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Effect of Reduced Dietary Crude Protein Levels on Growth Performance, Plasma Uric Acid and Electrolyte Concentration of Male Broiler Chicks

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ABSTRACT

Changes in dietary electrolyte balance influence the metabolic fate of protein and many amino acids. Furthermore, acid-base condition is achieved in part by the alteration of dietary amino acids pattern and quantity. Therefore, a trial was conducted in a completely randomized design to evaluate performance, carcass characteristics, plasma electrolyte and uric acid concentrations of 19 and 28-day-old male broilers fed three experimental diets in which CP was decreased in a stepwise manner from 21 to 18%. Ileal digestible quantities of all EAA were almost equal in the diets, and the total amount of each EAA was maintained at or above NRC 1994 requirements. Decreasing dietary CP did not affect performance and appetite but increased fat deposition in the whole body and abdominal cavity, significantly. High crude protein fed chickens generally produced breast, thighs and total carcasses that were lower in fat. Reducing dietary CP increased the concentrations of main plasma electrolytes including ionized forms of the electrolytes (Na^+, K^+, Cl^-) but its influence on Ca^{++} and HCO_3^- ions was not significant. On the other hand, plasma uric acid concentration was reduced in parallel with crude protein reduction. Therefore, although reduction of CP to 18% does not impair the performance of broiler chickens, deficiency in uric acid production in low CP diets may lead to blood electrolyte imbalance.

Keywords: Broiler chicken, Crude protein, Dietary electrolyte balance, Uric acid.

INTRODUCTION

It is well-established that a large portion of costs associated with poultry production involves meeting the protein or amino acids requirements of the bird (May *et al*., 1998; Corzo *et al*., 2004). In addition, lowering protein intake results in lower nitrogen excretion (Nahm, 2002; Donsbough *et al*, 2010) and improved ability to manage heat stress (Kidd *et al*., 1996). Attempts to reduce CP content of broiler diets have not been successful in all studies (Edmonds *et al*., 1985) and a failure in performance and feed intake is seen even with providing all requirements for those amino acids considered as essential especially when this reduction is below a minimum level.

Several theories for why performance is negatively affected have been proposed including amino acid imbalance, increase in blood ammonia level and change in the ratio of net energy to metabolizable energy (Aftab *et al*., 2006). Adekunmisi and Robbins (1987) suggested that dietary electrolyte balance should vary with dietary CP level because the growth of chickens fed low-CP diets decreases when dietary electrolyte balance is altered by Na and K additions. Murakami *et al.* (2003) detected the best dietary electrolyte balance for different

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levels of dietary protein. However, the relationship between low-CP amino acids fortified diets and dietary electrolyte balance in chickens is poorly understood.

The objective of this experiment was to investigate the effect of CP level of diets supplemented with crystalline amino acids on performance and important blood electrolytes including Na^+, K^+, Cl^-, Ca^{++} and HCO_3^-. Also the influence of uric acid molecules in surplus blood cations excretion was studied.

MATERIALS AND METHODS

Birds and Housing

Day-old male Ross 308 broiler chicks obtained from a local hatchery were housed in electrically heated battery cages (0.2 m^2 per bird) and had free access to water and a commercial starter diet for 10 days. On day 10, birds (215±15 g) were allotted to one of the 18, 19.5 and 21% dietary protein treatments on the basis of body weight (Tables 1 and 2). Each dietary treatment was applied to 8 replicates of 6 chicks, randomly. The experimental birds were given ad libitum access to water and diet. The ambient temperature was gradually decreased from 34 to 24°C over the period of 1 to 28 days of age. The birds were exposed to a 23L:1D cycle. Experimental diets were fed from 10 to 28 days of age.

Diet Formulation

Corn and soybean meals were sampled before preparing the diet formulation to determine CP as Kjeldahl *nitrogen×6.25*, moisture, and total amino acids content (Degussa AG, Rodenbacher Chaussee 4, Hanau-Wolfgang, Germany), after which the contents of true digestible amino acids were

Table 1. Ingredient composition of experimental diets (%).

	Crude Protein CP (%)		
	21	19.5	18
Ingredients			
Yellow Corn	54.50	58.69	62.67
Soybean meal	36.39	32.4	28.46
Soybean oil	4.98	4.37	3.84
Dicalcium phosphate	1.75	1.78	1.8
Calcium carbonate	1.10	1.11	1.12
Sodium chloride	0.34	0.34	0.35
Potassium sulphate	0.18	0.34	0.49
DL-Methionine	0.21	0.25	0.28
L-Lysine HCl	0.03	0.16	0.28
L-Threonine	0.01	0.06	0.11
L-Arginine	0.00	0.00	0.10
L-Tryptophan	0.00	0.00	0.01
Trace mineral premix[a]	0.25	0.25	0.25
Vitamin premix[b]	0.25	0.25	0.25
Total	100	100	100

[a] Trace mineral premix added the following (mg kg^{-1}) to the diet: Mn ($MnSO_4 \cdot H_2O$): 110.60; Zn ($ZnSO_4 \cdot 7H_2O$): 110.40; Fe ($FeSO_4 \cdot 7H_2O$): 50; Cu ($CuSO_4 \cdot 5H_2O$): 8.30; I ($Ca(IO_3)_2 \cdot H_2O$): 1.08; Se: 0.30; Co: 0.1; Mo: 0.05; a minimum of 6.98 mg of Ca, and a maximum of 8.02 mg of calcium per kg of diet. The carrier was calcium carbonate, and the premix contained less than 0.7% mineral oil.

[b] Vitamin premix added the following to the diet (per kg of diet): Vitamin A (Retinyl acetate): 11.023 IU; Vitamin D (Cholecalciferol): 118 IU ; Vitamin E (DL-α-tocopheryl acetate): 23.54 IU; Menadione (Menadione dimethylpyrimidinol): 1.47 mg; B12: 0.0151 mg; Riboflavin: 5.895 mg; Niacin: 42.93 mg; D-pantothenic acid: 12.11 mg; Choline (Choline chloride): 477.7 mg; Folic acid: 1.15 mg; Pyridoxine (Pyridoxine hydrochloride): 4.17 mg; Thiamin (Thiamin mononitrate): 1.23 mg, D-biotin: 0.075 mg.

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Table 2. Nutritional composition[a] of experimental diets.

CP (%)	21	19.5	18
AME_n (Kcal kg^{-1})	3175	3175	3175
CP (N×6.25) (%)	21.00	19.50	18.00
Ca (%)	0.9	0.9	0.9
Available P (%)	0.45	0.45	0.45
Na (%)	0.16	0.16	0.16
Cl (%)	0.24	0.27	0.29
Na+K-Cl (meq kg^{-1})	250	250	250
Standardized ileal digestible amino acids[b]			
Lys (%)	1.11	1.09	1.09
Met (%)	0.50	0.53	0.55
Met+Cys (%)	0.81	0.81	0.81
Arg (%)	1.28	1.24	1.23
Thr (%)	0.72	0.71	0.71
Leu (%)	1.70	1.61	1.59
Ile (%)	0.82	0.79	0.78
Val (%)	0.82	0.80	0.79
His (%)	0.55	0.53	0.49
Phe (%)	1.01	0.92	0.87
Trp (%)	0.23	0.22	0.21
Tyr (%)	0.98	0.96	0.93

[a] The AME_n, available P, and ileal digestible amino acid values were calculated amounts.
[b] Includes amino acids from intact protein and crystalline sources. Calculated using standardized ileal digestibility coefficients from Lemme *et al*. (2004). Crystalline amino acids were assumed 100% true digestible.

calculated from standardized ileal digestibility coefficients listed by Lemme *et al*. (2004). The amounts of calcium, phosphorus, potassium, sodium, and chloride were analyzed by AOAC (1995) procedures in all feeding ingredients. Each ingredient sample was analyzed in triplicate. The dietary electrolyte balance was set at 250 mEq kg^{-1} in all dietary treatments. All diets were formulated to be isoenergetic (3,175 kcal kg^{-1} of MEn). The concentrations of dietary calcium, available phosphorus, sodium and potassium were maintained equal in all treatments (Table 2). Three levels of CP were used in this study including 21, 19.5, and 18% with almost equal ileal digestible amounts of all EAA. Total EAA concentrations in all treatments were maintained at or above NRC (1994) recommended levels (Table 2). The L-Lys HCl, DL-Met, and L-Thr used in the diets were feed grade, whereas all other crystalline amino acids as well as K_2So_4 were reagent grade (minimum 99.5% purity) and purchased from Degussa Iran AG (Tehran, Iran).

Blood Samples

At the age of 15 and 28 days, blood samples from two birds per replicate were taken from the brachial vein, pooled and placed into evacuated heparinized tubes. Samples were immediately put on ice and processed within 1 hour of collection. Plasma was obtained by centrifuging the blood samples at 5,000×*g* for 10 minutes at 1°C. Plasma samples were frozen at −20°C until analysis. The plasma concentration of Na, K, Ca and HCO_3 were determined using ISE method and Cl plasma level was detected by spectrophotometry. Plasma uric acid was measured by the method of Liddle *et al*. (1959) by enzymatic (uricase) spectrophotometry. Briefly, this enzyme catalyzed the oxidation of uric acid to allantoin with subsequent production of H_2O_2. This method is based on the fact that uric acid has a UV absorbance peak at 293 nm, whereas allantoin does not. The difference in absorbance before and after incubation with uricase is proportional to the uric acid concentration.

Carcass Characteristics and Whole-Body Analyses

At the end of the experimental period (day 28), two birds per replicate (with a BW close to the replicate mean), were selected. After 16 hours of feed withdrawal, birds were killed by cervical dislocation. One of the sampled birds was used to determine carcass characteristics. The data on breast and thigh muscle (without bone and skin), abdominal fat, liver and heart were recorded at this stage. The other birds were stored in an airtight polyethylene bag at −22°C for later determination of whole-body composition. The whole body was thawed overnight at room temperature,

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homogenized for 2.5 minutes, and sampled according to procedures described by Barker and Sell (1994). Whole-body nitrogen content, and fat content were analyzed in triplicate, subsequently (Nutrition and Chemical Laboratory of Tehran University, Karaj, Iran). Whole-body CP was calculated as Kjeldahl *nitrogen*×*6.25*.

Statistical Analysis

Data were analyzed using the general linear model ANOVA (SAS, 2004) in a completely randomized design. Means were compared using Duncan's multiple range test. In all cases, significance was set at $P< 0.05$.

RESULTS

During 10 to 19 and 19 to 28 days of age, performance was not influenced by dietary treatments (Table 3). Table 4 summarizes the effect of the dietary treatments on relative weights of breast, thigh, and some visceral organs. No significant effect of dietary treatments was observed on relative weights of breast, thigh, liver and heart. Abdominal fat and whole-body fat were significantly affected by dietary CP ($P\leq 0.05$). Chickens fed high-CP diets generally produced breast, thighs and total carcasses that were lower in fat. In fact, the difference between the birds fed 18-CP and 21-CP diets was significant for the abdominal fat, thighs and whole-body fat. Fat content of breast muscle was significantly lower for birds fed 18-CP diet than those fed two other diets (Table 5).

Table 6 shows that the plasma uric acid and electrolyte levels were affected by the reduction of dietary CP. Reduction of CP in both periods of time led to increases in some electrolyte levels including Na^+, Cl^- and K^+ in plasma. The effect of dietary CP level on plasma Ca^{++} and HCO_3^- was not significant. In addition, plasma uric acid was affected by dietary CP level. The greatest value of

Table 3. Effects of Dietary CP level on performance during 10 to 19 and 10 to 28 days of age [a].

CP (%)	Body weight gain between 10-19 d	10 to 19 d Feed intake (g)	10 to 19 d FCR [b] (g g^{-1})	Body weight gain between 19-28 d (g)	10 to 28 d Feed intake (g)	10 to 28 d FCR (g g^{-1})
21	452	585	1.29	1037	1599	1.55
19.5	439	581	1.32	1053	1615	1.53
18	452	604	1.33	1038	1628	1.56
SEM	12	16	0.02	16	26	0.01

[a-b] Values within columns without a common letter differ significantly ($P\leq 0.05$).
[a] Results are means of 8 replicates (6 chicks per replicate) per treatment.
[b] Feed Conversion (Total feed consumed/Weight gain of birds).

Table 4. Effects of Dietary CP level on the percentage weight of heart, liver, thigh, breast and abdominal fat (28 days of age) [a].

CP (%)	Heart	Liver	Thigh	Breast	Abdominal Fat
21	0.55	2.1	19.71	22.21	1.81[b]
19.5	0.56	2.1	19.36	21.05	1.86[ab]
18	0.54	2.1	19.05	20.71	1.93[a]
SEM	0.01	0.06	0.41	0.51	0.03

[a-b] Values within columns without a common letter differ significantly ($P\leq 0.05$).
[a] Results are means of 8 replicates (one chick were slaughtered per replicate) per treatment.

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Table 5. Effects of Dietary CP level on breast and thigh nutrient composition (28 days of age) [a].

CP (%)	Breast		Thigh		Whole body	
	Fat (%)	Protein (%)	Fat (%)	Protein (%)	Fat (%)	Protein (%)
21	1.28[b]	23.80	5.98[b]	20.33	9.18[b]	16.21
19.5	1.46[b]	23.62	6.37[ab]	19.66	9.44[ab]	16.33
18	1.99[a]	23.58	7.21[a]	19.91	9.84[a]	15.64
SEM	0.17	0.17	0.35	0.28	0.17	0.49

[a-b] Values within columns without a common letter differ significantly ($P \leq 0.05$).
[a] Results are means of 8 replicates (one chick were slaughtered per replicate) per treatment.

Table 6. Effects of Dietary CP level on plasma uric acid and electrolyte ions including Na^+, K^+, Ca^{++}, Cl^- and HCO_3^- concentrations (mEq 1000 ml^{-1}) [a].

CP (%)	Na	K	Ca	Cl	HCO_3 (mmol L^{-1})	Uric acid (mg L^{-1})
19 days of age						
21	132[b]	7.14[b]	3.01	100[b]	13.4	110.9[a]
19.5	136[a]	7.94[a]	3.01	106[a]	13.6	110.5[a]
18	136[a]	7.95[a]	3.02	104[a]	13.3	100.8[b]
SEM	0.72	0.15	0.01	0.67	0.21	0.54
28 days of age						
1: 21	137[b]	6.2	2.99	101[b]	14	109.5[a]
2: 19.5	139[a]	5.9	2.98	102[ab]	13.4	111.2[a]
3: 18	139[a]	6.2	2.01	105[a]	13.8	102.1[b]
SEM	0.38	0.1	0.02	0.73	0.17	0.70

[a-b] Values within columns without a common letter differ significantly ($P \leq 0.05$).
[a] Results are means of 8 replicates per treatment.

plasma uric acid was obtained from the highest CP diet.

DISCUSSION

Many studies have tried to determine the minimum level of dietary CP below which even with sufficient amino acid levels, performance will be negatively affected. According to experimental conditions and treatments, the minimum level of CP could differ significantly. Various reports on the lowest level to which CP can be reduced with crystalline amino acids supplementation in broiler diets without reducing bird performance have been presented. These include 22.7% (Waldroup *et al*., 2005), 17.6% (Corzo *et al*., 2005), 18.2% (Dari *et al*., 2005), 19% (Namroud *et al*., 2008) and 16% (Aletor *et al*., 2000). Our results on fat deposition in different parts of carcass are in complete agreement with other researches (Yamazaki *et al*., 1998; 2006; Moran and Stilborn 1996). One of the mechanisms involved in decreasing carcass fatness by feeding greater protein level diets is the associated increased heat increment involved in deamination and transamination of surplus amino acids to other metabolites and finally uric acid. Rosebrough *et al*. (2002) showed that increase of CP can dramatically decrease in vitro lipogenesis. They suggested that a combination of mRNA stability and posttranscriptional events interact to regulate lipogenesis in the chicken.

Adekunmisi and Robbins (1987) concluded that electrolyte balance in the diet will be different for situations involving low CP and high levels of synthetic amino acids compared with formations with high CP diets. They observed that growth of chicks fed low CP (14.3%) content was depressed when electrolyte balance was changed by the addition of sodium and potassium.

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However adding these electrolytes to diets containing 28.6% CP improved growth rate. Summers (1996) also achieved similar results. The metabolism of majority of amino acids such a glutamic acid, lysine, arginine, serine, glycine and the branched chain amino acids appears to be influenced by acid-base balance (Patience, 1990). Namroud *et al*. (2008) suggested a minimum 19% dietary CP at 280 mEq kg^{-1} electrolyte balance (Na^+, K^+, Cl^-) of diet. The basic (cationic) amino acids (lysine, arginine and histidine) yield neutral end-products plus a proton; sulfur (methionine and cysteine) amino acids are also acidogenic because they generate sulfuric acid when oxidized. The dicarboxyllic (anionic) amino acids (aspartate and glutamate, but not asparagine and glutamine) consume acid when oxidized and thus reduce the acid load of the diet. Oxidation of protein is generally considered a net contributor of acid, although this depends on its amino acid profile. As described in detail in a review (Walser, 1986), the oxidation of neutral amino acids has no effect on acid-base status, whereas oxidation of dicarboxyllic amino acids cause metabolic alkalosis. Oxidation of dibasic amino acids results in metabolic acidosis. If the amino acid is phosphorylated, as in the case of phosphoserine, oxidation will result in a metabolic acidosis (Brosnan and Brosnan, 1982; Halperin *et al*., 1986). This will be important when in commercial low CP diets the major part of supplemented amino acids including methionine, lysine and arginine is acidogenic. By reducing each percent of CP, the portion of anionic amino acids in diet declines. Berres *et al*. (2010) suggested that adding glutamic acid to low CP amino acids-fortified diet could improve performance in low CP diets at 280 mEq kg^{-1} dietary electrolyte balance which may be due to its anionic effect. Based upon previous studies, in the present experiment dietary electrolyte balance was reduced to 250 mEq kg^{-1} in parallel with CP reduction. The speculative achievement of this alteration was the capability of CP reduction to 18% without any negative influence on performance. This finding was not in agreement with Adekunmisi and Robbins (1987). Nevertheless, the relationship between low-CP amino acid-supplemented diets and dietary electrolyte balance in broiler chickens is poorly understood (Martinez-Amezcua *et al*., 1998).

Given that the bird is in nitrogen balance and without acid-base disturbance, around 75-80% of total nitrogen excretes in uric acid and 15% is NH_4^+. Significant alterations result if the bird is fed on a low CP diet or if the acid-base load is varied. When the diet is low in protein, a meaningful shift was observed from uric acid to NH_4^+ (Tasaki and Okumura, 1964; McNabb and McNabb, 1975; Namroud *et al*., 2009). This shift is related to the acidosis induced by low protein intake (Hannaford *et al*., 1982). Okumura and Tasaki (1968) made a similar observation after the incorporation of HCl into the diet. These authors reported that uric acid fraction was proportionally reduced when urine passed from alkaline to acidic pH. Namroud *et al.* (2008, 2009) reported comparable results and concluded that low-CP essential amino acids fortified diet induces blood and excretory uric acid levels reduction but elevation in ammonia in blood and excreta. In the present study, similar results were obtained. An interesting fact regarding uric acid excretion is that important quantities of positive ions are physically trapped undissolved in these concrements (McNabb *et al*., 1973). In other words, trapped ions are adsorbed or elecrostatically attracted by uric acid molecules (McNabb, 1974). The results of McNabb *et al*. (1973) indicate that a high fraction of Na^+, K^+, Ca^{++} and Mg^{++} is excreted by trapping in uric acid particularly when nitrogen intake (and uric acid output) is high. Practically no Cl^- was bound to urinary uric acid. Two main factors affect trapping of cations in precipitates of uric acid and urates; first the total amount of precipitated uric acid and second the pH. McNabb and McNabb (1980) observed that cations were not bound to uric acid below about pH 6.2. The present results indicate

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that decreasing dietary CP may lead to elevated cations of blood particularly Na^+ and K^+ on the basis of the above mechanism, since in our experiment the plasma uric acid level was significantly reduced in the low protein diet. The change in blood ions may act as an acute stressor which can negatively affect performance because as previously suggested by Austic and Calvert (1981) and Adekunmisi and Robbins (1987), higher levels of cations simulate uric acid excretion. Therefore electrolyte imbalance can be expected to be more detrimental when nitrogen intake is low.

CONCLUSIONS

We conclude that dietary electrolyte balance plays a key role in homeostasis and maintaining performance in low CP amino acids fortified diets. By reducing CP and supplying entirely all essential amino acid requirements, the amount of uric acid production decreases to its minimum level and surplus positive electrolyte ions lose one of the most important ways of their excretion namely physical trapping by uric acid molecule. Therefore, it seems that the optimum dietary electrolyte balance varies with dietary CP level. In conclusion, in order to maintain performance and blood electrolytes homeostasis in broiler chicks fed low CP amino acids-supplemented diets, electrolyte balance should be decreased in parallel with decline in nitrogen intake.

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تاثیر استفاده از جیره‌های حاوی سطوح پایین پروتئین و دارای مکمل اسیدهای آمینه بر عملکرد رشد، غلظت اسید اوریک و الکترولیت‌ها در پلاسمای جوجه خروس‌های گوشتی

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چکیده

تغییر تعادل الکترولیتی جیره، متابولیسم پروتئین و بسیاری از اسیدهای آمینه را تحت تأثیر قرار می‌دهد. همچنین مقدار و وضعیت اسیدهای آمینه بر وضعیت اسید و باز بدن تاثیرگذار است. بنابراین آزمایشی در قالب طرح کاملا تصادفی طراحی شد و در آن اثر کاهش سطح پروتئین خام از ۲۱٪ به ۱۸٪ بر عملکرد، خصوصیات لاشه و غلظت اسید اوریک و الکترولیت‌ها در پلاسما در جوجه‌های گوشتی در سن ۱۰ تا ۲۸ روزگی مورد بررسی قرار گرفت. کاهش سطح پروتئین خام جیره تا سطح ۱۸ درصد با توجه به تأمین کامل نیاز آمینواسیدهای ضروری تأثیری بر عملکرد پرنده نداشت اما میزان چربی لاشه و چربی محوطه شکمی به شکل معنی‌داری ($p<0.05$) با کاهش سطح پروتئین جیره افزایش یافت. کاهش سطح پروتئین جیره با توجه به تأمین نیاز آمینواسیدهای ضروری، غلظت الکترولیت‌های موجود در پلاسما را به شکل معنی داری ($p<0.05$)افزایش داد. از طرفی با کاهش سطح پروتئین خام، غلظت اسید اوریک در پلاسما کاهش یافت. بنابراین هرچند کاهش پروتئین خام جیره تا سطح ۱۸٪ تاثیری بر عملکرد طیور نداشت، کاهش تولید اسید اوریک در سطوح پایین پروتئین می‌تواند منجر به برهم خوردن تعادل الکترولیتی گردد.

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