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Growth Performance, Blood Indices and Hormonal Responses of Broiler Chickens Fed Monosodium Glutamate

Research Article

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ABSTRACT

Effects of dietary monosodium glutamate on the performance, hormonal profiles, haematological, and serum biochemical indices of broilers were studied. Three hundred (300) day-old unsexed Abor – acre broilers were randomly allotted into six groups containing varied levels of monosodium glutamate (MSG) (0.00, 0.25, 0.50, 0.75, 1.00, and 1.25 g/kg diet) for 8 weeks. Each group was replicated five times with ten birds per replicate. Feed intake was significantly ($P<0.05$) increased at 0.75 and 1.00 g MSG/kg diet. MSG above 0.50 g/kg diet significantly ($P<0.05$) reduced the total weight gain. Inclusion at 1.00 and 1.25 g/kg diet significantly ($P<0.05$) reduced the packed cell volume, red blood cells, and haemoglobin counts. However, the mean corpuscular volume and mean corpuscular haemoglobin at 1.25 g MSG/kg diet were significantly ($P<0.05$) elevated. The varying inclusion levels did not significantly ($P>0.05$) influence the differential white blood counts and blood viscosity. Albumin, total protein, and high-density lipoprotein cholesterol levels were significantly ($P<0.05$) lowered from an inclusion level of 0.75 g MSG/kg diet while a significant ($P<0.05$) increase was observed in the serum urea and low-density lipoprotein cholesterol levels at the same inclusion rate. Inclusion of 1.00 and 1.25 g MSG/kg diet significantly ($P<0.05$) elevated the serum corticosterone concentration while there was a significant ($P<0.05$) decrease in other hormonal responses at the same level of inclusion. The result indicated that the inclusion of MSG up to 0.50 g/kg could be utilized in broiler feeds to enhance palatability and performance without any deleterious effects on the birds.

KEY WORDS chickens, enzymes, hormones, lipids, monosodium glutamate.

INTRODUCTION

Efficient animal production is achieved by good management practices whereby the provision of good quality feed in the right proportion is crucial. Several feed additives have been adopted in the poultry industry to enhance the performance and well-being of birds; one of which is flavoring agents. Flavoring agents are supplements added to the ration to enhance feed intake as they improve the palatability and in turn, the acceptability of feeds (Jay *et al.* 2010). One of these numerous flavor enhancers is monosodium glutamate (MSG) (Veronika and Daniela, 2013). Monosodium glutamate is safe for consumption with no specified average daily intake (Samuels, 1999) as the glutamate component of MSG is a bounteous non–essential amino acid in the blood plasma (Tapiero *et al.* 2002; Bartell and Batal, 2007). Diet composition has been explained to affect energy balance and / or energy-related metabolic pathways and consequently leading to an increase in body weight (Zemel, 2004). Similarly, the potential of MSG to induce weight gain has been linked with altered regulatory mechanisms that affect fat metabolism and also have an

effect on the hormones that influence weight gain (Hermanussen and Tresguerres, 2003). Therefore, using MSG in the diets of broiler chickens which are fast-growing birds will enhance them attaining market weight early. On the other hand, MSG has been reported to alter the behavior and to cause suppressed blood count in the male albino rat as well as reduced hemoglobin percentage (Hb), red blood cells (RBC), and white blood cell count (WBC) in female Swiss albino mice (Gasem, 2016). Several studies have highlighted the effects of MSG on serum enzymes, metabolites, proteins, hormones, and cholesterol in rat models but there is paucity of information in broiler chickens as far as the effects of MSG on serum biochemical and hormonal indices are concerned. Okediran *et al.* (2014) reported a significant increase in serum transaminases, total plasma cholesterol, and low-density lipoprotein cholesterol at a higher dose of 1.0 g of MSG. Therefore, carrying out this study on the blood parameters of broiler chickens is very expedient to ascertain the physiological and pathological state of the birds when fed with dietary MSG. For instance, increased blood viscosity is linked with increased resistance to blood flow and thereby increases the work of the heart and impairs organ perfusion. Hence, the objective of the study was to determine the possible effects of MSG inclusion on the heamatological, serum biochemical, and hormonal responses of broiler chickens fed with varying levels of dietary MSG.

MATERIALS AND METHODS

Experimental design and animals

A total of three hundred (300) day-old, unsexed Arbor–acre broiler chicks were used for the experiment which lasted for 8 weeks at the Poultry Unit of the livestock section of Teaching and Research Farm, The Federal University of Technology, Akure. The geographical coordinates of the location are between 7˚ 17' North and 5˚ 9' East with the rainfall of about 1524 mm per year and atmospheric temperature range of 28 ˚C to 31 ˚C and mean annual relative humidity of about 80% (Adu *et al.* 2017). The study was undertaken with approval from the institutional ethics committee of the institution for the care and use of animals for research. It was also conducted in accordance with the research ethics and guidelines of the Animal Production and Health Department of the institution (FUTA/APH/15/4750). On arrival of the chicks, they were weighed and randomly assigned to the six (6) treatment groups: A, B, C, D, E, and F containing 0.00 (control), 0.25, 0.50, 0.75, 1.00, and 1.25 g MSG/kg diet respectively in a completely randomized design (CRD). All the treatments were replicated five times with 10 birds per replicate.

The birds were fed with broiler starter (Table 1) and finisher (Table 2) diets *ad libitum* from 0-4 weeks and 5-8 weeks respectively.

Performance of broilers

Total weight gain of the birds was determined as additions of the weekly weight gains throughout the experimental period.

The feed conversion ratio (FCR), protein intake (PI), protein utilization (PU) and energy intake (EI) were determined as reported by Rabie *et al.* (2017) thus:

FCR= total feed intake (g) / weight gain (g)
PI (g/bird)= feed intake (g) × feed crude protein (%) / 100
PU= crude protein intake (g) / weight gain (g)
EI (Kcal/ME/bird)= feed intake (g) × feed metabolizable energy (Kcal/ME) / 1000

Blood sampling

On the 56th day of the experiment, five (5) birds per replicate were randomly selected and fasted overnight. Blood samples were collected from the jugular veins of the fasted birds into both heparinized tubes for determination of haematological parameters and dry clean plain centrifuged glass tubes for determination of serum biochemical indices. Blood samples for serum analyses were left for 15 mins at room temperature, after which tubes were centrifuged for 10 minutes at 3000 rpm to obtain a clear supernatant serum. The serum samples collected were kept frozen at -20 ˚C until the determination of serum hormones, enzymes, metabolites, lipids, and proteins.

Hematological parameters measurements

Packed cell volume (PCV, %) was determined by the micro hematocrit method, haemoglobin concentration (Hb) was estimated using the cyanmethaemoglobin method (Cannan, 1958) while red blood cell (RBC, cells x10^6mL^{-1}) and white blood cell counts (cells×10^3mL^{-1}) were determined using a haemocytometer with the improved Neubauer slide (Douglas and Harold, 2004). Mean corpuscular volume (MCV, µm3), mean corpuscular hemoglobin (MCH, pg), and mean corpuscular hemoglobin concentration (MCHC, g/dL) were calculated from PCV, RBC, and Hb using equations from Tazawa *et al.* (2011). Leucocyte differential counts (heterophils, lymphocytes, eosinophils, basophils, and monocytes) were carried out on blood smears stained with May-Grunwald–Giemsa stain. Erythrocyte sedimentation rate (ESR) was determined by the Westergren method (Ritchie *et al.* 1994) from blood diluted with 4 parts citrate solution (3.3% sodium citrate) allowed to stand in Westergren tube.

Table 1 Ingredient composition of the broiler starter diets at 0-4 weeks

Ingredients	Control	Inclusion level of MSG (kg)				
		0.25	0.50	0.75	1.00	1.25
Maize	510.00	510.00	510.00	510.00	510.00	510.00
Soybean meal	150.00	150.00	150.00	150.00	150.00	150.00
Groundnut cake	150.00	150.00	150.00	150.00	150.00	150.00
Fish meal (72%CP)	50.00	50.00	50.00	50.00	50.00	50.00
Corn bran	106.00	105.75	105.50	105.25	105.00	104.75
Bone meal	15.00	15.00	15.00	15.00	15.00	15.00
Limestone	10.00	10.00	10.00	10.00	10.00	10.00
Salt	3.50	3.50	3.50	3.50	3.50	3.50
MSG	0.00	0.25	0.50	0.75	1.00	1.25
Lysine	1.00	1.00	1.00	1.00	1.00	1.00
Methionine	2.00	2.00	2.00	2.00	2.00	2.00
Broiler premix	2.50	2.50	2.50	2.50	2.50	2.50
Total	1000.00	1000.00	1000.00	1000.00	1000.00	1000.00
Calculated nutrients						
Metabolizable energy (Kcal/kg)	2985.82	2977.24	2968.66	2960.08	2951.5	2925.76
Crude protein (%)	22.60	22.58	22.56	22.24	22.22	22.15
Calcium (%)	1.28	1.28	1.28	1.28	1.28	1.28
Phosphorus (%)	0.52	0.52	0.52	0.51	0.51	0.51
Lysine (%)	1.15	1.15	1.15	1.15	1.15	1.15
Methionine (%)	0.56	0.56	0.56	0.56	0.56	0.56
Crude fibre (%)	3.68	3.68	3.67	3.66	3.66	3.65

* Composition of premix: 2.5 kg of premix contains: vitamin A: 10000000 IU; vitamin D_3: 2500000 IU; vitamin E: 12000 IU; vitamin B_1: 2000 mg; vitamin B_6: 1500 mg; vitamin B_{12}: 10 mg; vitamin K_3: 2000 mg; Niacin: 15000 mg; Biotin: 20 mg; Folic acid: 600 mg; Panthothenic acid: 7000 mg; Chlorine chloride: 150000 mg; Manganese: 80000 mg; Iron: 40000 mg; Copper: 10 mg; Zinc: 60000 mg; Selenium: 150 mg; Iodine: 1000 mg; Magnesium: 100 mg; Ethoxyquine: 500 g and BHT: 700 g.

Table 2 Ingredient composition of the broiler finisher diets at 4-8 weeks

Ingredients	Control	Inclusion level of MSG (kg)				
		0.25	0.50	0.75	1.00	1.25
Maize	500.00	500.00	500.00	500.00	500.00	500.00
Soybean meal	130.00	130.00	130.00	130.00	130.00	130.00
Groundnut cake	130.00	130.00	130.00	130.00	130.00	130.00
Fish meal	0.00	0.00	0.00	0.00	0.00	0.00
Rice bran	133.00	132.75	132.50	132.25	132.00	131.75
Corn bran	60.00	60.00	60.00	60.00	60.00	60.00
Bone meal	25.00	25.00	25.00	25.00	25.00	25.00
Limestone	10.00	10.00	10.00	10.00	10.00	10.00
Salt	3.50	3.50	3.50	3.50	3.50	3.50
MSG	0.00	0.25	0.50	0.75	1.00	1.25
Lysine	3.00	3.00	3.00	3.00	3.00	3.00
Methionine	3.00	3.00	3.00	3.00	3.00	3.00
Broiler premix	2.50	2.50	2.50	2.50	2.50	2.50
Total	1000.00	1000.00	1000.00	1000.00	1000.00	1000.00
Calculated nutrients						
Metabolizable energy (Kcal/kg)	2964.16	2955.58	2947	2938.2	2929.84	2921.26
Crude protein (%)	18.56	18.54	18.52	18.5	18.48	18.46
Calcium (%)	1.24	1.24	1.23	1.23	1.23	1.23
Phosphorus (%)	0.56	0.56	0.56	0.55	0.55	0.55
Lysine (%)	1.08	1.08	1.08	1.08	1.08	1.08
Methionine (%)	0.57	0.57	0.57	0.57	0.57	0.57
Crude fibre (%)	4.85	4.83	4.83	4.82	4.82	4.81

* Composition of premix: 2.5 kg of premix contains: vitamin A: 10000000 IU; vitamin D_3: 2500000 IU; vitamin E: 12000 IU; vitamin B_1: 2000 mg; vitamin B_6: 1500 mg; vitamin B_{12}: 10 mg; vitamin K_3: 2000 mg; Niacin: 15000 mg; Biotin: 20 mg; Folic acid: 600 mg; Panthothenic acid: 7000 mg; Chlorine chloride: 150000 mg; Manganese: 80000 mg; Iron: 40000 mg; Copper: 10 mg; Zinc: 60000 mg; Selenium: 150 mg; Iodine: 1000 mg; Magnesium: 100 mg; Ethoxyquine: 500 g and BHT: 700 g.

Blood viscosity determination

Blood viscosity (BV) was determined using the remaining blood after haematological measurements. It was well stirred to prevent sedimentation and 0.5 mL of blood was placed into the sample cup of a Wells-Brookfield Viscometer (Model LVTD-CP, Middleboro, MA, USA) and maintained at 38.0 ± 0.1 ˚C with an Isotemp 1006S recirculating water bath (Fisher Scientific, Waltham, MA, USA).

Serum proteins, metabolites, and enzymes analyses

The serum total protein (TP) was determined by Biuret method, globulin (GLB) was determined by bromocresol green method as described by Tietz (1995) and albumin (ALB) was calculated as the difference between the TP and ALB while creatinine, bilirubin, and urea were estimated by deproteinization and Urease-Berhelot colorimetric methods using a commercial kit (Randox Laboratories Ltd, UK). The serum enzymes: alanine transaminase (ALT), aspartate transaminase (AST), and alkaline phosphatase (ALP) were obtained using auto analyzing test kits from Randox Laboratories, Crumlin, UK. The results were expressed as mg/dL.

Serum lipids analyses

Total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) were assayed by standard enzymatic endpoint method (Roschlan *et al.* 1974), precipitation method (NCEP, 2001), and colorimetric method (Tietz, 1995) respectively using commercially available assay kit supplied by Randox Laboratories, Crumlin, UK. The serum very-low-density lipoprotein cholesterol (VLDL-C) and low-density lipoprotein cholesterol (LDL-C) were estimated by employing the formula postulated by Friedewald *et al.* (1972):

VLDL-C= TG / 5.

The LDL-C was estimated as the difference between TC and the sum of VLDL-C and HDL-C. LDL-C= T C-(VLDL-C+HDL-C). The results were expressed as mg/dL.

Serum hormonal assay

Serum insulin, corticosterone (CS), insulin-like growth factor (IGF), growth hormone (GH), triiodothyronine (T3), and thyroxine (T4) concentrations were determined by double-antibody radioisotope assay (RIA) using commercially available RIA kits (China Institute of Atomic Energy, Beijing, China) as described by (Darras *et al.* 1992).

Statistical analyses

The completely randomized experimental design (CRD) was used with the model equation:

$Y_{ij}= \mu + ai + e_{ij}$

Where:

Y_{ij}: any of the response variables.

μ: overall mean.

a_i: effect of the ith treatment (i=diets 1, 2, 3, 4 and 5).

e_{ij}: random error due to experimentation.

All data collected were subjected to One-Way analysis of variance (ANOVA) using SAS (2008) version 9.2. Duncan Multiple Range Test of the same software was used for means comparison where significant differences existed.

RESULTS AND DISCUSSION

Performance

The performance of the broiler chickens fed different inclusion levels of MSG are presented in Table 3. It was observed that the birds on the diets containing 0.25 and 0.50 g MSG/kg diet recorded a non-significant increase ($P>0.05$) in the feed intake when compared with the birds on the control diet while those on 0.75 and 1.00 g MSG/kg diet recorded significantly ($P<0.05$) higher feed intake. However, birds on 1.25 g MSG/kg diet showed the least ($P<0.05$) feed intake. Furthermore, the inclusion of 0.25 and 0.50 g MSG/kg significantly ($P<0.05$) increased the total weight gain (TWG) of the birds. However, it was observed that an increase in the inclusion level of MSG above 0.50 g MSG/kg diet translated to a corresponding and progressively significant ($P<0.05$) reduction in TWG. It was also discovered that the birds on diets containing 0.25, 0.50, and 1.25 g MSG/kg had the best ($P<0.05$) feed conversion ratio (FCR). The protein and energy intakes among the birds on the diet containing 0.75 g MSG/kg were found to be significantly ($P<0.05$) higher than values recorded among birds on other diets. Furthermore, birds on the diet containing 1.00 g MSG/kg recorded a significant ($P<0.05$) higher protein utilization (PU) than birds from other treatments. For broiler chickens, MSG inclusion level up to 0.50 g MSG/kg diet positively enhanced total weight gain, probably, as a result of the increase in feed intake observed while a drop in total weight gains was observed in birds fed 0.75 g MSG/kg diet and above though feed intake still maintained an upward trend up to 0.75 g MSG/kg diet inclusion level before a drop in feed intake was noticed when MSG inclusion was increased to1.00 and 1.25 per kg.

Haematology

The haematological indices (Table 4) showed that respective MSG inclusion at 1.00 and 1.25 g/kg diet significantly ($P<0.05$) reduced the PCV (20.50±0.65 and 19.25±0.95), RBC (1.72±0.08 and 1.48±0.25), and Hb (6.83±0.21 and 5.10±0.31) of the broiler chickens when compared with those on the control diet (30.50±0.29, 3.09±0.09 and

10.15±0.09 for PCV, RBC, and Hb, respectively) with birds on the diet containing 1.25 g MSG/kg recording the least significant (P<0.05) difference among means. However, significant (P<0.05) increases were observed in the values of MCV and MCH among the birds fed diets containing 1.25 g MSG/kg diet when compared with the birds on the control diets and their means values were numerically higher than those of birds on the other treatment diets. The varied inclusion levels of MSG used in the present study had no significant (P>0.05) influence on the MCHC, ESR, whole blood, serum, and plasma viscosities, as well as all the differential white blood, counts parameters

Serum biochemistry

The serum biochemical parameters, ALB, TP and HDL-C, (Table 5) were significantly (P<0.05) lowered respectively among the birds fed diets containing 0.75 (9.86±1.53, 19.06±1.05 and 74.57±0.01), 1.00 (9.66±1.93, 18.08±2.62 and 69.14±0.01) and 1.25 (7.58±0.77, 14.29±1.37 and 69.39±0.08) g MSG/kg when compared with the birds on the control diet for ALB, TP and HDL-C at 14.99±0.79, 27.52±3.96 and 84.23±0.02, respectively. The respective serum TG, urea, VLDL-C, and LDL-C were significantly (P<0.05) elevated among the broiler chickens fed diets containing 1.00 (87.89±0.06, 9.20±0.06, 17.58±0.01 and 57.83±0.01) and 1.25 (90.22±0.01, 10.00±0.06, 18.04±0.01 and 57.57±0.01) g MSG/kg diet when compared with the control (80.24±0.02, 7.10±0.07, 16.05±0.08 and 42.20±0.01). However, the varied inclusion levels of MSG did not significantly (P>0.05) affect the serum creatinine, bilirubin, TC, GLB, ALP, AST, and ALT concentrations. From the results of the present study, the serum albumin (ALB), globulin (GLB) and total protein (TP) levels among the birds across all the treatment diets were statistically (P>0.05) similar to that of the control except for those on the diets containing above 0.50 g MSG/kg respectively for ALB and TP which showed a significant (P<0.05) difference when compared with the birds on the control diets.

Hormonal responses

The hormonal response shown in Table 6 indicates that inclusion levels of 1.00 and 1.25 g MSG/kg significantly (P<0.05) elevated the serum corticosterone (CS) concentrations (597.67±1.45 and 644.00±4.65, respectively) when compared with that of the control birds (535.33±8.19). Furthermore, the significantly (P<0.05) reducing effects of MSG inclusions on insulin and growth hormone; insulin-like growth factor (IGF) and triidothyronine (T3) as well as thyroxine (T4) were observed among the birds fed diets containing MSG from 0.75; 1.00 and 1.25 g/kg respectively.

The results of this experiment showed statistical (P>0.05) similarities in serum corticosterone (CS) levels among birds on diets containing 0.25 to 0.75 g MSG/kg and the control group while there was an elevation in the mean values of those on diets containing 1.00 and 1.25 g MSG/kg.

The performance of birds in this study corroborated the report of Nosseir *et al.* (2012) who observed a significant increase in the body weight of rats fed MSG and Zhelyazkov (2018) who reported a significant weight gain in fish fed diets supplemented with 1% MSG. It equally agreed with the observation of Abd El-Aziz *et al.* (2014) an increase in feed intake and body weight gain were observed in rats fed graded levels of MSG. However, this study contradicts the findings of Khadiga *et al.* (2009) who reported a significant decrease in body weight gain in chicks fed 0.25 and 0.5% MSG. The decrease in final body weight observed in birds on diets 0.75 and 1.00 g MSG/kg despite higher feed intake when compared with other groups might be due to several reasons. The stress impact of the high inclusion rate of MSG in diets had been reported to cause increased energy expenditure which will eventually lead to reduced weight gain (Kondoh and Toril, 2008). According to Yamazaki (2011), reduction in body weight, as well as body length in MSG-obese rats despite increased feed intake, were reported to be due to depressed activity of growth and sex hormones. The reduction in weight gain could also be a result of the reduction in fat content and fat deposition occasioned by an increasing level of MSG inclusion (Amaduruonye *et al.* 2018).

The increase in feed intake in relation to the higher MSG inclusion level as observed in this study is indicative that MSG improves feed palatability and acceptability in birds, and therefore, stimulates the orosensory receptors in the oral cavity of the birds and positively influenced the appetite, thereby, inducing weight gain. This speculated that chickens have an orosensory perception system for umami taste (Yoshida *et al.* 2021).

The significant reduction in both feed intake and weight gain observed among the birds on the diet containing 1.25 g MSG/kg was indicative that high-level administration of MSG can induce terminal suppression of feed palatability and consumption broilers. This result is consistent with the finding of Abd El-Aziz *et al.* (2014) who explained that induced gastric mucosal damage due to a high level of MSG inclusion in diet can, consequently, lead to decreased feed intake and body weight gain. The observed best feed conversion ratio and feed efficiency among the broilers on diet 0.50 g MSG/kg in the present study is similar to the result of the work carried out by Rezaei *et al.* (2013) in post-weaning pigs but contradicted by the report of Khadiga *et al.* (2009).

Table 3 Performance of broilers fed diets with different levels of monosodium glutamate (MSG) at 0-8 weeks

Parameters	A (0.00)	B (0.25)	C (0.50)	D (0.75)	E (1.00)	F (1.25)	P-value
IW (g/bird)	40.54±0.08	40.56±0.38	40.52±0.48	40.52±0.55	40.50±0.38	40.35±0.51	0.9996^{ns}
FW (g/bird)	1820.00±59.90^{b}	2012.50±16.77^{a}	2087.50±50.31^{a}	1737.50±27.95^{b}	1600.00±0.00^{c}	1416.67±9.62^{d}	< 0.0001*
TWG (g/bird)	1779.46±59.82^{b}	1971.94±16.54^{a}	2046.98±49.86^{a}	1696.98±28.20^{b}	1559.5±0.38^{c}	1376.32±9.58^{d}	< 0.0001*
TFI (g/bird)	4595.00±55.92^{b}	4765.00±120.17^{b}	4970.10±79.38^{b}	5783.00±257.17^{a}	5421.40±114.94^{a}	3337.50±173.49^{c}	< 0.0001*
FCR	2.58±0.09^{bc}	2.42±0.05^{c}	2.41±0.20^{c}	3.41±0.10^{b}	3.48±0.07^{a}	2.42±0.14^{c}	< 0.0001*
PI (g/bird)	868.37±0.27^{e}	896.27±0.65^{d}	934.49±0.33^{c}	1085.55±0.54^{a}	1017.69±0.29^{b}	631.36±0.29^{f}	< 0.0001*
PU	0.49±0.00^{c}	0.45±0.00^{d}	0.55±0.01^{b}	0.53±0.00^{b}	0.65±0.00^{a}	0.46±0.00^{d}	< 0.0001*
EI (kcal/bird)	13628.65±0.63^{e}	14130.59±0.58^{d}	14738.63±0.66^{c}	17148.29±0.59^{a}	16076.05±0.65^{b}	9899.28±0.66^{f}	< 0.0001*

IW: initial weights; FW: final weights; TWG: total weight gain; TFI: total feed intake; FCR: feed conversion ratio; PI: protein intake; PU: protein utilization and EI: energy intake.
The means within the same row with at least one common letter, do not have significant difference (P>0.05).
* (P<0.05).
NS: non significant.

Table 4 Haematology of broiler chickens fed diets with different levels of monosodium glutamate (MSG)

Parameters	A (0.00)	B (0.25)	C (0.50)	D (0.75)	E (1.00)	F (1.25)
Haemogram						
PCV (%)	30.50±0.29^{ab}	28.50±0.29^{b}	31.25±1.31^{ab}	33.50±0.87^{a}	20.50±0.65^{c}	19.25±0.95^{c}
RBC (×10^{6} mm^{3})	3.09±0.09^{ab}	2.81±0.07^{b}	3.20±0.13^{ab}	3.44±0.08^{a}	1.72±0.08^{c}	1.48±0.25^{c}
MCHC (g/dL)	33.28±0.03	33.33±0.07	33.33±0.04	33.36±0.03	33.27±0.03	33.40±0.04
MCV (fl)	98.91±1.33^{b}	101.44±1.72^{b}	97.79±0.43^{b}	97.52±0.97^{b}	103.02±1.99^{ab}	111.95±1.18^{a}
MCH (pg)	32.91±1.09^{b}	33.81±0.53^{b}	32.88±0.36^{b}	32.53±0.33^{b}	34.31±0.68^{ab}	37.39±2.09^{a}
Hb (g/dL)	10.15±0.09^{ab}	9.50±0.12^{b}	10.43±0.44^{ab}	11.18±0.28^{a}	6.83±0.21^{c}	5.10±0.31^{c}
ESR (mm)	2.25±0.25	2.50±0.29	2.00±0.00	2.00±0.00	2.50±0.29	3.00±0.41
Blood viscosity (mPa. s)						
Whole	1.93±0.00	1.93±0.00	1.93±0.00	1.94±0.01	1.96±0.00	1.95±0.00
Serum	1.32±0.00	1.32±0.00	1.33±0.00	1.34±0.00	1.34±0.00	1.35±0.00
Plasma	1.04±0.00	1.04±0.00	1.03±0.00	1.03±0.01	1.06±0.00	1.06±0.01
Differentials WBC (%)						
Heterophils	23.25±2.17	22.25±1.89	22.00±1.47	21.98±1.47	22.50±2.25	21.50±2.02
Eosinophils	1.30±0.41	1.25±0.48	1.25±0.25	2.00±0.00	1.75±0.25	1.15±0.48
Basophils	2.75±0.48	3.00±0.41	2.50±0.29	2.25±0.25	3.00±0.41	2.00±0.00
Lymphocytes	68.25±2.39	64.75±3.35	64.00±2.48	64.25±1.97	69.75±1.03	63.25±1.38
Monocytes	1.25±2.06	1.20±1.22	1.30±0.58	1.28±0.63	1.43±0.48	1.15±1.55

PCV: packed cell volume; RBC: red blood cells; MCHC: mean corpuscular haemaglobin concentration; MCV: mean corpuscular volume; MCH: mean corpuscular haemoglobin; Hb: haemoglobin; ESR: erythrocyte sedimentation rate; mPa .s: one millipasacal-second and WBC: white blood counts.
The means within the same row with at least one common letter, do not have significant difference (P>0.05).
* (P<0.05).
NS: non significant.

Hence, the economic efficiency in the experimental diets containing 0.25 to 0.50 g MSG/kg exhibited a better economic conversion ratio than that of the other groups when the FCR, FE, EI, and PI have used viz a viz the total weight gain (TWG) as indicators for marginal profitability.

A decreased PCV observed among the birds on diets containing 1.00 and 1.25 g MSG/kg was suggestive of red blood cell loss which could be due to several reasons such as cell destruction, blood loss, or failure of bone marrow production induced by a high inclusion level of MSG. Therefore, the mean values of PCV, Hb, and RBC recorded among the birds on diets containing 1.00 and 1.25 g MSG/kg were below the reference values of 22.0-49.0; 7.0-15.1 and 2.0-4.0 respectively, Harrison and Lightfoot (2005) for broiler chickens. This signified that the birds affected were anaemic. Other signs of anaemia in chickens are paleness, anorexia, depression, reduced weight gain, and high mortality rate. In this study, anorexia (reduced feed intake) which was observed among birds on diet containing 1.25 g/kg MSG and reduced weight gain among birds on diets 1.00 and 1.25 g/kg MSG could be a result of their anaemic conditions. The result, therefore, supported the finding of Abdulsalam *et al.* (2017) who also concluded that sub-chronic oral monosodium glutamate administration led to adverse effects on the haematological profiles of rats which ultimately resulted in anaemia. Salmanzadeh *et al.* (2020) also opined that the decrease in PCV, Hb, and RBC was due to high levels of MSG consumption could be brought about a generation of reactive oxygen species, that can cause lysis of the RBCs. This means inclusion beyond a tolerable range of 0.25 to 0.75 g MSG/kg diet is capable of inducing oxidative stress in the bone marrow of the broiler chickens resulting in the formation of micro-nucleated polychromatic erythrocytes.

Table 5 Serum biochemistry of broilers fed diets with different levels of monosodium glutamate (MSG)

Parameters	A (0.00)	B (0.25)	C (0.50)	D (0.75)	E (1.00)	F (1.25)	P-value
Serum protein (g/dL)							
Albumin	14.99±0.79^{a}	13.14±1.71^{ab}	12.91±3.81^{ab}	9.86±1.53^{bc}	9.66±1.93^{bc}	7.58±0.77^{b}	0.0176*
Globulin	13.53±2.75	13.00±1.68	12.86±3.25	9.20±3.25	8.42±1.91	6.71±1.91	0.3638^{ns}
TP	27.52±3.96^{a}	26.14±2.70^{ab}	25.77±1.55^{ab}	19.06±1.05^{b}	18.08±2.62^{bc}	14.29±1.37^{c}	0.0150*
Serum enzymes (U/L)							
ALP	133.26±1.29	127.28±2.78	133.16±0.50	131.29±0.66	127.89±2.97	131.72±1.00	0.1234^{ns}
AST	51.25±10.10	49.75±10.24	47.00±21.81	47.90±15.20	48.00±7.27	48.75±9.20	0.9699^{ns}
ALT	15.25±4.19	16.00±9.06	12.00±4.56	15.75±5.98	13.00±4.20	16.50±11.70	0.7576^{ns}
Serum lipids (mg/dL)							
TC	142.48±0.58	142.41±0.03	142.50±0.12	143.05±0.16	144.55±0.23	145.00±0.04	0.8330^{ns}
HDL-C	84.23±0.02^{a}	82.24±0.06^{ab}	82.01±0.02^{ab}	74.57±0.01^{b}	69.14±0.01^{c}	69.39±0.08^{bc}	< 0.0001*
TG	80.24±0.02^{b}	84.97±0.01^{ab}	85.88±0.00^{ab}	86.02±0.05^{ab}	87.89±0.06^{a}	90.22±0.01^{a}	< 0.0001*
VLDL-C	16.05±0.08^{b}	16.20±0.00^{b}	17.18±0.03^{ab}	17.20±0.00^{ab}	17.58±0.01^{a}	18.04±0.01^{a}	< 0.0001*
LDL-C	42.20±0.01^{c}	43.18±0.01^{bc}	43.31±0.01^{bc}	51.28±0.00^{b}	57.83±0.01^{a}	57.57±0.01^{a}	< 0.0001*
Serum metabolites (mg/dL)							
Creatinine	0.26±0.03	0.17±0.06	0.26±0.04	0.16±0.02	0.21±0.03	0.22±0.05	0.4443^{ns}
Bilirubin	2.60±0.23	4.57±1.36	4.09±1.17	2.63±0.69	3.35±0.38	3.24±0.80	0.5474^{ns}
Urea	7.10±0.07^{c}	7.60±0.06^{bc}	7.60±0.10^{bc}	8.40±0.05^{bc}	9.20±0.06^{ab}	10.00±0.06^{a}	< 0.0001*

TP: total protein; ALT: alanine transaminase; AST: aspertate transaminase; ALP: alkaline phosphatase; TC: total cholesterol; HDL-C: high density lipoprotein cholesterol; TG: triglyceride; VLDL-C: very low density lipoprotein cholesterol and LDL-C: low density lipoprotein cholesterol.
The means within the same row with at least one common letter, do not have significant difference (P>0.05).
* (P<0.05).
NS: non significant.

Table 6 Growth hormonal response of broilers fed different levels of monosodium glutamate (MSG)

Parameters	A (0.00)	B (0.25)	C (0.50)	D (0.75)	E (1.00)	F (1.25)	P-value
Corticosterone (ng/mL)	535.33±8.19^{c}	530.33±7.75^{c}	530.67±6.36^{c}	576.00±2.65^{cb}	597.67±1.45^{ab}	644.00±4.65^{a}	0.0008*
Insulin (µ/U/mL)	4.35±0.28^{a}	4.03±0.06^{ab}	3.99±0.06^{ab}	3.91±0.11^{b}	3.79±0.07^{b}	3.67±0.05^{b}	0.0449*
GH (ng/mL)	134.33±2.33^{a}	130.00±2.89^{a}	122.00±1.53^{ab}	115.33±1.45^{bc}	111.67±2.73^{c}	111.67±2.03^{c}	< 0.0001*
IGF (ng/mL)	29.97±0.27^{a}	29.83±0.28^{a}	28.17±0.53^{ab}	27.13±0.55^{ab}	24.73±0.38^{c}	23.57±0.38^{c}	< 0.0001*
T3 (ng/mL)	1.74±0.04^{a}	1.78±0.05^{a}	1.79±0.02^{a}	1.74±0.03^{a}	1.60±0.01^{b}	1.55±0.04^{b}	0.0013*
T4 (nmol/L)	16.24±0.01^{a}	16.21±0.04^{a}	16.26±0.03^{a}	16.18±0.09^{a}	15.97±0.19^{a}	15.52±0.25^{b}	0.0147*
T3:T4	0.11±0.00^{ab}	0.11±0.00^{a}	0.11±0.00^{ab}	0.11±0.00^{ab}	0.10±0.00^{cb}	0.10±0.00^{c}	0.0210*

GH: growth hormone; IGF: insulin-like growth factor; T3: triidothyronine and T4: thyroxine.
The means within the same row with at least one common letter, do not have significant difference (P>0.05).
* (P<0.05).
NS: non significant.

Another study recorded similar trends with a high dose of MSG and concluded the decrease in PCV was due to the direct toxic effect of a high dose of MSG on the haemato-poietic stem cells of the bone marrow interfering with erythropoiesis (Ibukun *et al.* 2015). Based on the explanation made by George and Kumaran (2016), the observed decrease in Hb in the MSG-treated birds fed diets containing 1.00 and 1.25 g MSG/kg could be attributed to lysis of RBC caused by exposure to a high dose of MSG. This study further revealed that the inclusion levels of MSG up to 1.00 g/kg diet did not have a significant adverse effect on the MCV and MCH of the birds. It was those on diet containing 1.25 g MSG/kg that recorded a significant increase when compared with birds on the control and other diets. Elevated MCV and MCH are suggestive of macrocytic anemia in the birds on diet containing 1.25 g MSG/kg (Ashaolu *et al.* 2011). This condition occurs when the blood cells are too big (macrocytic cells) and this could be a sign that a high dose of MSG inclusion induced vitamin B12 or folic acid deficiency in the birds. The result of this finding was consistent with the reports of George and Kumaran (2016) as well as Abdulsalam *et al.* (2017). Hence, this revealed that the immune statuses of the broilers were compromised by the experimental diet containing 1.25 g MSG/kg.

However, the MCHC and ESR of the birds across all the treatment diets were not significantly affected. Erythrocyte sedimentation rate (ESR) indirectly measures the degree of inflammation present in the body. It measures the rate of fall (sedimentation) of erythrocytes (red blood cells) in a sample of blood. The non-significant difference recorded in ESR in this study proved there was no presence of inflam-

mation caused by the varied inclusion levels of MSG. A lower than normal ESR has been reported to be a result of a disease or condition that increases red blood cell production, white blood cell production, or production of abnormal red blood cells (sickle cell anemia) (Marx *et al.* 2013).

Furthermore, the study revealed that the white blood cells (WBC) differential counts did not show any significant difference across all the treatment diets. This showed the dietary MSG had no significant effect on lymphocyte, monocytes, heterophil, eosinophil, and basophil when compared with birds on the control diet. This explained that the bird's immunity was not impaired despite its anaemic condition and this finding agreed with the study of Newsholme *et al.* (2003) but was at variance with Ashaolu *et al.* (2011); Hellen *et al.* (2013) and Ghadhban (2017). The statistical similarities in WBC differential counts in the MSG-treated birds explained there were no observable metabolic changes induced by the MSG, thus, immunologic functions were not affected while the risk of infections was reduced. However, the possibility of cell lyses and bone marrow effect might be observed among the birds on diets containing 1.25 g MSG/kg if prolonged feeding on this diet is encouraged. Increased non-significant whole blood and plasma viscosities observed in birds fed the diet containing 1.00 g MSG/kg and above as well as increased serum viscosity on the diet containing 0.75 g MSG/kg could lead to increased risk of morbidity such as cardiovascular and cerebrovascular disease (Naghedi-Baghdar *et al.* 2018) in the broilers if prolonged feed at these inclusion levels is maintained. The results of the whole blood, plasma and serum viscosity recorded in this study suggested that the inclusion levels above 0.50 g MSG/kg diet in the diets could be hazardous to the health of the birds.

Albumin constitutes a large part of the TP fraction of serum and the ALB will increase when protein intake increases (Bertholf, 2014). This present experiment showed that the inclusion levels of MSG did not adversely influence the GLB levels among the hens across the dietary groups. The non-significant difference observed in the GLB indicates that the protein content of the diets was adequate and available and that MSG inclusion levels did not interfere with protein metabolism.

Since it has been established that abnormal levels of ALB indicate problems in the liver or kidney, and it can also indicate a lack of nutrients (Quinlan *et al.* 2010). The lowest ALB recorded among the birds on the diet containing 1.25 g MSG/kg is suggestive of the adverse impact of the high dose of MSG on these organs. This study agreed with Oladipo *et al.* (2015) in that they documented a decrease in the activity levels of the ALB and TP in response to an increase in the inclusion of MSG. The Liver is also the primary site of the synthesis of blood proteins. The lowest concentration of TP recorded among the birds on diet 1.25 g MSG/kg is indicative of disturbance of protein synthesis in the liver which resulted as a consequence of impaired hepatic function and consequently led to a decrease in their TP concentration (Okediran *et al.* 2014). The significant reduction in the TP concentration among birds on diets above 0.75 g MSG/kg could indicate a reduction in the synthetic function of the liver or an increased rate of protein degradation in response to a high dosage of MSG in the diets.

Furthermore, the dietary MSG did not have any noticeable effect on the serum total cholesterol (TC) and this corroborated the findings obtained by Khadiga *et al.* (2009) and Ayazi (2014). There was also consistency between the result of this study and the finding of Inuwa *et al.* (2011) as far as variation in TC level was a concern. They equally recorded insignificant differences within the groups but the group treated with the highest dose had the most elevated total cholesterol level, though not significant in this study, which indicated possible interference with fat metabolism. The decrease in the serum high-density lipoprotein cholesterol (HDL-C) observed in the present study among the birds fed diets containing 0.75 g MSG/kg and above was in agreement with the findings of Alwaleedi (2016) but disagreed with El Malik and Sabahelkhier (2019) who reported an increase in serum HDL-C at an increasing dosage of MSG above 0.50 g in rats as well as Sani *et al.* (2015) who reported a non-significant influence of MSG on serum HDL-C of rats at an elevated dosage above 0.50 g.

The recorded significant increase in the serum levels of triglyceride (TG) and very low lipoprotein cholesterol (VLDL-C) among the birds on the diet containing 1.25 g MSG/kg when compared with the control and low-density lipoprotein cholesterol (LDL-C) among the birds fed diets containing from 0.75 g MSG/kg were consistent with the findings of Alwaleedi (2016) and El Malik and Sabahelkhier (2019). The increased levels of serum TC in response to an increasing level of MSG (though not significant in this study), are indicative of impairment of cholesterol metabolism and could result in coronary heart disease in broiler chickens. The recorded elevated level of serum TG and VLDL-C is suggestive of hyperlipoproteinemia and this might be due to the excessive dietary intake of fat and carbohydrates beyond the body's need as induced by excessive consumption by the MSG containing feed. This must have led to their hepatic conversion to TG, which is later converted into VLDL-C for export to the various tissues (Nduka, 1999). The increased activities of lipoxygenase and lipid peroxidation products have been explained to be stimulated by hyperglycemia (Ahluwalia and Singh, 2002). It has been documented that exposure to a high dosage of MSG increased oxidative stress and then could stimulate

the oxidation of LDL-C (Singh *et al.* 2011). Therefore, the increased levels of serum cholesterol and other lipid fractions in response to an increasing level of MSG may predispose broilers to health risks such as atherosclerosis, coronary heart disease, and diabetes mellitus (Kondoh and Torii, 2008).

The transaminases, present in the liver, are released into the bloodstream due to hepatocellular damage, making them a sensitive marker of liver damage (Al-Mamary *et al.* 2002). Hepatic cell damage is characterized by a significant rise in plasma enzyme activities such as alanine aminotransferase (ALT), aspertate aminotransferase (AST), and alkaline phosphatase (ALP) and thus causes alterations in liver function (Kim *et al.* 2008). It is ALT that is employed as a marker enzyme in the cytosol of hepatocytes and its level within limits is indicative of hepatocellular damage and that can, therefore, provide a quantitative assessment of the degree of damage done to the liver (Aniagu *et al.* 2004; Okediran *et al.* 2014). Ewuola *et al.* (2008) also opined that elevated serum enzyme activities of AST, ALT and ALP are an indication of heart, kidney, and/or liver damage due to cellular destruction caused by toxins. This investigation revealed that there was no sign of hepatocellular damage induced by MSG among the treated birds. This result was contrary to the findings of Inuwa *et al.* (2011) who reported hepatocellular effects in Wistar rats treated with 200 to 400 mg MSG/kg body weight, as well as Onyema *et al.* (2006) and Egbuonu *et al.* (2009), reported an increase in the serum aminotransferases in male albino rats fed a high dose of MSG. This present study did not validate their claim that ALP was significantly increased by MSG which indicated that the hepatic capacity of the liver is grossly affected by MSG. The variance in the two studies could be a result of differences in animal species, variation in dosage applied, or length of exposure to MSG consumption.

This investigation did not show any significant difference in the serum creatinine and bilirubin levels within the MSG treated birds when compared with the control. Elevation in the concentration of serum creatinine as well as urea is a sign of a decline in the ability of the kidney to filter fluid within the body (Edwards and Bouchier, 1991). This means there was no recorded sign of renal impairment among the birds used for this study irrespective of the inclusion levels of MSG used. Prolonged feeding on diets containing 0.75 g MSG/kg and above could predispose the birds to renal impairment as a significant increase in serum urea concentration was noted among these birds. Elevated levels of serum total bilirubin indicate liver damage or disease. The non-significant difference in the total bilirubin concentrations observed in this study validated the result that normal liver function was not impaired by the inclusion levels used in this experiment.

The elevated CS level of birds on the two diets is indicative of acute stress in the birds leading to increased CS production in the adrenal glands with a lower degradation rate in the liver (Thaxton and Puvadolpirod, 2000). The result reported by Macho *et al.* (1999) was in line with the present finding but contradicted the findings of Seo *et al.* (2010) who observed a significant decrease in the CS levels of MSG-treated rats when compared with the control group. However, since, it has been documented that sustained elevated levels of serum CS indicated a form of chronic stress (Post *et al.* 2003), the birds on diets containing 0.75 to 1.25 g MSG/kg could be said to be experiencing nutritional stress induced by an increased level of MSG.

Corticosterone had also been reported to promote the conversion of proteins and lipids into glucose during the stress-induced activation of the HPA (hypothalamic pituitary adrenal) axis (Dong *et al.* 2007; Gross and Cidlowski, 2008).

Therefore, this stress-induced high level of CS among the birds on diets containing 0.75 to 1.25 g MSG/kg will be accompanied by the mobilization of proteins and peripheral lipids, stimulation of hepatic gluconeogenesis, and increased central fat deposits in the birds as explained by Song *et al.* (2011) and the increased levels of serum lipids that had resulted would stimulate insulin biosynthesis and secretion (Obici and Rossetti, 2003). Stress-induced high plasma fatty acids and glucose are connected with insulin resistance in broiler chickens (Yuan *et al.* 2008) and this can explain the corresponding inverse level of insulin recorded among the birds.

The result of this study further showed that MSG inclusion levels of 0.25 and 0.50 g MSG/kg diet favored the elevation of serum GH and IGF hormones in the affected birds unlike those on diets containing 0.75 to 1.25 g MSG/kg. The observed reduction in the concentration of these hormones on diets containing above 0.50 g MSG/kg supported the claim that MSG induced GH regulatory deficit, especially at an inclusion level above 0.50 g MSG/kg diet (Millard *et al.* 1982). This result buttressed the reports of Hermanussen *et al.* (2006) and Malozowski *et al.* (1995) who observed a similar trend in MSG-treated rats given an increasing level of MSG per day.

MSG inclusion level in the broilers' diets up to 0.75 g/kg does not significantly affect the serum levels of T3, T4, and their ratio. A significant decline was, however, observed in their serum concentration when the MSG inclusion level was increased to 1.00 and 1.25 g/kg diet for T3 and T4 respectively. This is amplifying the effects of the high inclusion level of MSG on the hypophysis-thyroid axis. They have been reported to play key roles in the development, growth, and metabolism of broiler chickens (Yen, 2001; Brent, 2012).

CONCLUSION

The study established that the inclusion of dietary MSG up to 0.50 g/kg diet optimally enhanced performance and normal physiological functions in the broiler chickens. It could be concluded that higher doses of MSG above 0.50 g/kg diet adversely affected weight gain and hormonal balance while the inclusion of 0.25-0.50 g MSG/kg diet enhances improved hormonal functions such as lowered corticosterone level in the blood which reduced the stress on the bird and ensured better functioning of the growth hormone and hormones of the thyroid gland. . Furthermore, it was apparent that MSG does not have significant adverse effects on the PCV, Hb, and RBC when the inclusion level does not exceed 0.75 g MSG/kg diet in broiler chickens. However, a higher MSG inclusion level above 0.75 g/kg diet lowered the blood counts of the birds although their immunity was unaffected due to its non-significant effect on white blood cell constituents of the broiler chickens. The physiology of the bird could become compromised resulting in pathological conditions due to the anaemic state of the broiler chicken fed MSG above 0.75 g/kg diet. There is also every tendency of renal and hepatic toxicity induction at higher inclusion rates of MSG above 0.50 g/kg diet as indicated by decreased liver protein concentration and accumulation of urea in the blood.

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REFERENCES

Abd El-Aziz G.S., El-Fark M.O., Hassan S.M. and Badawoud M.H. (2014). Effects of prolonged oral intake of monosodium glutamate (MSG) on body weight and its correlation to stomach histopathological changes in male rats. *Thai J. Vet. Med*. **44 (2),** 201-208.

Abdulsalam H., Adamu S., Sambo S.J., Gadzama J.J., Chiroma M.A., Adeke J.T., Atata J.A. and Mohzo D.L. (2017). Haematological response of adult male wistar rats experimentally exposed to oral monosodium glutamate. *J. Vet. Appl. Sci.* **7(2),** 29-34.

Adu O.A., Olarotimi O.J., Olayode S.O. and Adelowo A.O. (2017). Effects of dietary supplementation of copper sulphate and copper oxide on some egg quality parameters of laying hens. *Slovak J. Anim. Sci.* **50(3),** 118-127.

Ahluwalia P. and Singh K. (2002). Alteration in lipid peroxidation, cytochrome P450, glutathione and its metabolizing enzymes upon msg administration in hepatic tissue of adult male mice. *Indian J. Toxicol.* **9,** 23-27.

Al-Mamary M., Al-Habori M., Al-aghbari A.M. and Basker M.M. (2002). Investigation into the toxicological effects of *Catha edulis* leaves. A short term study in animals. *Phytother. Res.* **16(2),** 127-132.

Alwaleedi S.A. (2016). Adverse effects of monosodium glutamate on serum lipid profile, cholesterol status and blood glucose in adult rats. *Res. J. Pharm. Biol. Chem. Sci.* **7(1),** 732-739.

Amaduruonye W., Ikwunze K., Oguike M.A. and Onunkwo D.N. (2018). Impact of ginger (*Zingiber officinale*) on intestinal, caeca microbial loads and growth performance of broilers. *Nigerian J. Anim. Sci.* **20(1),** 123-133.

Aniagu S.O., Nwinyi F.C., Akumka D.D., Ajoku G.A., Dzarma S., Izebe K.S., Ditse M., Nwaneri P.E.C., Wambebe C. and Gamaniel K. (2004). Toxicity studies in rats fed nature cure bitters. *African J. Biotechnol.* **4(1),** 72-78.

Ashaolu J.O., Ukwenya V.O., Okonoboh A.B., Ghazal O.K. and Jimoh A.A.G. (2011). Effect of monosodium glutamate on hematological parameters in wistar rats. *Int. J. Med. Med. Sci.* **3(6),** 219-222.

Ayazi M. (2014). The effect of dietary glutamine supplementation on performance and blood parameter, carcass characteristics, quality and characteristics meat of broiler chickens under continuous heat stress condition. *Int. J. Farm. Alli. Sci.* **3(12),** 1234 – 1242.

Bartell S.M. and Batal A.B. (2007). The effect of supplemental Glutamine on growth performance, development of the gastrointestinal tract and humoral immune response of broilers. *Poult. Sci.* **86,** 1940-1947.

Bertholf R.L. (2014). Protein and albumin. *Lab. Med.* **45(1),** 25-41.

Brent G. (2012). Mechanisms of thyroid hormone action. *J. Clin. Invest.* **122(9),** 3035-3043.

Cannan R.K. (1958). Book of Clinical Practical Chemistry. Publisher and Distributors, New Delhi, India.

Darras V.M., Visser T.J., Berghman R. and Kuhn E.R. (1992). Ontogeny of type I and type III deiodinase activities in embryonic and posthatch chicks: Relationship with changes in plasma triiodothyronine and growth hormone levels. *Comp. Biochem. Physiol.* **103,** 131-136.

Dong H., Lin H., Jiao H.C., Song Z.G., Zhao J.P. and Jiang K.J. (2007). Altered development and protein metabolism in skeletal muscles of broiler chickens (*Gallus domesticus*) by corticosterone. *Comp. Biochem. Physiol.* **147,** 189-195.

Douglas W. and Harold T. (2004). Small Animal Clinical Diagnosis by Laboratory Methods. Saunders and Elsevier Publisher, Philadelphia, Pennsylvania, USA.

Edwards M.B. and Bouchier A.D. (1991). Principle and Practice of Medicine. ELBS Churchill Living Stone. Man Group Ltd., Hong Kong, China.

Egbuonu A.C.C., Obidoa O., Ezeokonkwo C.A., Ezeanyika L.U.S. and Ejikeme P.M. (2009). Hepatotoxic effects of low dose oral administration of monosodium glutamate in male albino rats.

African J. Biotechnol. **8(13),** 3031-3032.

El Malik A. and Sabahelkhier M.K. (2019). Changes in lipid profile and heart tissues of wistar rats induces by using monosodium glutamate as food additive. *Intl. J. Biochem. Physiol.* **4(1),** 141-147.

Ewuola E.O., Ogunlade J.T., Gbore F.A. and Egbunike G.N. (2008). Serum biochemistry and organ traits of growing rabbits fed *Fusarium verticilloides* cultured maize-based diet. Pp. 199-203 in Proc. 33rd Nigerian Annu. Anim. Prod. Conf., Nigerian Society of Animal Production, Ago Iwoye, Nigeria.

Friedewald W.T., Levy R.I. and Fredrickson D.S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.* **18,** 499-502.

Gasem M.A. (2016). Effect of monosodium glutamate and aspartame on behavioral and biochemical parameters of male albino Mice. *African J. Biotechl.* **15(15),** 601-612.

George B. and Kumaran B. (2016). Effect of *Nigella sativa* oil against monosodium glutamate induced toxicity on haematological parameters in rats. *Intl. J. Recent Sci. Res.* **7(6),** 11592-11596.

Ghadhban R.F. (2017). Effects of monosodium glutamate on some hematological parameters in adult rats. *Indian J. Appl. Res.* **7(2),** 688-690.

Gross K.L. and Cidlowski J.A. (2008). Tissue-specific glucocorticoid action: a family affair. *Trends Endocrin. Metabol.* **9,** 331-339.

Harrison G.J. and Lightfoot T.L. (2005). Clinical Avian Medicine. Hardcover. Spix Publishing, Inc. Palm Beach, Florida, USA.

Hellen D.B., Miguel A., Areas P.B. and Felix G.R. (2013). Evaluation of biochemical, haematological and histological parameters in non-diabetic and diabetic Wister rats fed with Monosodium glutamate. *Food Nutr. Sci.* **4(1),** 66-76.

Hermanussen M. and Tresguerres J.A. (2003). Does the thrifty phenotype result from chronic glutamate intoxication? A hypothesis. *J. Perinat. Med.* **31(6),** 489-495.

Hermanussen M., García A.P., Sunder M., Voigt M., Salazar V. and Tresguerres J.A. (2006). Obesity, voracity, and short stature: the impact of glutamate on the regulation of appetite. *European J. Clin. Nutr.* **60(1),** 25-31.

Ibukun O.O., Monday T., Abiola S.O. and Oladele S.O. (2015). Haematological effect of ethanolic extract of *Uvaria chamae* on monososdium glutamate-induced toxicity in sprague-dewley rats. *Ann. Biol. Res.* **6(7),** 17-22.

Inuwa H.M., Aina V.O., Gabi B., Ola I.A. and Ja'afaru L. (2011). Determination of nephrotoxicity and hepatoxicity of monosodium glutamate (MSG) consumption. *British J. Pharmacol. Toxicol.* **2(3),** 148-153.

Jay Y.J., Joel M.D., Mike D.T., Robert D.G., Jim L.N., David G.R. and Steve S.D. (2010). Feed additives for swine: Fact sheets – flavors and mold inhibitors, mycotoxin binders and antioxidants. *J. Swine Health Prod.* **18(1),** 27-32.

Khadiga A.A., Ati S., Mohammed A.M. and Saad H.E. (2009). Response of broiler chicks to dietary monosodium glutamate. *Pakistan Vet. J.* **29(4),** 165-168.

Kim W.R., Flamm S.L., Di Bisceglie A.M. and Bodenheimer H.C. (2008). Serum activity of alanine aminotransferase (ALT) as an indicator of health and disease. *Hepatology.* **47,** 1363-1370.

Kondoh T. and Torii K. (2008). MSG intake suppresses weight gain, fat deposition, and plasma leptin in male Sprague-Dawley rats. *Physiol. Behav.* **95,** 135-144.

Macho L., Jezova D., Zorad S. and Fickova M. (1999). Post natal monosodium glutamate treatment results in attenuation of corticosterone metabolic rate in adult rats. *Endorin. Regul.* **33,** 61-67.

Malozowski S., Parmer T.G., Trojan S., Merriam G.R., Gibori G., Roberts C.T., Le Roith D., Werner H. and Zilberstein M. (1995). Growth hormone (GH) modulates insulin-like growth factor I (IGF-I) and type I IGF receptor mRNA levels in the ovary of prepubertal GH-deficient rats. *European J. Endocrin.* **132(4),** 497-501.

Marx J., Hockberger R. and Walls R. (2013). Rosen's Emergency Medicine Concepts and Clinical Practice. Elsevier, St. Louis, Missouri.

Millard W.J., Martin J.B., Audet J., Sagar S.M. and Martin J.B. (1982). Evidence that reduced growth hormone secretion observed in monosodium glutamate-treated rats is the result of a deficiency in growth hormone-releasing factor. *Endocrinology.* **110(2),** 540-550.

Naghedi-Baghdar H., Nazari S.M., Taghipour A., Nematy M., Shokri S., Mehri M.R., Molkara T. and Javan R. (2018). Effect of diet on blood viscosity in healthy humans: A systematic review. *Electro. Physics.* **10(3),** 6563-6570.

National Cholesterol Education Programme (NCEP). (2001). Third report of the NCEP on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. *J. American Med. Assoc*. **285(19),** 2486-2497.

Nduka N. (1999). Clinical Biochemistry for Students of Pathology. Animo Press Ltd, Nigeria.

Newsholme P., Joaquim P., Manuela M., Tania C.P. and Rui C. (2003). Glutamine and glutamate: Their central role in cell metabolism and function. *Cell Biochem. Funct.* **21,** 1-9.

Nosseir N.S., Ali M.H.M. and Ebaid H.M. (2012). A histological and morphometric study of monosodium glutamate toxic effect on testicular structure and potentiality of recovery in adult albino rats. *Res. J. Biol.* **2,** 66-78.

Obici S. and Rossetti L. (2003). Minireview: nutrient sensing and the regulation of insulin action and energy balance. *Endocrinology.* **144,** 5172-5178.

Okediran B.S., Olurotimi A.E., Rahman S.A., Michael O.G. and Olukunle J.O. (2014). Alterations in the lipid profile and liver enzymes of rats treated with monosodium glutamate. *Sokoto J. Vet. Sci.* **12(3),** 42-46.

Oladipo I.C., Adebayo E.A. and Kuye M.O. (2015). Effects of monosodium glutamate on ovaries of Female Aprague-Dawley rats. *Int. J. Curr. Microbiol. Appl. Sci.* **4(5),** 737-745.

Onyema O., Farombi E., Emerole G., Ukoha A. and Onyeze G. (2006). Effect of vitamin E on monosodium glutamate induced hepatotoxicity and oxidative stress in rats. *Indian J. Biochem. Biophys.* **43,** 20-24.

Post J., Rebel J.M.J. and ter Huurne A.A.H.M. (2003). Physiological effects of elevated plasma corticosterone concentrations in broiler chickens: An alternative means by which to assess the physiological effects of stress. *Poult. Sci.* **82,** 1313-1318.

Quinlan G.J., Martin G.S. and Evans T.W. (2010). Albumin: Biochemical properties and therapeutic potential. *Hepatology.* **41(6),** 1211-1219.

Rabie M.H., Sherif K.E., Abd El-Khalek A.M. and El-Gamal A.A.A. (2017). Effect of dietary energy and protein on growth performance and carcass traits of mamourah cockerels. *Asian J. Anim. Vet. Adv.* **12(3),** 142-151.

Rezaei R., Knabe D.A., Tekwe C.D., Dahanayaka S., Ficken M.D., Fielder S.E., Eide S.J., Lovering S.L. and Wu, G. (2013). Dietary supplementation with monosodium glutamate is safe and improves growth performance in post weaning pigs. *Amino Acids*. **44(3),** 911-923.

Ritchie B.W., Harrison J.G. and Harrison R.L. (1994). Avian Medicine. Winger's Publishing, Inc., Florida, USA.

Roschlan P., Bernet E. and Gruber W. (1974). Enzymatische bestimmung des gesamt choles terium in serum. *J. Clin. Biochem.* **12,** 403-407.

Salmanzadeh M., Ebrahimnezhad Y., Aghdam Shahryar H. and Ghiasighaleh Kandi J. (2020). The effects of *in ovo* administration of glutamine on hatchability, subsequent performance, digestive enzyme activities, immune response and some of blood parameter in broiler chickens. *Iranian J. Appl. Anim. Sci.* **10(3),** 535-545.

Samuels A. (1999). The toxicity/Safety of MSG: A study in suppression of information. *Account. Res.* **6,** 259-263.

Sani M.M., Bello A.B., Adam A.A., Abubakar H., Ahmad S. and Muhammad M.G. (2015). The effects of oral monosodium glutamate consumption on lipid profile of experimental rats. *Int. J. Ethnomed. Pharmacog.* **2(1),** 6-12.

SAS Institute. (2008). SAS®/STAT Software, Release 9.4. SAS Institute, Inc., Cary, NC. USA.

Seo H.J., Ham H.D., Jin H.Y., Lee W.H., Hwang H.S., Park A.A., Kim Y.S., Choi S.C., Lee S., Oh K.J., Kim B.S., Park B.R. and Lee M.Y. (2010). Chronic administration of monosodium glutamate under chronic variable stress impaired hypothalamic-pituitary-adrenal axis function in rats. *Korean J. Physiol. Pharmacol.* **14,** 213-221.

Singh K., Sharma J., Kaur A. and Ahluwalia P. (2011). Alteration upon oral ingestion of monosodium glutamate in various lipid and lipoprotein fractions in serum of adult male rat. *J. Lif Sci.* **3,** 17-21.

Song Z., Yuan L., Jiao H. and Lin H. (2011). Effect of corticosterone on hypothalamic corticotropin-releasing hormone expression in broiler chicks (*Gallus gallus domesticus*) fed a high energy diet. *Asian-Australasian J. Anim. Sci.* **24(12),** 1736-1743.

Tapiero H., Mathe G., Couvreur P. and Tew K.D. (2002). Free amino acids in human health and pathologies -II. Glutamine and glutamate. *Biomed. Pharmacol.* **56,** 446-457.

Tazawa H., Andrewartha S.J. and Burggren W.W. (2011). Development of hematological respiratory variables in late chicken embryos: The relative importance of incubation time and embryo mass. *Comp. Biochem. Physiol.* **159,** 225-233.

Thaxton J.P. and Puvadolpirod S. (2000). Model of physiological stress in chickens: Quantitative evaluation. *Poult. Sci.* **79,** 391-395.

Tietz N.W. (1995). Clinical Guide to Laboratory Tests. WB Saunders Company, Philadelphia, Pennsylvania, USA.

Veronika H. and Daniela O. (2013). Monosodium glutamate toxic effects and their Implications for human intake. *A Rev. J. Med. Sci. Res.* **60,** 8765-12.

Yamazaki R.K., Brito G.A. and Coelho I. (2011). Low fish oil intake improves insulin sensitivity, lipid profile and muscle metabolism on insulin resistant MSG obese rats. *Lipids Health. Dis.* **10,** 66-72.

Yen P.M. (2001). Physiological and molecular basis of TH action. *Physiol. Rev.* **81,** 1097-1142.

Yuan L., Lin H., Jiang K.J., Jiao H.C. and Song Z.G. (2008). Corticosterone administration and high-energy feed results in enhanced fat accumulation and insulin resistance in broiler chickens. *British Poult. Sci.* **49,** 487-495.

Yoshida Y., Kawabata F., Nishimura S. and Tabata S. (2021). The umami receptor T1R1-T1R3 heterodimer is rarely formed in chickens. *Sci. Rep.* **11(1),** 12318-12326.

Zemel M.B. (2004). Role of calcium and dairy products in energy partitioning and weight management. *Am. J. Clin. Nutr.* **79(5),** 907-912.

Zhelyazkov G. (2018). Effect of monosodium glutamate dietary supplementation on some productive traits of common carp (*Cyprinus carpio*) cultivated in net cages. *Agric. Sci. Technol.* **10(3),** 204-207.