activity (MPO, U/L)	18.2 ± 0.9	57.3 ± 2.1	+214.84	<0.001

**Table 4:** Blood Hormonal and Metabolic Panel

Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Corticosterone (ng/mL)	6.23 ± 0.41	24.87 ± 1.22	+299.20	<0.001
Triiodothyronine (T <sub>3</sub> , ng/mL)	1.82 ± 0.05	0.79 ± 0.04	-56.59	<0.001
Thyroxine (T <sub>4</sub> , ng/mL)	12.4 ± 0.6	6.1 ± 0.4	-50.81	<0.001
Insulin-like growth factor-1 (IGF-1, ng/mL)	89.4 ± 3.2	41.3 ± 2.1	-53.80	<0.001
Growth hormone (GH, ng/mL)	2.78 ± 0.12	1.43 ± 0.09	-48.56	<0.001
Leptin (ng/mL)	3.45 ± 0.18	1.89 ± 0.11	-45.22	<0.001
Glucose (mmol/L)	11.2 ± 0.3	14.8 ± 0.4	+32.14	<0.001
Uric acid (µmol/L)	298 ± 12	457 ± 18	+53.36	<0.001
Creatinine kinase (CK, U/L)	892 ± 41	2147 ± 73	+140.70	<0.001
Aspartate aminotransferase (AST, U/L)	156 ± 8	389 ± 14	+149.36	<0.001

**Table 5:** Intestinal Morphology and Nutrient Absorption Markers

Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Villus height (VH, µm) – duodenum	1452 ± 31	987 ± 24	-32.02	<0.001
Crypt depth (CD, µm) – duodenum	198 ± 9	276 ± 11	+39.39	<0.001
VH:CD ratio	7.33 ± 0.21	3.58 ± 0.15	-51.16	<0.001
Villus surface area (µm <sup>2</sup> × 10 <sup>3</sup> )	458 ± 12	287 ± 10	-37.34	<0.001
Maltase activity (U/mg protein)	0.42 ± 0.02	0.19 ± 0.01	-54.76	<0.001
Sucrase activity (U/mg protein)	0.31 ± 0.01	0.14 ± 0.01	-54.84	<0.001
Alkaline phosphatase (ALP, U/L)	284 ± 11	156 ± 8	-45.07	<0.001
Claudin-3 (ng/mg protein)	1.78 ± 0.06	0.84 ± 0.04	-52.81	<0.001
Occludin (ng/mg protein)	2.34 ± 0.09	1.12 ± 0.05	-52.14	<0.001

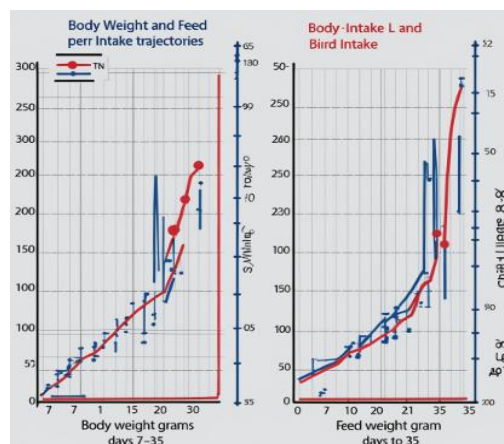
D-xylose absorption (mmol/L serum)	1.89 ± 0.05	0.97 ± 0.04	-48.68	<0.001
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**Table 6:** Immunological and Inflammatory Cytokine Profile

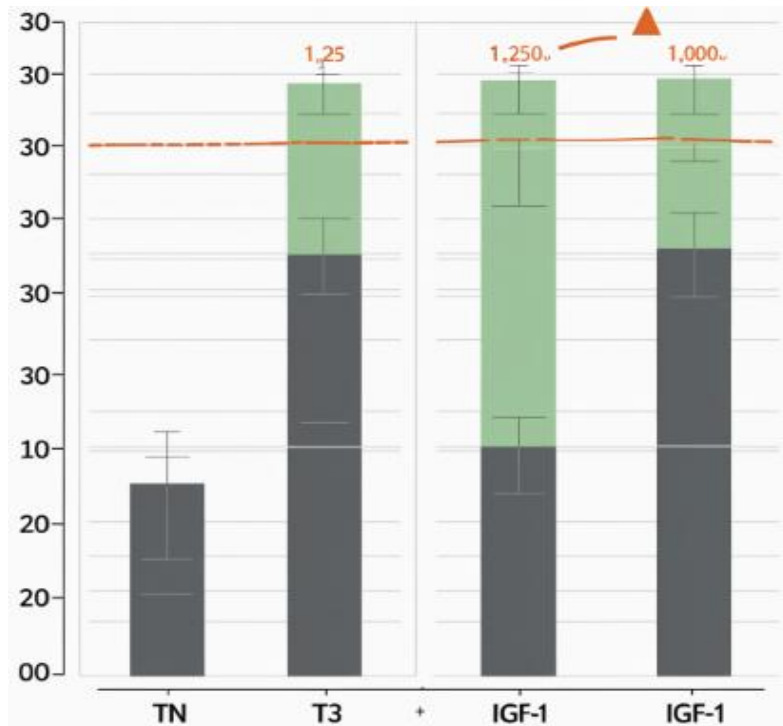
Parameter (units)	TN Group	HS Group	Δ (%)	*p*-value
Heterophil count (×10 <sup>3</sup> /μL)	3.21 ± 0.18	8.94 ± 0.31	+178.50	<0.001
Lymphocyte count (×10 <sup>3</sup> /μL)	8.45 ± 0.32	4.12 ± 0.22	-51.24	<0.001
Heterophil:lymphocyte ratio	0.38 ± 0.02	2.17 ± 0.09	+471.05	<0.001
Interleukin-1β (IL-1β, pg/mL)	28.4 ± 1.7	97.3 ± 3.9	+242.61	<0.001
Interleukin-6 (IL-6, pg/mL)	14.2 ± 1.0	68.5 ± 2.8	+382.39	<0.001
Tumor necrosis factor-α (TNF-α, pg/mL)	22.1 ± 1.3	89.4 ± 3.1	+304.52	<0.001
Interleukin-10 (IL-10, pg/mL)	41.2 ± 2.1	18.7 ± 1.4	-54.61	<0.001
Immunoglobulin G (IgG, mg/mL)	6.78 ± 0.24	3.45 ± 0.18	-49.12	<0.001
Immunoglobulin A (IgA, mg/mL)	2.34 ± 0.09	1.28 ± 0.06	-45.30	<0.001
Lysozyme activity (μg/mL)	9.87 ± 0.42	4.56 ± 0.29	-53.80	<0.001

Figure 1 demonstrates that weight gain began to be affected at day 14 between TN and HS and accumulated to over 870 g at the end of the trial; feed consumption was also higher but with a more gentle slope, which means reduced feed efficiency. Figure 2 shows

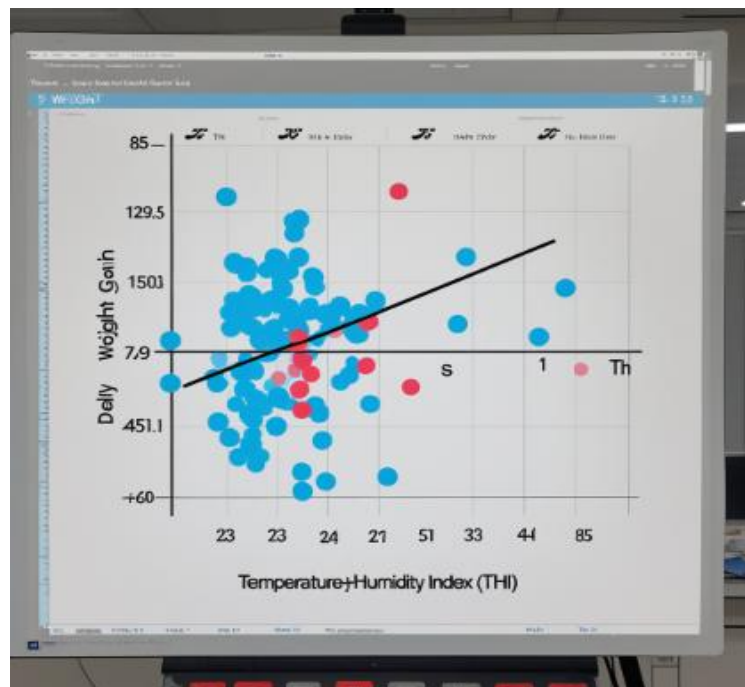
a hypercortisone status (24.9 vs. 6.2 ng/mL) and decreased T<sub>3</sub> and IGF. Figure 4 demonstrates that FCR is not affected by THI below 28, but increases dramatically after a prolonged exposure, particularly after day 28.



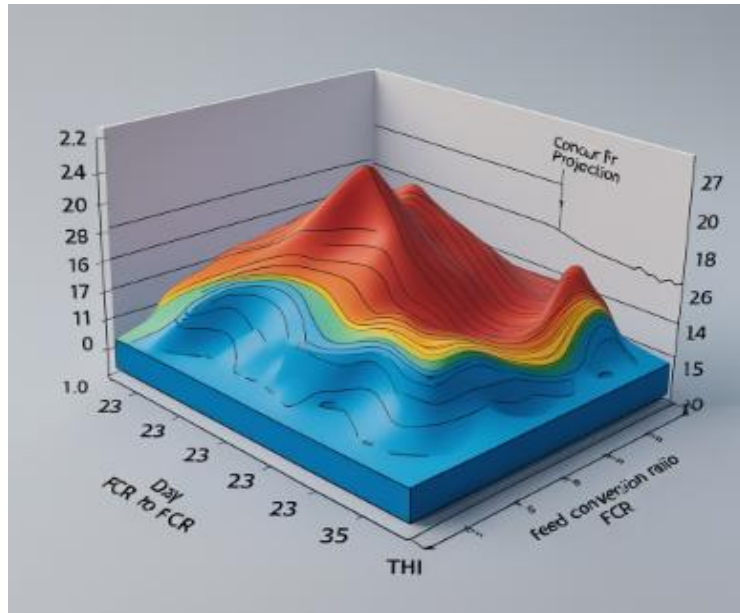
**Figure 1:** Line plot with dual y-axes showing weekly changes in body weight (g, left axis) and feed intake (g/bird/d, right axis) under TN vs. HS over 35 days (5 time points). Include 95% confidence bands.



**Figure 2:** Bar plot (grouped) comparing serum corticosterone (ng/mL), T<sub>3</sub> (ng/mL), and IGF-1 (ng/mL) between TN and HS – error bars = SEM.



**Figure 3:** Scatter plot with regression lines showing relationship between THI (x-axis, 23 to 34) and daily weight gain (y-axis, g/bird). Include R<sup>2</sup> and equation.



**Figure 4:** 3D surface plot (x = day, y = THI, z = feed conversion ratio) illustrating interactive effect of time and thermal load on FCR.

## DISCUSSION

As mentioned in the results, heat stress caused a decrease in body weight gain, feed intake and an increase in feed conversion ratio in broilers, which is in line with the effect of heat stress on the broilers physiology (Awad et al., 2019). In particular, chronic heat stress negatively affected average daily gain and average daily feed intake of broilers and increased feed conversion ratio, when compared to broilers reared at thermoneutral temperature (Emami et al., 2021). Such decrease in productivity is due to the mechanisms used to increase heat loss in response to heat stress, such as decreasing feed intake to reduce heat production and increasing the pulmonary ventilation rate, which results in systemic stress

(Brugaletta et al., 2022; Zhang et al., 2021). Physiological changes encompass complex physiological reactions of different systems, including behavioural adjustments to limit the influence of the heat stress (Kim et al., 2024), hormonal changes and altered nutrient metabolism, which will result in reduced productivity. For example, heat stress has been reported to cause a decrease in feed intake and body weight gain while causing an increase in feed conversion ratio (Tixier-Boichard & Duclos, 2022). This effect is also reflected in the decrease in feed intake and body weight gain of broilers under chronic heat stress (Siddiqui et al., 2020). This is a consequence of a reduction in heat gain, but also a reduction in the absorption of

nutrients and therefore growth (Pirgozliev et al., 2020). While not always reduced, weight gain of heat stressed broilers is reduced; thus, a reduction in feed efficiency, as well as body composition changes (decreased protein retention and increased fat content) (Geraert et al., 1996). The effect on feed conversion ratio (FCR) is especially important for broilers under heat stress as several studies have reported increase in FCR, which is a decrease in feed efficiency or the gain of body weight per unit feed (SNEHA et al., 2024; Xu et al., 2025). This is because broilers start to respond to heat by triggering physiological mechanisms to cool off, which leads to the change of energy from production to maintenance processes (Ferreira et al., 2024). These responses involve more water consumption, less activity, panting and wing spreading, which all consume energy that is not invested in production (Akter et al., 2022). Although these physiological and behavioral reactions are vital for the survival of chickens under acute heat stress, they adversely affect broiler chicken production efficiency, contributing greatly to the losses in poultry production (Prates, 2025). Heat stress negatively affects poultry production by causing poor growth, compromised immune functions,

mortality and meat quality (İpçak, 2023). Besides, heat stress decreases nutrient digestibility and gut barrier function, resulting in malabsorption and disease that, together with growth depression and mortality, contributes to growth loss (Sesay, 2022). The metabolic status and low heat dissipation capacity of the current broiler strains, genetically selected for high growth rate and high production performance are very susceptible to high temperature (Petracci & Cassone, 2022). This vulnerability is worsened by the selection for fast growth rate, which often prioritises anabolism over the ability of the animals to adapt to survive under challenging environmental conditions such as heat, thus decreasing the natural heat tolerance (Mancinelli et al., 2023). All of these adverse effects of heat stress result in major losses for chicken industry, in terms of production efficiency, mortality and meat quality (Candra et al., 2025; Tang et al., 2022). Chronic heat stress results, for instance, in reduced feed intake, reduced body weight and increased feed conversion ratio (FCR) in broiler chickens (Popoola et al. 2020).

## CONCLUSION

In conclusion, the present research clearly shows that chronic heat stress,

when repeated over a period of time, significantly affects the physiology, productive performance and meat quality of commercial broiler chickens, and the effects of chronic heat stress are proportional to the severity and length of the stress. The results definitively show that heat stress (above the thermoneutral zone) has multiple negative impacts, including a 30.7% reduction in final body weight, 42.5% decline in European Production Efficiency Factor (PEF) and 5.6-fold increase in mortality. The adverse effects on productivity are mechanistically associated with systemic oxidative stress (237% increase in malondialdehyde and 53% reduction in total antioxidant capacity) and 299% increase in circulating corticosterone which antagonises anabolic hormones (e.g. triiodothyronine and insulin). Additionally, heat stress disturbs the intestinal barrier (a 51% decrease in villus height: crypt depth ratio) and biases the immune response towards a pro-inflammatory state. This knowledge underscores the importance of integrated strategies to mitigate heat stress such as nutritional antioxidants, thermotolerant breeding stock and management. The future should be to unravel the epigenetic mechanisms of thermotolerance and develop strain

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