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Effect dietary glutamine and vitamin E supplementation on performance, some blood antioxidant indices in broiler chickens under continuous heat stress temperature

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ABSTRACT: Effects of different levels of 0.0 glutamine (ZG), 0.25 (LG), 0.50 (MG) and 1 percent (HG) along with 100 mg/kg VE were investigated on performance and blood antioxidant status of broiler chickens under heat stress condition. There was significant differences were observed between the treatments for FI during week 4 the experimental period. No significant difference were observed between the treatments for FI during the whole the experimental periods. There was significant differences were observed between the treatments for BWG during the week 1 and 3 the experimental period, wasn't significant differences between the treatments for BWG during the whole the experimental period. Meanwhile during the whole period FI of VE birds were lower than that of ZG, LG and HG ones. For whole the experimental period, BWG of MG and VE fed birds was higher than that of ZG and LG fed birds. There was significant difference between the treatments for FCR during the week 3 experimental period; there wasn't significant difference between the treatments for FCR during the whole experimental period. Meanwhile during the whole period FCR of VE and MG birds were lower than that of ZG, LG and HG ones. There wasn't significant differences were observed between the treatments for TAC, MDA, SOD and GPx between the treatments for during experiment period. It was concluded that although dietary Glutamine and VE consumption can improve the performance of broiler chickens during the weekly period under heat stress.

Keywords: antioxidant status, broiler chickens, heat stress, performance, Glutamine

INTRODUCTION

Chronic heat stress (HS) is a great concern in all types of poultry production. Feed consumption, growth rate, mortality, and other important traits are adversely affected by severe HS. Reduction of feed intake in order to reduce metabolic heat production (Teeter ., 1985) and lower growth rate as well as a reduction in feed efficiency (Geraert ., 1996). Have been reported in poultry under heat stress condition. Heat stress stimulates the release of corticosterone and catecholamine's and initiates lipid peroxidation in cell membranes (Freeman and Crapo, 1982), including membranes of T and B lymphocytes. Animals stressed under improper environmental conditions or subjected to an artificial stress via ACTH and epinephrine injections are found to have reduced α-tocopherol, retinol, and ascorbic acid concentrations in plasma and blood cells (McDowell, 1989), whereas lipid peroxidation levels were found to be high in plasma and tissues due to increased production of free radicals (Naziro glu ., 2000) When the animal was surfing from heat stress , the secretion of catecholamine (including epinephrine and norepinephrine)by chromaffin tissue of adrenal gland would be increased (edens and sigel.,1975). As the chromaffin part was innervated by the sympathetic nervous system, so the secretion of catecholamine in response to stress was immediate (Hillman ,

1987). Haggendal (1987) reported that experimental stress (restraint stress) could result in the reaction of sympathetic nervous system and adrenal system, and resulted in the release of catecholamine which could increase free radicals and induce injury of heat cell. Singal (1982) reported that catecholamine -induced change involve free radicals, by promoting lipid peroxidation which increase membrane permeability (Duthie rt al. 1989). Free radicals are chemicals that have one or more unpaired electrons in their outer orbit. The free radical is produced in normal metabolism processes of the body (chance .1979). In normal conditions, the generated free radicals are extinguished by the antioxidant protective system which includes protective compounds of vitamin E, B-Carotene, ascorbic acid, glutathione and uric acid and the protective enzymatic systems such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase recycling enzymes (glutathione peroxidase and GSH reductase) (duthie .,1989). if the amount of free radicals exceeds reduction capacity of the antioxidant protective system, the free radicals can attack the polyunsaturated fatty acids (PUFA) of membrane and results in lipid peroxidation (duthie .,1989). Lipid peroxidation has numerous detrimental effects on cell function, such as the cross linking of proteins resulting from the proxidantion which can inactivate membrane bound enzymes, and the physical changes within the membrane can alter tertiary. Heat loss in broilers is limited by feathering and the absence of sweat glands. When the temperature and relative humidity exceed the comfort level of a bird, it loses the ability to efficiently dissipate heat. This leads to physiological changes including a reduction in feed intake in order to reduce metabolic heat production (Teeter ., 1985) and lower growth rate as well as a reduction in feed efficiency (Geraert., 1996). in recent year a number of studies have demonstrate that elevated environmental temperatures influence the amino acid needs of broilers, either as a function of reduced amino acid digestibility (Zuprizal ,1993;hai ., 2000)or as a result of decreased feed intake (Deaton at al., 1978; Howlider and rose, 1987). Conversely, other researchers have reported that heat-stressed birds respond positively to increased amino acid consumption (Fuller and Mora, 1973; Dale and Fuller 1980). The Maryland broiler amino acid requirements also indicate that broiler raised at 27 °C and 32°C need higher levels of amino acids than birds raised at 21°C (Thomas at al., 1992). These authors along with many others have experimented with various temperature levels above what is considered thermo neutral (TN; approximately 23.9°C). and their reports have shown consistent effects of these temperatures on bird performance. Broilers exposed to an environmental temperature of 32°C showed a 14% decrease in feed intake by 4 week of age and a 24% reduction by 6 week of age (Geraert ., 1996). High ambient temperature reduces feed intake, live weight gain, feed efficiency (Donkoh, 1989), and thus negatively influences the performance of broilers. Hurwitz. (1980) suggested that decrease in growth rate was due partly to the decrease in feed intake. When the production of free radicals increased, lipid peroxidation in cells occurred and the content of lipid peroxide (LPO) rose (Duthie rt al.1989). The LPO concentrations in plasma and liver were increased in the birds exposed to high temperature for 24 h, which indicated the acute heat stress induced peroxidation of the tissues. The increase in LPO concentration might be caused by two possible reasons. One might be an increase in production of free radicals that resulted in the elevation of LPO. Another reason might be a decrease of activation of protective enzymes. The protective enzymatic systems, which comprised superoxide dismutase (SOD), and GSH recycling enzymes, could reduce the amount of free radicals and repair the free radical damage of tissue. Among these enzymes, SOD is most important and it catalyzes the dismutation of O₂ to H₂O₂. So the changes in the enzyme activity under heat stress might have an important effect on the free radicals damage. In recent years, a number of studies have demonstrated that elevated environmental temperatures influence the amino acid need broilers, either as a function of reduced amino acid digestibility (Wallis and Balnave, 1984). L-glutamine is the most prevalent amino acid in the bloodstream, accounting for 30 to 35 percent of the plasma amino acid nitrogen and the free amino acid pool in the body (Souba, 1993). Because glutamine contains two ammonia groups, one from its precursor, glutamate, and the other from free ammonia in the bloodstream, one of glutamine's roles is to act as a "nitrogen shuttle," which helps protect the body from high levels of ammonia in the blood. Glutamine also plays a role in eliminating free radicals because it acts as a precursor for the antioxidant glutathione synthesis (Wu, 1998). The role of glutamine in glutathione synthesis suggests that the availability of this nutrient may have a profound effect on the regulation of the cellular redox status. Glutamine supplementations in the diets of poultry and swine have been reported to beneficial in several aspects (Yi., 2001; Kitt ., 2002). Exogenous glutamine had the effect of antioxidant protection for rats with the implanted tumor (Kaufmann., 2008) and mice with dystrophic muscles (Mok., 2008). As a precursor of Glutathione (GSH), Glutamine also showed its anti-inflammatory and anticancer effects by up-regulating the gut GSH metabolism in the post-sepsis or murine models of asthma (Kaufmann., 2008; Singleton., 2008).

MATERIALS AND METHODS

A total of two hundred and fifty one-day-old broiler chicks (Ross 308) were obtained from a commercial hatchery. Chickens were weighed and allotted into 5 groups randomly. Each treatment comprised 5 replicate pens with 10 birds in each. All chickens had ad lib access to water and feed and the diets were available as mash from. Diets were based on corn-wheat- soybean meal and formulated to meet the chicken's recommended levels of NRC requirements (NRC, 1984). Diets had similar nutritive value (Table 1). The chickens were fed the same starter (from day 1 to day 21of age) and grower (from day 22 to day 42 of age) diets throughout the whole experiment but received different levels of 0.0, 0.25, 0.5, and 1 percent glutamine and 100 mg/kg vitamin E (as alpha tocopherol acetate). Chicks were raised at 32±1 ° C for inducing heat stress from day one to the end of the experimental period (day 42 of age). Birds were exposed to 23h and 1h darkness during the experiment. Body weight gains, feed intake and feed conversion ratio were determined for the weekly and whole the experimental periods. At the end of the experiment (week 6), five birds per treatment were randomly selected and slaughtered. At slaughter, two series of blood samples were collected in anticoagulant tubes (EDTA). The blood samples immediately transferred to laboratory, then one series of blood samples was centrifuged at 5000 rpm for 5 min and their plasma separated and stored at -20 ° C along with other series of blood samples for the later analyses Plasma TAC was determined using Randox total antioxidant status test kit (Randox Laboratories Ltd, UK), blood SOD activity by Ransod spectrophotometric kit (Ransod, Randox Laboratories Ltd. UK), blood GPX activity by Ransel spectrophotometric kit (Ransel, Randox Laboratories Ltd. UK) and plasma MDA concentration by MDA reaction with thiobarbituric acid followed by extraction with butanol (Kolahi ., 2011). The data were analyzed based on a completely randomized design using the GLM procedure of SAS (SAS Institute, 2002). Duncan's multiple range test was used to separate the means when treatment means were significant (P≤ 0.05). The experimental protocols were reviewed and approved by the Animal Care Committee of the Urmia University.

Table 1. Ingredient composition and chemical analysis of the experimental diets

	Starter diet (0-21 days)			Grower d	Grower diet (22-42 days)			
	ZG	LG	MG	HG	ZG	LG	MG	HG
Ingredients								
Corn	31.08	31.08	31.08	31.08	31.98	31.98	31.98	31.98
Wheat	20.00	20.00	20.00	20.00	25.00	25.00	25.00	25.00
Soybean Oil	3.50	3.50	3.50	3.50	3.95	3.95	3.95	3.95
Soybean meal (44 %)	39.68	39.68	39.68	39.68	33.92	33.92	33.92	33.92
protein)								
Dicalcium phosphate	2.10	2.10	2.10	2.10	2.15	2.15	2.15	2.15
Calcium carbonate	1.10	1.10	1.10	1.10	0.86	0.86	0.86	0.86
DL-methionine	0.38	0.38	0.38	0.38	0.08	0.08	0.08	0.08
L-Lysine	0.29	0.29	0.29	0.29	0.22	0.22	0.22	0.22
Vitamin and mineral premix1	0.50	0.50	0.50	0.50	0.50	0.50	0.50	0.50
Salt .	0.37	0.37	0.37	0.37	0.34	0.34	0.34	0.34
Sand	1.00	0.75	0.50	0.00	1.00	0.75	0.50	0.00
L-glutamine	0.00	0.25	0.50	1.00	0.00	0.25	0.50	1.00
Total	100	100	100	100	100	100	100	100
Chemical Analysis								
Metabolizable energy (MJ/kg) (kcal/g)	2.85	2.85	2.85	2.85	2.95	2.95	2.95	2.95
CP (%)	22.01	22.01	22.01	22.01	20.00	20.00	20.00	20.00
Calcium (%)	1.00	1.00	1.00	1.00	0.90	0.90	0.90	0.90
Available Phosphorus (%)	0.45	0.45	0.45	0.45	0.45	0.45	0.45	0.45
Sodium (%)	0.16	0.16	0.16	0.16	0.15	0.15	0.15	0.15
Arginine (%)	1.54	1.54	1.54	1.54	1.38	1.38	1.38	1.38
Methionine+ Cystine (%)	1.07	1.07	1.07	1.07	0.73	0.73	0.73	0.73
Tryptophan (%)	2.13	2.13	2.13	2.13	1.94	1.94	1.94	1.94
Glycine (%)	0.97	0.97	0.97	0.97	0.89	0.89	0.89	0.89
Serine (%)	1.14	1.14	1.14	1.14	1.04	1.04	1.04	1.04
Glycine+ serine (%)	2.11	2.11	2.11	2.11	1.93	1.93	1.93	1.93
Histidine (%)	0.60	0.60	0.60	0.60	0.55	0.55	0.55	0.55
Isoleucine (%)	0.97	0.97	0.97	0.97	0.88	0.88	0.88	0.88
Leucine (%)	1.81	1.81	1.81	1.81	1.66	1.66	1.66	1.66
Lysine (%)	1.43	1.43	1.43	1.43	1.24	1.24	1.24	1.24
Methionine (%)	0.70	0.70	0.70	0.70	0.38	0.38	0.38	0.38
Cystine (%)	0.37	0.37	0.37	0.37	0.35	0.35	0.35	0.35
Phenylalanine (%)	2.13	2.13	2.13	2.13	1.04	1.04	1.04	1.04
Tyrosine (%)	0.98	0.98	0.98	0.98	0.89	0.89	0.89	0.89
Phenylalanine + Tyrosine (%)	2.13	2.13	2.13	2.13	1.94	1.94	1.94	1.94
Threonine (%)	0.85	0.85	0.85	0.85	0.77	0.77	0.77	0.77
Valine (%)	1.08	1.08	1.08	1.08	0.98	0.98	0.98	0.98

Supplied per kilogram of diet: retinol, 9000 IU; cholecaciferol, 2000 IU; tochopherol, 18 IU; cyanocobalamin, 0 .15 mg; riboflavin, 6.6 mg; pantothenate, 10 mg; niacin, 30 mg; choline, 500 mg; biotin, 0.1 mg; thiamine, 1.8 mg; piridoxin, 3 mg; fulic acid, 1 mg; menadione, 2 mg; ethoxyquin, 100 mg; zinc, 50 mg; manganese, 100 mg; copper, 10 mg; iron, 50 mg; lodine, 1 mg; selenium, 0.2 mg. 2mg: Antioxidant (etuxi cuin). 100mg

RESULTS AND DISCUSSION

Dietary supplementation effect of glutamine on total and weekly BWG, FI and FCR is shown in Table 2. Significant differences FI were observed between the treatments for week 4 (P<0.05). But no significant differences were observed between the treatments for total FI (P>0.05). On week 4, VE birds had significantly lower FI as compared to other treatments (P<0.05). There was significant differences between the treatments for BWG on weeks 1 and 3 of age. On week 1 of age, VE fed birds had higher BWG than glutamine fed birds (P<0.05). On week 3 of age, only VE birds had greater BWG than other treatments (P<0.05). No significant differences were observed between the treatments during the other weeks or whole the experimental period (P>0.05). FCR only affected by dietary VE on week 3. During this week, VE fed birds showed lower FCR as compared to other treatments (P<0.05).

Table 2. Average feed intake (FI, g d –1), Body weight gain (BWG, g d –1) and Feed conversion ratio (FCR) of broiler chickens 0.0 (ZG), 0.25 (LG), 0.5 (MG), 1%(HG) and 100 IU/kg alpha tochopherol (VE) feed broiler chickens at day 42 of age under continues heat stress condition

F I (g)							
TRET	W1	W2	W3	W4	W5	W6	TOTAL
ZG	112.70	357.20	596.18	841.28 a	852.46	1185.71	3945.5
LG	104.90	341.10	592.91	841.75 a	843.50	1232.79	3957.0
MG	106.87	354.63	611.48	832.66 a	895.91	1304.02	4105.8
HG	104.30	350.25	598.12	840.18 a	901.03	1263.54	4061.7
VE	109.62	339.4	607.97	749.29 b	855.17	1214.6	3876.1
p-value	0.13	0.66	0.66	0.009	0.37	0.41	0.24
±SEM	1.24	4.47	4.50	10.48	11.31	20.62	32.79
BWG(g)							
ZG	102.00 ab	214.40	364.10 b	385.73	459.52	441.22	1975.25
LG	96.20 b	201.80	363.40 b	386.31	460.78	492.55	2001.04
MG	96.62 b	220.50	379.10 b	384.61	469.00	613.15	2179.41
HG	97.00 b	210.88	358.13 b	400.83	425.53	527.78	2053.05
VE	104.55 a	223.26	411 ^a	354	223.26	475.03	2012.37
P-value	0.03	0.38	0.004	0.45	0.85	0.22	0.09
±SEM	1.09	3.69	5.28	7.44	12.15	24.98	24.62
FCR							
ZG	1.10	1.67	1.64 ^a	2.18	1.86	2.68	1.97
LG	1.09	1.69	1.64 ^a	2.19	1.88	2.55	1.98
MG	1.06	1.61	1.61 ^a	2.16	1.91	2.17	1.88
HG	1.07	1.66	1.67 ^a	2.10	2.11	2.45	1.97
VE	1.04	1.52	1.47 ^b	2.13	1.95	2.31	1.93
P-value	0.19	0.4	0.01	0.83	0.47	0.31	0.43
±SEM	0.01	0.03	0.02	0.03	0.05	0.08	0.02

a-b Means with no common superscript in each column differ significantly (P≤ 0.05).

At week 6 of age, none of the blood SOD and GPX, TAC and MDA activities were affected by dietary treatments (P>0.05) (table 3). Meanwhile there wasn't significant difference between the treatments for blood TAC content at week 6 of age. VE fed birds showed lower MDA and increased GPX, SOD and TAC as compared to other treatments (P >0.05).

Table 3. Blood activity of superoxide dismutase (SOD) and glutathione peroxidase (GPX) enzymes, total antioxidant capacity (TAC) and Malondialdehyde (MDA) of 0.0 (ZG), 0.25 (LG), 0.5 (MG), 1%(HG) and 100 IU/kg alpha tochopherol (VE) feed broiler chickens at day 42 of age under continues heat stress condition

Treatment	GPX(u/g Hb)	SOD(u/gHb)	TAC(mmol/L)	MDA(nm/mL)
ZG	42.98	1471.4	0.67	1.26
LG	41.81	1505.1	0.66	1.1
MG	39.85	1435.5	0.68	1.1
HG	42.55	1585.8	0.55	1.32
VE	49.70	1610.4	0.93	0.94
P-value	0.14	0.75	0.13	0.30
±SEM	1.24	43.66	0.04	0.09

In recent study, dietary supplementation of 0.5 percent Glutamine or 100 mg/kg alpha tocopherol acetate improved the performance of broiler chickens during the weekly period but no effect significant on the total period. Performance mean quantity feed intake than quantity increased body weight or feed conversation ratio. Dietary supplementation Glutamine and Vitamin E improved significant increased performance week three periods. Dietary supplementation Glutamine and Vitamin E no significant performance on total period but Vitamin E cussed increased performance total period. Dietary glutamine and Vitamin E lower significant feed intake in weeks 4 but no effect significant feed intake in total period. Meanwhile dietary supplementations Glutamine and Vitamin E increased significant body weight on weeks 1 and 3 period. Dietary supplementation Vitamin E cussed lower feed intake and high body weight harvest improved significant performance.

The positive effect of vitamin E has been well established in broiler chickens. For example, dietary supplementation of 250 mg/kg or 100 mg/kg of α -tocopheryl (Sahin ., 2002, De Winne 1996, Yahav . 1997, Hosseini-Mansoub ., 2010). Also These results are consistent with those reported by De Colnago . (1984) reported that dietary vitamin E could improve broiler growth performance. Feed conversion of broilers receiving 100 mg/kg of vitamin E

was improved than that of the other diets. Sahin (2001) found that dietary vitamin E inclusions resulted in a greater performance in Japanese quails reared under heat stress (34°C). The low FCR in vitamin E supplemented group is also in agreement with the earlier reports of Villar . (2002) who reported that feed efficiency increased statistically with vitamin E supplementation. Diets rich in vitamin E have been shown to reduce the catabolic response induced by immune stimulation and may be effective in promoting growth (Rymer and Givens 2005).

In addition, The positive effect of glutamine has been well established in broiler chickens. For example, dietary supplementation Consumption 5 grams of glutamine individually or combined with 100 mg of gamma-aminobutyric acid improves weight gain of 21 to 42-day-old broilers under heat stress (30 to 32 °C) was (Dai., 2011). In another experiment, the consumption of a combination of glutamine and glycine 0.01 and 0.05 g/kg improved weight gain, feed intake and feed conversion ratio in weeks 3, 5, 7, 10 broiler chickens grown Yue-Huang in normal conditions (18 -20 ° C) is (Shu., 2007). Murakami. (2007), Batall and Bartell (2007), Soltan (2009) found 0.5% of glutamine consumption improves the performance of broilers from 1 to 42 days under normal temperature (18-22 ° C) is. Consumption of 1% glutamine and asparagine chick broiler under normal conditions improved weight gain, feed intake and feed conversion were (De-lian ., 2009). Yi (2001) turkey pullets fed diets improved body weight gain in the first weeks of age were reported as 1% glutamine. Yi (2001) reported that the addition of 1 % glutamine in the diet of turkey poults in normal conditions and the conversion efficiency is improved week after hatching. Kitt (2002) reported that the addition of 1% glutamine in the diet improves feed conversion in pigs are weaned. Yi (2001) improved the body weight gain of turkey poults fed diets containing a percentage of glutamine and glutamine in the first weeks of age were reported had no effect on this parameter in starter periods (0-21), grower (22-42) and whole period(0-42). Soltan (2009) stated that the addition of 0.5 and 1% glutamine in the diet of broiler chickens no significant effect on feed intake is 42 days in normal temperature. Ebadiasl (2011) also conducted experiments, the effect of dietary supplementation of 0.5 and 1% glutamine and glutamate levels on performance, improving digestive tract morphology in the jejunum and cecum of broiler chicks under heat stress Prfnjs C. (32 ° C) and found that supplementation of the diet with glutamine and glutamate changes in feed intake of broilers up to 35 days did not cause. The possible beneficial effects on weight gain in recent expriment substance, the immune system power increase heat stress. Research has shown that glutamine supplementation can improve the thermal stress irregularities in the digestive tract (Peter., 1997). The major portion of amino acids provide energy to the cells lining the digestive tract and increases the rate of lymphocyte proliferation (Windmueller., 1980). Although the use of improved weight gain was 0.5 % in our experiment, but no effect of higher levels of glutamine (1%) on weight gain or other performance parameters were determined. Soltan (2009) reported that the addition of higher than 0.5 percent (1%) and glutamine in the diet can be toxic and cause weight loss. While Bartell and Batall (2007) observed that the addition of 1% glutamine causes a significant increase in body weight gain in broilers at normal temperature (18-22 °C), respectively. Because the intestinal villi height can be increased early in the chick's life, then the chick may be able to utilize nutrients more efficiently earlier in life and thus have improved growth performance (Bartell and Batal, 2007). The increase in villi height that was reported by Bartell and Batal, (2007) might indicate that the birds fed diets supplemented with glutamine might have had greater nutrient absorption and utilization because increases in villi height result in more surface area for nutrient utilization. It could also suggest that in fact increased villi height does necessarily lead to increased nutrient utilization and then increased performance. Majorka. (2000), who observed no effect of supplementing 1% Gln on the feed intake, BW gain, or FCR in any of the development phases in broiler chickens, sakamoto (2006) who observed no effect of supplanting 1% Glutamine and 500 mg VE/kg on the feed intake, BW gain and FCR on whole during. Murakami (2007) Expressed that diets with 10 mg of VE/kg of diet, supplemented with Glutamine (for the first 7 d of life) provided better development of the intestinal mucosa in broiler chickens on 41 d of life. This was proven by the behavior of the villi, which can be attributed to the trophic effect of these nutrients. Shorter villi hinder the absorption of nutrients by reducing the area of the intestinal epithelium cells, as a result of the decrease in the osmotic absorption of water.

Vitamin E may have acted in the protection of cells at the membrane level because the action of tocopherylacetate is supplemented by the presence of glutathione in the soluble component of the cell, catabolizing the conversion of organic peroxides and H+ peroxides in alcohols or water and avoiding cell lesion (Ewan, 1993). Leshchinsky and Klasing (2001) showed, small doses of this vitamin E were shown to be sufficient in the protection of the cells. Deficiency or excess of VE decreases the activity of glutathione peroxidase, unbalancing the antioxidant action in the cells and enabling the increase in the formation of free radicals in the cytosol, thus damaging the immunomodulatory system of the birds. Recent experiment showed Consumption VE and glutamine supplementations cussed increased GPX and SOD enzyme in additional supplementations cussed increased capacity anti oxidation, in other hand the supplementations cussed reduces the index MDA is. Although the results anti-oxidation were not significant, but in agreement with our results regarding Puthpongsiriporn . (2001) stated that

the level of plasma MDA continued to rise in hens exposed to heat stress (32 °C) and that laying hens fed rations containing the highest dose of vitamin E (65 IU) had the lowest plasma MDA values. As environmental temperature increased so did the hens' respiration and evaporation as they tried to maintain optimal body temperature, which in turn increased their metabolism and energy consumption (Gomez .,2002). If increased energy needs are not met by feed, mobilization of lipids from stored fat takes place. With regards to lipid mobilization, the malondialdehyde level, an indicator of lipid peroxidation, increases (Whittow ., 1994). Yardibi (2008) present study feed consumption decreased in accordance with the degree of heat stress; thus, the rising energy requirement of the animals could not be supplied by the feed; consequently, MDA levels were high as a result of increased lipid peroxidation due to the mobilization of lipids, and the generation of free radicals increased. Higher levels of free radicals increase the level of MDA. The smaller rise in plasma malondialdehyde levels in the animals that were given a higher dose of vitamin E, as compared to the control groups, suggests that this is due to the fact that vitamin E is an antioxidant agent and reduces lipid peroxidation caused by heat stress (Tengerdy , 1973).

Glutamine supplementation also increased the synthesis of superoxide dismutase and glutathione peroxidases in mice (Kul., 2008). the GPX and SOD is These parameters did not cause effect significant recent experiment. It seems that glutamine metabolism; especially the effects on improving the antioxidant vary in chickens and mice.

Conclusions

In conclusion, the results of this study suggest that 0.5% Glutamine and 100 mg/kg vitamin E of dietary supplementation improved performance by increased ant oxidation enzymes and total ant oxidation during the weekly period. Although tended to improve the FCR and blood MDA content, it could obviate the deleterious effects of intensive heat stress (32 ° C).

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