

## **Effect of Dietary Cholecalciferol , 1,25-Dihydroxy-Cholecalciferol and Ascorbic Acid on Growth Performance and Bone Mineralization in Turkey Poults**

**I. H. Hermes<sup>(1)</sup>, P. E. Waibel<sup>(2)</sup>, F. A. Atia<sup>(3)</sup> and H. M. Sabri<sup>(3)</sup>**

*<sup>(1)</sup>Department of Animal Production and Breeding,*

*King Saud University Al-Qassim Branch, Bureidah, Saudi Arabia;*

*<sup>(2)</sup>Department of Animal Science, University of Minnesota, St. Paul, MN, 55108, USA; and*

*<sup>(3)</sup>Department of Animal Production, University of Suez Canal, 41522 Ismailia, Egypt*

(Received 1/1/1420; accepted for publication 12/8/1420)

**Abstract.** An experiment was conducted to determine the influence of cholecalciferol (Vit D<sub>3</sub>), 1,25-dihydroxycholecalciferol (1,25-(OH)<sub>2</sub>D<sub>3</sub>) and ascorbic acid (AA) at the level of 500 mg/kg diet on growth performance and bone development of male large White turkeys during 3 to 21 days of age. Poults were housed in electrically heated batteries and the fluorescent lighting was sleeved to remove ultraviolet radiation. The corn-soybean meal basal diet contained 0.83% Ca and 0.66% available-P. Vit D<sub>3</sub> was supplemented at 300, 600, 900 and 1800 ICU/kg diet, with and without AA. 1,25-(OH)<sub>2</sub>D<sub>3</sub> was supplemented at 5, 10 and 20 ug/kg alone and 10 ug with Vit D<sub>3</sub> (900 ICU/kg) or AA. Near maximum gain occurred with 900 ICU of Vit D<sub>3</sub>/kg with or without AA. Maximum gain and no rickets occurred with all levels of 1,25-(OH)<sub>2</sub>D<sub>3</sub> alone or with Vit D<sub>3</sub>. Bone ash (tibia and toe) and breaking strength were reduced with 900 ICU of Vit D<sub>3</sub>/kg or 5ug 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg. AA did not affect criteria measured. This research indicated that :1) 5 ug 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet met the requirement of the turkey poult for gain and rickets prevention but more was required for maximum bone ash and breaking strength. 2) AA did not influence the requirement of Vit D<sub>3</sub>.

### **Introduction**

The antirachitic Vit D occurs in several forms. Cholecalciferol (Vit D<sub>3</sub>) is the form considered most important as an essential nutrient in animal diets [1, p. 131]. Under exposure to ultraviolet irradiation, 7-dehydrocholesterol is converted to cholecalciferol subcutaneously. Cholecalciferol is supplied to the animal from the dietary sources and absorbed in the intestine at an efficiency of about 60-70% [2].

It is well known from early literatures [3,4] that the requirements of Vit D<sub>3</sub> is largely dependent on the ratio of Ca: P in the ration. The requirement of turkey poults for Vit D<sub>3</sub> was listed by NRC [5, p. 29] as 900 ICU/kg of diet containing 1.2% Ca and 0.6% available P. While the recent edition of NRC [6, p. 36] referring to studies by [7] indicated that

1100 ICU/kg of diet was necessary to maximize both growth and toe ash content of turkey poulters when the diet containing 1.2% Ca and 0.6% available P.

Deluca [8] concluded that 1,25-dihydroxy cholecalciferol [ $1,25(\text{OH})_2 \text{D}_3$ ], a steroid hormone, is a biologically active metabolite of Vit  $\text{D}_3$  and is ten times more effective than Vit  $\text{D}_3$  in preventing and curing rickets. The active metabolite  $1,25(\text{OH})_2 \text{D}_3$  acts directly at the intestinal level to stimulate intestinal Ca and P absorption. Sanders and Edwards [9] reported that the use of  $1,25(\text{OH})_2 \text{D}_3$  as a dietary supplement, to aid in prevention of leg abnormalities and to increase bone ash in turkeys, was promising. In addition, Edwards *et al.* [10] reported that supplementation of 3 ug of  $1,25(\text{OH})_2 \text{D}_3$ /kg of diet could satisfy the broiler's Vit  $\text{D}_3$  requirement as measured by body weight, bone ash, rickets and tibia dyschondroplasia.

Traditionally, AA is not required in poultry diets because avian species are capable of producing AA biosynthetically. AA is required for the hydroxylation of proline to produce collagen bone matrix. The functional form of Vit  $\text{D}_3$  metabolite and AA are produced in turkeys. It has been suggested that AA is important in the conversion of Vit  $\text{D}_3$  to its functional form,  $1,25(\text{OH})_2 \text{D}_3$ , thus enhancing Ca absorption [11, pp. 71-76; 12, pp. 644-653]. This indicates that AA could enhance Ca absorption. On the other hand, [13] reported that in the first 15 days, post-hatch, chicks can not synthesize AA at a high rate to meet the requirement and cope with stress factors and the calcification process. Therefore, young chicks may benefit from the addition of AA to the diet in the sense that it synergistically affects the synthesis of  $1,25(\text{OH})_2 \text{D}_3$ .

The present study was conducted to determine the influence of dietary Vit  $\text{D}_3$ ,  $1,25(\text{OH})_2 \text{D}_3$  and AA on growth performance and bone development of male large White turkeys during 3 to 21 days of age.

### Materials and Methods

The present study was carried out at the Department of Animal Science, College of Agriculture, University of Minnesota, USA (1993/1995) as a channel system program between Egypt and USA. This research was completed as a partial requirement for the Ph.D. degree for Mrs. F.A. Atia.

Four hundred and forty eight 1-day old male large White turkeys (Nichols strain) were obtained from a commercial hatchery. Birds were randomized, wing-banded and caged by group in electrically heated battery brooders with wire mesh floors in the absence of ultraviolet light.

The poulters were fed for 3-days on a commercial typical corn-soybean meal type diet as a pre-experimental period, and were weighed individually at 3 days of age, sorted into similar body weight, then distributed equitably into groups of 8 poulters per pen. There were four replicates (pens) per treatment. The pens in each battery comprised a block and

treatments were randomized within each block. At 3-days of age poults were fed the experimental diets until 21 days of age. Feed and water were supplied *ad – libitum*.

The composition of basal, control and experimental diets are shown in Table (1). Experimental diets were formulated by using Acufeed, a least – cost linear program of diet formulation provided by the Wala Group, St Paul, MN, USA, and NRC [6] was used as nutrient guidelines for turkey poult requirements.

**Table 1. Composition of the basal and control diets and the experimental treatments**

Ingredients	Basal diet g/kg	Control diet g/kg	Treatments	Vitamin D <sub>3</sub>		1,25 (OH) <sub>2</sub> D <sub>3</sub> µg/kg	Ascorbic acid mg/kg
				Units ICU/kg	Weight µg/kg		
Ground yellow corn	433.21	428.20	1	300	7.5	0	0
Soybean meal (47%)	505.40	500.00	2	600	15.0	0	0
Corn oil	20.08	19.98	3	900	22.5	0	0
Fermentation residue product <sup>1)</sup>	2.50	2.50	4	1800	45.0	0	0
DL-methionine	2.26	2.24	5	300	7.5	0	500
Dicalcium phosphate	26.09	25.82	6	600	15.0	0	500
Calcium carbonate	3.56	14.36	7	900	22.5	0	500
Sodium chloride	3.00	3.00	8	1800	45.0	0	500
Mineral premix <sup>2)</sup>	1.25	1.25					
Vitamin premix <sup>3)</sup>	2.65	2.65	9	0	0	5	0
	1000	1000	10	0	0	10	0
Calculated nutrients composition			11	0	0	20	0
ME (MJ/kg)	12.18	12.05					
Crude protein	278.5	275.52	12	900	22.5	10	0
Calcium	8.3	12.3	13	0	0	10	500
Total-P	9.2	9.1					
Non phytate-P	6.2	6.2	14 <sup>4)</sup>	900	22.5	0	0

<sup>1)</sup> Fermaacto<sup>®</sup>, a natural feed supplement, based upon a primary fermentation. Pet-Ag. Inc., Elgin, Ill. 60120.

<sup>2)</sup> Trace mineral mixture MNTM contains: 2% iron, 0.3% copper, 0.6% manganese, 0.6% zinc, 0.12% iodine, 0.02% cobalt and 0.161% selenium.

<sup>3)</sup> Vitamin mix MTS-74 supplies (per kg of mixture): 4,400,000 IU vitamin A acetate, 5,500 IU vitamin E acetate, 1.1g menadion dimethyl pyrimidinol bisulfate, 2.65 g riboflavin, 4.0 g dicalcium pantothenate, 26.5 g niacin, 198.4 g choline chloride, 4 mg vitamin B12, 22 g folic acid, 0.55 g pyridoxine and 22 mg biotin.

<sup>4)</sup> Control diet (treatment 14) contained 12.3 g Ca/kg and all other treatments contained 8.3 g Ca/kg diet.

The poult were observed daily for mortality and indications of leg problems. Individual body weight, leg rickets score and pen feed consumption were recorded at 9, 15 and 21 days of age. At 21 days of age three poult per pen closest to the average weight were chosen for blood and bone samples.

### Leg/rickets score

Each bird was given a score for leg condition. The score were; 0: Standing birds with normal legs; 1: bird may be affected; 2: bird obviously unsteady, sits down,

reluctant to move; 3: bird has rotated tibia or severely bowed legs, but still able to walk some and 4: bird unable to stand [14].

### Breaking bone strength and bone ash

The left tibia was separated and cleaned of all adhering tissues. An Instron (Model 1122) Universal testing machine (Instron Canton, MA) equipped with 500-kg load cell was used to determine the force necessary to break the tibia bone. The force was applied to the center of the tibia. The bone fragments resulting from the breaking strength determination were collected, dried, fat extracted with ether, dried and weighed. The dry fat-free bones were ashed (550 °C/ 24 hours). The ash was expressed as the percentage of the fat-free bone weight. The determination of toe ash was done according to the procedure of [15].

### Determination of Calcium, phosphorus and alkaline phosphatase

Blood serum were analyzed for Ca, P and alkaline phosphatase activity using Sigma procedures No. 587, 360 UV and 104, respectively, Sigma Diagnostics, St. Louis, Mo., USA.

### Experimental design and statistical analysis

A randomized complete block design was used with blocks based on location and the block by treatment interaction representing the error term. The mathematical models used were:

$$Y_{ijk} = \mu + B_i + T_j + BT_{ij} + e_{ijk} \quad (\text{Model 1})$$

where :  $Y_{ijk}$ : observation on the  $k^{\text{th}}$  individual bird in the  $j^{\text{th}}$  treatment in the  $i^{\text{th}}$  block.

$\mu$ : overall mean.

$B_i$ : the fixed effect of the  $i^{\text{th}}$  block.

$T_j$ : the fixed effect of the  $j^{\text{th}}$  treatment.

$BT_{ij}$ : the interaction between the  $i^{\text{th}}$  block and the  $j^{\text{th}}$  treatment.

and  $e_{ijk}$ : the random error associated with the observation  $Y_{ijk}$ .

This model was applied to body weight, weight gain and breaking bone strength where individual observation was measured.

$$Y_{ij} = \mu + B_i + T_j + e_{ij} \quad (\text{Model 2})$$

Where :  $Y_{ij}$ : pen average in the  $j^{\text{th}}$  treatment in the  $i^{\text{th}}$  block.

$\mu$  : overall mean.

$B_i$ : the fixed effect of the  $i^{\text{th}}$  block.

$T_j$ : the fixed effect of the  $j^{\text{th}}$  treatment.

and  $e_{ij}$ : the random error associated with the observation  $Y_{ij}$ .

This model was applied to feed intake, feed efficiency, blood measurements, rickets score, and tibia and toe ash, where one value was calculated for each pen.

Data from experiment were analyzed by the General Linear Models (GLM) procedure of SAS [16]. Significant differences among treatment means were separated by Duncan's new multiple range test [17] with a 5% level of probability.

## Results

### Body weight and gain

The addition of graded levels of Vit D<sub>3</sub> with or without AA (trt 1-8) had no significant effect 6 days after onset of the experiment (Table 2). Small reductions in growth (body weight and gain) were observed due to 10 or 20 µg of 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 10 and 11) during the first experimental period (3-9 days). The latter 20 µg of 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet, (trt 11) caused a significant depression in body weight and gain compared with 600 and 900 ICU Vit D<sub>3</sub>/kg diet alone (trt 2 and 3) or 300, 600, and 900 ICU Vit D<sub>3</sub>/kg diet combined with AA (trt 5-7).

Starting at the second experimental period 9-15 then third 15-21 days of age, feeding low level of Vit D<sub>3</sub> (300 ICU/kg diet, trt 1) caused a significant decrease in body weight and gain (Table 2). Increasing the level of Vit D<sub>3</sub> in the basal diet from 300 to 900 ICU/kg diet (trt 2 and 3) reflected a significant increase in body weight (at 21 days) and gain (15-21 and 3-21 days). The average body weight at 900 ICU D<sub>3</sub>/kg and 8.3 g Ca/kg diet (trt 3) was comparable to the control (900 ICU D<sub>3</sub>/kg and 12.3 g Ca/kg diet, trt 14). When Vit D<sub>3</sub> was fed at a higher level (1800 ICU/kg diet, trt 4), compared to 900 ICU/kg (trt 3), body weight (21 days) and gain (9-15, 15-21 and 3-21 days) were non significantly heavier. The addition of 500-mg AA/kg diet, with the same levels of Vit D<sub>3</sub> (trt 5-8), resulted in no significant effect on body weight or gain.

Supplementation of the diet with graded levels of 1,25-(OH)<sub>2</sub>D<sub>3</sub> (5, 10, and 20 µg/kg diet, trt 9-11) resulted in no significant effect on body weight or gain at all ages. The addition of 20 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet, (trt 11) did not produce any depression on growth at 21 days of age. The combination of 900 ICU D<sub>3</sub> and 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 12) resulted in body weight and gain similar to those fed higher levels of Vit D<sub>3</sub> (900 and 1800 ICU/kg diet, trt 3 and 4) and to those fed different levels of 1,25-(OH)<sub>2</sub>D<sub>3</sub> (trt 9-11). Reduction observed in body weight was due to the addition of 500 mg AA/kg diet with 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub> (trt 13) compared with 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 10) alone.

**Table 2.** Effect of dietary Vit D<sub>3</sub>, 1,25-(OH)<sub>2</sub>D<sub>3</sub> and AA on live body weight and weight gain of male turkey poults from 3 to 21 days of age

Treatments	Body weight, at days (g)				Weight gain, during days (g)			
	3 <sup>1)</sup>	9	15	21	3-9	9-15	15-21	3-21
1	66.9±.9 <sup>a</sup>	131±3 <sup>ab</sup>	215±7 <sup>c</sup>	282±11 <sup>d</sup>	64.4±3.0 <sup>abc</sup>	83±4 <sup>ef</sup>	65±7 <sup>d</sup>	215±11 <sup>d</sup>
2	66.8±.9 <sup>a</sup>	133±3 <sup>ab</sup>	231±6 <sup>abc</sup>	367±12 <sup>bc</sup>	66.3±2.5 <sup>ab</sup>	98±4 <sup>bcde</sup>	136±8 <sup>bc</sup>	300±11 <sup>bc</sup>
3	66.8±.9 <sup>a</sup>	133±4 <sup>ab</sup>	245±7 <sup>a</sup>	411±13 <sup>a</sup>	66.4±3.8 <sup>ab</sup>	112±4 <sup>abc</sup>	167±8 <sup>a</sup>	345±13 <sup>a</sup>
4	66.9±.9 <sup>a</sup>	130±3 <sup>abc</sup>	244±6 <sup>ab</sup>	424±10 <sup>a</sup>	63.4±2.6 <sup>abc</sup>	114±4 <sup>ab</sup>	180±7 <sup>a</sup>	358±9 <sup>a</sup>
5	67.1±.8 <sup>a</sup>	136±3 <sup>ab</sup>	215±5 <sup>c</sup>	294±8 <sup>d</sup>	68.9±2.3 <sup>ab</sup>	78±3 <sup>f</sup>	75±6 <sup>d</sup>	226±8 <sup>d</sup>
6	66.7±.9 <sup>a</sup>	138±3 <sup>a</sup>	234±5 <sup>abc</sup>	351±11 <sup>c</sup>	70.8±2.9 <sup>a</sup>	97±3 <sup>cde</sup>	117±8 <sup>c</sup>	284±11 <sup>c</sup>
7	66.8±.8 <sup>a</sup>	137±3 <sup>a</sup>	248±6 <sup>a</sup>	416±12 <sup>a</sup>	69.9±3.0 <sup>ab</sup>	111±4 <sup>abc</sup>	168±7 <sup>a</sup>	349±12 <sup>a</sup>
8	66.8±.9 <sup>a</sup>	127±4 <sup>abc</sup>	240±6 <sup>ab</sup>	417±10 <sup>a</sup>	60.2±3.4 <sup>abc</sup>	113±3 <sup>a</sup>	177±6 <sup>a</sup>	350±10 <sup>a</sup>
9	66.7±.9 <sup>a</sup>	130±3 <sup>abc</sup>	243±6 <sup>ab</sup>	417±10 <sup>a</sup>	63.2±3.1 <sup>abc</sup>	113±3 <sup>abc</sup>	174±6 <sup>a</sup>	350±10 <sup>a</sup>
10	66.5±.9 <sup>a</sup>	125±3 <sup>bc</sup>	239±5 <sup>ab</sup>	414±10 <sup>a</sup>	58.5±2.5 <sup>bc</sup>	114±3 <sup>ab</sup>	174±5 <sup>a</sup>	347±10 <sup>a</sup>
11	66.8±.9 <sup>a</sup>	121±3 <sup>c</sup>	237±6 <sup>abc</sup>	417±9 <sup>a</sup>	54.4±3.1 <sup>c</sup>	113±3 <sup>abc</sup>	179±4 <sup>a</sup>	350±9 <sup>a</sup>
12	67.2±.9 <sup>a</sup>	128±3 <sup>abc</sup>	244±7 <sup>ab</sup>	419±12 <sup>a</sup>	61.0±2.8 <sup>abc</sup>	116±3 <sup>a</sup>	175±6 <sup>a</sup>	351±11 <sup>a</sup>
13	66.8±.8 <sup>a</sup>	126±3 <sup>abc</sup>	221±6 <sup>bc</sup>	378±10 <sup>abc</sup>	59.4±2.7 <sup>bc</sup>	94±3 <sup>dc</sup>	158±7 <sup>ab</sup>	312±10 <sup>abc</sup>
14 <sup>2)</sup>	66.8±.9 <sup>a</sup>	130±3 <sup>abc</sup>	237±6 <sup>abc</sup>	408±11 <sup>ab</sup>	63.6±3.1 <sup>abc</sup>	107±3 <sup>abcd</sup>	171±6 <sup>a</sup>	341±11 <sup>ab</sup>
Mean±SE	66.8±.2	130±0.9	235±.2	389±.4	63.6±0.8	105±.1	153±.3	322±.4

<sup>1)</sup> Weight at commencement of the experiment.

<sup>2)</sup> Control diet (treatment 14) contained 12.13 g Ca/kg diet, all other treatments contained 8.3 g Ca/kg diet.

<sup>a-f</sup> Values in a column not followed by a common letter are significantly different at P < .05.

### Feed intake and efficiency

Data of feed intake and feed efficiency (feed/gain) are given in Table 3. The lowest average daily feed intake and highest value (less efficient) for feed efficiency (3-21 days) were obtained with feeding of 300 ICU Vit D<sub>3</sub>/kg with (trt 5) or without AA (trt 1). The best feed efficiency was obtained when 1800 ICU Vit D<sub>3</sub> (trt 4) or 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub> (trt 10) was added to the basal diet (3-21 days).

### Leg condition and mortality

Leg abnormality (rickets score) and mortality data are given in Table 4. The effect of low levels of Vit D<sub>3</sub> (300 and 600 ICU/kg diet, trt 1 and 2) on leg disorders started after 9 days of age. Increasing the level of Vit D<sub>3</sub> in the diet from 300 to 600 ICU/kg diet (trt 1 and 2) significantly reduced the rickets score at 15 and 21 days of age. Increasing the level to 900 ICU Vit D<sub>3</sub>/kg diet (trt 3) resulted in no rickets at 15 days of age and minimum score of rickets was noticed at 21 days of age. Feeding 1800 ICU Vit D<sub>3</sub>/kg diet (trt 4) resulted in no rickets neither at 15 nor at 21 days of age. There were no significant differences in rickets score between 900 and 1800 ICU Vit D<sub>3</sub>/kg

diet (trt 4) resulted in no rickets neither at 15 nor at 21 days of age. There were no significant differences in rickets score between 900 and 1800 ICU Vit D<sub>3</sub>/kg diet treatments.

**Table 3. Effect of dietary Vit D<sub>3</sub>, 1,25-(OH)<sub>2</sub>D<sub>3</sub> and AA on feed intake and feed efficiency (g feed/g gain) of male turkey poults from 3 to 21 days of age**

Treatments	Daily feed intake, during days				Feed efficiency, during days			
	3-9	9-15	15-21	3-21	3-9	9-15	15-21	3-21
	g				g			
1	19.99± 0.54 <sup>ab</sup>	28.95± 0.95 <sup>cd</sup>	26.16± 2.69 <sup>d</sup>	24.96± 0.96 <sup>d</sup>	1.87±.07 <sup>b</sup>	2.11±.08 <sup>a</sup>	2.52±.22 <sup>a</sup>	2.13±.13 <sup>a</sup>
2	19.08± 0.74 <sup>ab</sup>	31.32± 0.76 <sup>bc</sup>	40.10± 2.33 <sup>c</sup>	30.17± 1.25 <sup>c</sup>	1.74±.07 <sup>b</sup>	1.94±.09 <sup>abc</sup>	1.78±.05 <sup>bc</sup>	1.82±.39 <sup>cd</sup>
3	19.54± 0.50 <sup>ab</sup>	33.41± 1.29 <sup>ab</sup>	47.15± 1.16 <sup>ab</sup>	33.25± 0.81 <sup>ab</sup>	1.76±.04 <sup>b</sup>	1.80±.05 <sup>bc</sup>	1.70±.05 <sup>bc</sup>	1.74±.03 <sup>cd</sup>
4	19.01± 0.54 <sup>ab</sup>	32.66± 0.89 <sup>ab</sup>	48.36± 1.27 <sup>ab</sup>	33.34± 0.62 <sup>ab</sup>	1.81±.05 <sup>b</sup>	1.73±.08 <sup>bc</sup>	1.61±.06 <sup>c</sup>	1.68±.03 <sup>d</sup>
5	20.05± 0.52 <sup>ab</sup>	27.04± 0.69 <sup>d</sup>	28.26± 2.64 <sup>d</sup>	24.97± 0.92 <sup>d</sup>	1.75±.06 <sup>b</sup>	2.10±.12 <sup>a</sup>	2.33±.10 <sup>a</sup>	2.01±.06 <sup>ab</sup>
6	20.52± 0.38 <sup>a</sup>	32.34± 0.65 <sup>ab</sup>	37.85± 1.40 <sup>c</sup>	30.06± 0.48 <sup>c</sup>	1.74±.05 <sup>b</sup>	2.01±.04 <sup>ab</sup>	1.96±.09 <sup>b</sup>	1.91±.06 <sup>bc</sup>
7	19.70± 0.31 <sup>ab</sup>	34.76± 0.40 <sup>a</sup>	48.77± 0.74 <sup>ab</sup>	34.41± 0.33 <sup>a</sup>	1.70±.03 <sup>b</sup>	1.88±.05 <sup>abc</sup>	1.74±.03 <sup>bc</sup>	1.78±.02 <sup>cd</sup>
8	19.12± 0.81 <sup>ab</sup>	33.70± 0.65 <sup>ab</sup>	50.59± 1.38 <sup>a</sup>	34.47± 0.61 <sup>a</sup>	1.91±.06 <sup>b</sup>	1.80±.06 <sup>bc</sup>	1.73±.08 <sup>bc</sup>	1.78±.07 <sup>cd</sup>
9	19.00± 0.58 <sup>ab</sup>	33.34± 0.46 <sup>ab</sup>	49.16± 0.46 <sup>ab</sup>	33.83± 0.49 <sup>a</sup>	1.81±.03 <sup>b</sup>	1.77±.05 <sup>bc</sup>	1.71±.10 <sup>bc</sup>	1.74±.04 <sup>cd</sup>
10	18.29± 0.37 <sup>b</sup>	31.91± 1.03 <sup>ab</sup>	47.19± 1.45 <sup>ab</sup>	32.46± 0.9 <sup>abc</sup>	1.89±.07 <sup>b</sup>	1.68±.03 <sup>c</sup>	1.62±.01 <sup>bc</sup>	1.68±.01 <sup>d</sup>
11	19.21± 1.11 <sup>ab</sup>	32.89± 0.76 <sup>ab</sup>	49.02± 1.35 <sup>ab</sup>	33.13± 1.07 <sup>ab</sup>	2.19±.24 <sup>a</sup>	1.76±.07 <sup>bc</sup>	1.64±.03 <sup>bc</sup>	1.71±.03 <sup>d</sup>
12	18.71± 0.17 <sup>ab</sup>	33.39± 0.83 <sup>ab</sup>	48.81± 0.86 <sup>ab</sup>	33.64± 0.53 <sup>ab</sup>	1.85±.06 <sup>b</sup>	1.73±.04 <sup>c</sup>	1.68±.04 <sup>bc</sup>	1.73±.03 <sup>cd</sup>
13	19.34± 0.80 <sup>ab</sup>	28.83± 1.35 <sup>cd</sup>	44.86± 1.57 <sup>b</sup>	31.01± 1.04 <sup>bc</sup>	1.97±.07 <sup>ab</sup>	1.88±.18 <sup>abc</sup>	1.78±.21 <sup>bc</sup>	1.81±.07 <sup>cd</sup>
14 <sup>1)</sup>	18.87± 0.59 <sup>ab</sup>	32.83± 0.74 <sup>ab</sup>	47.77± 1.32 <sup>ab</sup>	33.15± 0.81 <sup>ab</sup>	1.80±.08 <sup>b</sup>	1.85±.07 <sup>abc</sup>	1.70±.10 <sup>bc</sup>	1.76±.07 <sup>cd</sup>
Mean ± SE	19.31± 0.16	31.96± 0.35	43.86± 1.10	31.63± 0.45	1.84±.03	1.86±0.03	1.82±.04	1.80±.02

<sup>1)</sup> Control diet (treatment 14) contained 12.3 g Ca/kg diet, all other treatments contained 8.3 g Ca/kg diet.

<sup>a-d</sup> Values in a column not followed by a common letter are significantly different at P < .05.

The addition of AA to 600 ICU Vit D<sub>3</sub>/kg diet (trt 6) significantly increased rickets score. The addition of 1,25-(OH)<sub>2</sub>D<sub>3</sub> alone to the basal diet at 5, 10, and 20 µg/kg diet

(trt 9-11) resulted in no rickets at all ages. Also, the addition of 900 ICU Vit D<sub>3</sub> or 500 mg AA per kg diet with 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 12 and 13, respectively) resulted in no rickets. The highest mortality was 15.6% and occurred with the lowest level of Vit D<sub>3</sub> (300 ICU D<sub>3</sub>/kg diet) combined with 500 mg AA/kg (trt 5) diet. The addition of 20 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 11) resulted in 9.4% mortality.

### Bone ash and strength

Tibia and toe ash and bone breaking strength data are given in Table 4. Increasing the level of Vit D<sub>3</sub> in the diet (trt 1-4) significantly increased bone ash (tibia and toe) and strength. The values for bone ash and strength were lower when AA was added, (trt 5-8) however, there were no significant differences compared with the different levels of Vit D<sub>3</sub> alone.

**Table 4.** Effect of dietary Vit D<sub>3</sub>, 1,25-(OH)<sub>2</sub>D<sub>3</sub> and AA on rickets score, mortality rate and bone (tibia and toe) measurements of male turkey poults from 3 to 21 days of age

Treatments	Rickets score, at days		Mortality	Bone ash		Bone strength
	15	21		Tibia	Toe	
	Score <sup>1)</sup> (0-4)		-----%-----	-----%-----		Kg
1	0.54±0.12 <sup>a</sup>	1.39±0.07 <sup>a</sup>	12.5±7.2 <sup>ab</sup>	38.2±1.3 <sup>f</sup>	11.5±.3 <sup>d</sup>	2.48±.35 <sup>f</sup>
2	0.06±0.04 <sup>b</sup>	0.47±0.21 <sup>c</sup>	0 <sup>c</sup>	45.3±1.6 <sup>e</sup>	14.1±.2 <sup>c</sup>	5.71±.78 <sup>e</sup>
3	0 <sup>b</sup>	0.06±0.04 <sup>d</sup>	3.1±3.1 <sup>bc</sup>	50.3±0.3 <sup>bcd</sup>	15.5±.7 <sup>ab</sup>	9.68±.69 <sup>bcd</sup>
4	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	52.6±0.1 <sup>ab</sup>	16.2±.2 <sup>a</sup>	11.10±.55 <sup>abc</sup>
5	0.59±0.37 <sup>a</sup>	1.52±0.23 <sup>a</sup>	15.6±3.13 <sup>a</sup>	37.6±1.2 <sup>f</sup>	10.9±.4 <sup>d</sup>	2.35±.19 <sup>f</sup>
6	0.55±0.15 <sup>a</sup>	0.91±0.20 <sup>b</sup>	3.1±3.1 <sup>bc</sup>	44.1±0.9 <sup>e</sup>	13.6±.2 <sup>c</sup>	4.20±.41 <sup>ef</sup>
7	0.06±0.06 <sup>b</sup>	0.22±0.08 <sup>cd</sup>	0 <sup>c</sup>	49.0±0.5 <sup>d</sup>	15.5±.3 <sup>ab</sup>	8.00±.46 <sup>d</sup>
8	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	52.2±0.4 <sup>abc</sup>	15.6±.2 <sup>ab</sup>	10.69±.49 <sup>abc</sup>
9	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	49.9±0.9 <sup>cd</sup>	15.0±.3 <sup>b</sup>	9.08±.53 <sup>cd</sup>
10	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	52.3±0.2 <sup>ab</sup>	16.2±.4 <sup>a</sup>	10.97±.67 <sup>abc</sup>
11	0 <sup>b</sup>	0 <sup>d</sup>	9.4±9.4 <sup>abc</sup>	52.7±0.1 <sup>ab</sup>	16.1±.2 <sup>a</sup>	11.83.77 <sup>ab</sup>
12	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	53.2±0.3 <sup>a</sup>	16.2±.4 <sup>a</sup>	12.63±.42 <sup>a</sup>
13	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	52.8±0.6 <sup>ab</sup>	15.5±.2 <sup>ab</sup>	10.66±.71 <sup>abc</sup>
14 <sup>2)</sup>	0 <sup>b</sup>	0 <sup>d</sup>	0 <sup>c</sup>	53.7±0.3 <sup>a</sup>	16.0±.2 <sup>a</sup>	12.38±.57 <sup>a</sup>
Mean ± SE	0.13±0.04	0.33±0.08	3.1±1.1	48.8±0.7	14.9±.2	8.71±.31

<sup>1)</sup> Where score 0: normal legs, 1-3 degree of rickets severity, 4 bird unable to stand.

<sup>2)</sup> Control diet (treatment 14) contained 12.3 g Ca/kg diet, all other treatments contained 8.3 g Ca/kg diet.

<sup>a-d</sup> Values in a column not followed by a common letter are significantly different at P < .05.

Increasing the level of 1,25-(OH)<sub>2</sub>D<sub>3</sub> from 5 to 10 µg/kg diet (trt 10) showed a significant increase in bone ash. However, bone-breaking strength was increased but not significantly. The addition of 20 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 11) did not produce any increases in toe ash but increased tibia ash and bone strength. The combination of 900

ICU Vit D<sub>3</sub>/kg diet and 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 12) resulted in increased tibia bone ash and strength, however, toe ash was not affected, compared to 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 10). In comparison to 900 ICU Vit D<sub>3</sub>/kg diet (trt 3), there were significant increases in bone tibia ash and strength. The combination of 500 mg AA/kg diet and 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 13) had no effect on tibia and toe ash and bone breaking strength.

### Blood serum measurements

Data of serum Ca, inorganic P and alkaline phosphatase activity are given in Table 5. Serum Ca was increased significantly with increasing Vit D<sub>3</sub> level in the basal diet from 300 to 600 ICU/kg diet (trt 1 and 2). Increasing the level up to 1800 ICU/kg diet (trt 3 and 4) further increased serum Ca, but not significantly. AA reduced serum Ca concentration of the birds when fed with different levels of Vit D<sub>3</sub> (trt 5-8) or 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub> (trt 13).

**Table 5. Effect of dietary Vit D<sub>3</sub>, 1,25(OH)<sub>2</sub>D<sub>3</sub> and AA on serum calcium, inorganic phosphorus and alkaline phosphatase of male turkey poults at 21 days of age**

Treatments	Serum		
	Calcium -----mg./dl-----	Inorganic phosphorus -----mg./dl-----	Alkaline phosphatase sigma units/ml
1	5.66±.21 <sup>l</sup>	7.91±0.79 <sup>a</sup>	154.7±20.3 <sup>a</sup>
2	7.32±.24 <sup>fg</sup>	7.18±0.66 <sup>a</sup>	98.1±9.8 <sup>b</sup>
3	7.67±.28 <sup>efg</sup>	6.44±0.34 <sup>a</sup>	63.8±3.7 <sup>cd</sup>
4	8.12±.26 <sup>cde</sup>	6.61±0.64 <sup>a</sup>	49.9±6.7 <sup>d</sup>
5	4.99±.31 <sup>l</sup>	7.32±0.22 <sup>a</sup>	143.9±9.7 <sup>a</sup>
6	6.50±.38 <sup>h</sup>	6.50±0.27 <sup>a</sup>	85.9±7.9 <sup>bc</sup>
7	7.13±.11 <sup>gh</sup>	6.70±0.49 <sup>a</sup>	66.4±2.2 <sup>cd</sup>
8	7.42±.08 <sup>efg</sup>	6.96±0.25 <sup>a</sup>	60.5±3.5 <sup>d</sup>
9	8.00±.29 <sup>def</sup>	6.30±0.40 <sup>a</sup>	68.1±3.5 <sup>cd</sup>
10	8.59±.28 <sup>bcd</sup>	6.83±0.21 <sup>a</sup>	69.6±5.0 <sup>cd</sup>
11	8.83±.26 <sup>bc</sup>	6.60±0.61 <sup>a</sup>	64.5±1.8 <sup>cd</sup>
12	9.88±.05 <sup>a</sup>	7.37±0.38 <sup>a</sup>	65.2±2.1 <sup>cd</sup>
13	8.50±.16 <sup>bcd</sup>	7.20±1.18 <sup>a</sup>	63.8±3.0 <sup>cd</sup>
14 <sup>1)</sup>	9.17±.30 <sup>b</sup>	7.53±1.05 <sup>a</sup>	52.3±1.9 <sup>d</sup>
Mean±SE	7.70 ±0.18	6.96±0.15	79.1±4.5

<sup>1)</sup>Control diet (treatment 14) contained 12.3 g Ca/kg diet, all other treatments contained 8.3 g Ca/kg diet.

<sup>a-i</sup> Values in a column not followed by a common letter are significantly different at P <.05.

This decrease of serum Ca (trt 6) was significant compared with 600 ICU Vit D<sub>3</sub>/kg diet (trt 2). Increasing the level of 1,25-(OH)<sub>2</sub>D<sub>3</sub> from 5 to 10 µg/kg diet (trt 10) had no

significant effect on serum Ca, while further increase 20  $\mu\text{g}$  (trt 11) resulted in significantly higher Ca concentration in the serum. The combination of 900 ICU Vit D<sub>3</sub>/kg and 10  $\mu\text{g}$  1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 12) resulted in an increased serum Ca above any other treatment.

Serum inorganic P was not affected significantly by any of the treatments. Although in a small range, some of the differences may be biologically significant. Increasing the level of Vit D<sub>3</sub> in the diet from 300 to 900 (trt 1-3) significantly decreased alkaline phosphatase activity. Feeding 1800 ICU Vit D<sub>3</sub>/kg diet (trt 4) reduced the activity further but not significantly when compared with 900 (trt 3) or control diet (trt 14). AA (trt 5-8) had no significant effect on alkaline phosphatase activity over the addition of Vit D<sub>3</sub>. The addition of graded levels of 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 9-11) had no effect on the enzyme activity even when 10  $\mu\text{g}$  1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg combined with 900 ICU Vit D<sub>3</sub> (trt 12) or 500 mg AA (trt 13) per kg diet.

### Discussion

Adding Vit D<sub>3</sub> less than the NRC [5] recommended level (trt 1 and 2) with 8.3 g Ca/kg produced at 9 days of age normal growth and no rickets appeared in spite of the special precautions taken to exclude the ultraviolet light from the fluorescent lamps in the battery brooders of the poults. This could be explained by more than one factor: a) Reasonable carryover effect of Vit D<sub>3</sub> were reserved from the dams in the day-old poults, may be still present till 9 days of age. Stevens *et al.* [18] reported that the poults quantitative requirement for Vit D<sub>3</sub> at the early age is influenced by the amount of Vit D<sub>3</sub> present in the poult at hatching. This is an indication that Vit D<sub>3</sub> level in the breeding diet was important during the first two weeks of the new hatched turkey. After that, Vit D<sub>3</sub> content of the poults starting diet exerted its influence on growth and bone development. However, Hedstrom [19] reported that turkey poults fed a diet deficient in Vit D<sub>3</sub> developed rickets by 10 days of age. b) The effect of feeding the poults for the first three days of age on control diet, which contained 900 ICU Vit D<sub>3</sub>/kg diet.

Low supplementation of Vit D<sub>3</sub> had negative effect on all criteria. Feeding dietary levels of Vit D<sub>3</sub> less than 900 ICU/kg (trt 1 and 2) decreased body weight, gain, feed intake, bone ash, tibial breaking strength, and serum Ca and increased alkaline phosphatase activity and rickets score. Feeding Vit D<sub>3</sub> at dietary level of 900 ICU/kg diet (trt 3) increased gain, bone ash (tibia and toe), bone breaking strength and serum Ca. The severity of rickets and the activity of serum alkaline phosphatase decreased with increasing Vit D<sub>3</sub> levels. The similarity in growth performance between 900 ICU Vit D<sub>3</sub>/kg level (trt 3) and control diet (trt 14) could indicate that 8.3 g Ca/kg diet is adequate with 900 ICU Vit D<sub>3</sub> for growth but not enough for bone development and freedom from rickets. When Vit D<sub>3</sub> was fed at higher level (1800 ICU/kg, trt 4), compared to 900 ICU/kg, (trt 3) body weight gain values were similar but there were consistent improvement in bone ash, bone breaking strength and serum Ca with no

rickets observed. This indicates that 900 ICU Vit D<sub>3</sub>/kg diet met the requirements for gain with ultraviolet light excluded, but more was needed for bone development. Higher level of Vit D<sub>3</sub> has a sparing effect on Ca level, since the main established role of Vit D<sub>3</sub> is to stimulate Ca-binding protein and enhance Ca absorption from the intestine [8]. Also, bone ash values were more responsive to Vit D<sub>3</sub> manipulation than body weight. Similar results with poults and chicks were found in other studies [9, 10, 20- 24]. These results are confirmed by the NRC [6], where the requirement of Vit D<sub>3</sub> for turkey poults were increased from 900 ICU/kg by the NRC [5] to 1100 ICU/kg. NRC [6].

Supplementation of the diet with graded levels of 1,25-(OH)<sub>2</sub>D<sub>3</sub> over 5 µg/kg (trt 10 and 11) had no significant effect on gain, alkaline phosphatase activity, serum P or occurrence of rickets. Similar results were reported by others [10, 22, 25]. The addition of 1,25-(OH)<sub>2</sub>D<sub>3</sub> at 5 µg/kg diet (trt 9) improved all criteria comparing with the low levels of Vit D<sub>3</sub> (7.5 and 15.0 µg/kg, Trt 1 and 2), and was comparable to the higher levels of Vit D<sub>3</sub> (trt 3 and 4) and control (trt 14). This indicates that 1,25-(OH)<sub>2</sub>D<sub>3</sub> has more activity than Vit D<sub>3</sub> in improving growth, rickets free, maintaining bone ash and enhancing Ca mobilization in turkey. These results were in accordance with previous observation [26] in chickens and rats, and [10, 27] in chickens.

In order for Vit D<sub>3</sub> to carry out its physiological function, it must undergo a two-step hydroxylation; first in the liver to produce 25-OH D<sub>3</sub>, the major circulating form of Vit D<sub>3</sub>, followed by a second hydroxylation in the kidney to produce 1,25-(OH)<sub>2</sub>D<sub>3</sub> the biologically active hormonal form of Vit D<sub>3</sub>. This hormonal form has a key role in Ca homeostasis, directly controlling the absorption of dietary Ca from the small intestine and the resorption of Ca from bone as well as influencing reabsorption of Ca from the proximal tubules of the kidney [8]. The rapidly growing young broiler is unable to produce 1,25-(OH)<sub>2</sub>D<sub>3</sub> from dietary Vit D<sub>3</sub> efficiently enough to stimulate maximum Ca absorption and bone formation [21, 28]. On the other hand, Hedstrom [19] reported that Vit D<sub>3</sub> was more effective than 1,25-(OH)<sub>2</sub>D<sub>3</sub> when fed to turkey.

Increasing the level from 5 to 10 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 10) increased tibia and toe ash, breaking strength and serum Ca. These results were likely due to the direct effect of 1,25-(OH)<sub>2</sub>D<sub>3</sub> on both increasing Ca absorption utilization and reutilization during modeling in bone development. The addition of 20 µg 1,25(OH)<sub>2</sub>D<sub>3</sub>/kg diet (trt 11) did not produce any depression on growth and had no significant effect over 10 µg/kg diet. In the same order, no significant reduction on growth of chick with increasing the level of 1,25-(OH)<sub>2</sub>D<sub>3</sub> over 5 µg/kg as was shown by some investigators [22, 29], while others have reported a significant depression in growth with higher level of 1,25-(OH)<sub>2</sub>D<sub>3</sub> [ 30, 31, 24]. The toxic level of 1,25-(OH)<sub>2</sub>D<sub>3</sub> was reported to be 15 µg/kg in broiler diets. Edwards et al [10] reported that the addition of 3 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet of boilers increased growth rate, while 15 µg decreased it. Maximum bone ash was

obtained from 3  $\mu\text{g}/\text{kg}$  supplementation of 1,25-(OH) $_2\text{D}_3$ , while supplementation with 9, 12 and 15  $\mu\text{g}/\text{kg}$  actually decreased bone ash from this maximum values. Xu et al. [30] found that a toxic level of 1,25-(OH) $_2\text{D}_3$  (15  $\mu\text{g}/\text{kg}$ ) resulted in poor growth rate and feed efficiency of the broilers.

The variation in the results between different reports might be due to differences in Ca level of the diets. Mitchell and Edwards [32] indicated that lowering the dietary Ca level may prevent some of the decreases in body weight in birds given 1,25-(OH) $_2\text{D}_3$ . It could be that increasing the level of 1,25-(OH) $_2\text{D}_3$  increased Ca absorption and this resulted in hypercalcemia and depressed P concentration in blood. All the mentioned reports were done on chickens and to our knowledge no work on the use of 1,25-(OH) $_2\text{D}_3$  alone on turkey was reported yet.

The combination of 10  $\mu\text{g}$  1,25-(OH) $_2\text{D}_3/\text{kg}$  diet (trt 12) improved gain, tibia and toe ash, and increased serum Ca and breaking bone strength over 10  $\mu\text{g}$  1,25-(OH) $_2\text{D}_3$  (trt 10) or 900 ICU Vit D $_3$  (trt 3) alone and over the control (trt 14). In good agreement with the present results, [10, 30] on chick and [9] with the turkey.

In this experiment, the addition of 500 mg AA/kg diet to graded levels of Vit D $_3$  (trt 5-8) or 10  $\mu\text{g}$  1,25-(OH) $_2\text{D}_3/\text{kg}$  diet (trt 13) had no beneficial effect on all criteria measured. The lack of AA effect on body weight gain was in contradiction with previous work where level of 0.5% dietary AA decreased body weight gain in young coturnix and poults compared with those which were not supplemented with AA [33]. AA is required for the hydroxylation of proline for the production of the collagen matrix of bone. The results from the present study indicated that AA does not alter the development of bone with Vit D $_3$ -deficient diet or with higher levels of Vit D $_3$  or 1,25-(OH) $_2\text{D}_3$  in the first 3 weeks of age. This is in agreement with others [34,35]. Although poultry can produce AA endogenously, stress, feed or environmental factors can interfere with this process. Weiser, et al. [12] reported a stimulating or synergistic effect of AA with Vit D $_3$  and 1,25-(OH) $_2\text{D}_3$  in broiler chicks. Broilers at early age (2-3 weeks) can not synthesis AA at a high rate and that the availability of endogenously synthesized AA is low. Hence, young chicks may benefit from addition of AA to the diet in the sense that it synergistically affects the synthesis of 1,25-(OH) $_2\text{D}_3$  [13].

The high activity of alkaline phosphatase was accompanied with severe rickets, i.e., birds fed a low level of Vit D $_3$  had both high rickets score and high alkaline phosphatase activity. Mos and Henderson [36] reported that alkaline phosphatase enzyme is involved in the calcification process in bone (osteoblasts). In rickets, alkaline phosphatase activity two to four times normal may be observed, and these fall to normal upon treatment with Vit D $_3$ . The graded levels of 1,25-(OH) $_2\text{D}_3$  above 5  $\mu\text{g}/\text{kg}$  did not affect the alkaline phosphatase activity. Similar finding was reported by others [22, 37, 38].

### Conclusion

This research indicated that: (1). The use of 1,25-(OH)<sub>2</sub>D<sub>3</sub> as a dietary supplement to prevent rickets and to enhance bone development in turkey is promising. (2) 5 µg 1,25-(OH)<sub>2</sub>D<sub>3</sub>/kg diet met the requirements of the turkey poult for gain and rickets prevention but more was required for maximum bone ash and breaking strength. (3). The addition of AA was not beneficial and did not influence the requirement of Vit D<sub>3</sub> and more studies on the role and levels of AA in Vit D and Ca metabolism of the turkey are needed.

### References

- [1] Deluca, H.F. *Vit D<sub>3</sub> Metabolism and Function. 1. Monographs on Endocrinology* Vo.. 13. New York: Springer- Verlag, N.Y., 1979.
- [2] Bar, A.S., Hurwitz, S. and Maoz, A. "The 25- hydroxycalciferol. 1. Hydroxylase Activity of Chick's Kidney Cells; Direct Effect of Parathyroid". *FEBS, Letters*, 113 (1980), 328-330.
- [3] Motzok, I. "Factors Affecting the Utilization of Calcium and Phosphorus from Soft Phosphate by Chicks". *Poultry Sci.*, 44 (1965), 1261-1270.
- [4] Fritz, J.C.; Roberts, T.; Bochner, J. W., and Hove, E.L. "Factors Affecting the Chick's Requirement for Phosphorus". *Poultry Sci.*, 47 (1968), 307-320.
- [5] National Research Council (NRC). *Nutrient Requirements of Poultry*. 8th rev. ed. Washington, DC: National Academy Press, 1984.
- [6] National Research Council (NRC). *Nutrient Requirements of Poultry*. 9th rev. ed. Washington, DC: National Academy Press, 1994.
- [7] Neagle, L.H., Blaylock, L. G. and Goihl, J.H. "Calcium, Phosphorus and Vitamin D<sub>3</sub> Level and Interactions in Turkeys to 4 Weeks of Age". *Poultry Sci.*, 47 (1968), 174-180.
- [8] Deluca, H.F. Vitamin D "The Vitamin and the Hormone". *Federation Proceedings*, 33 (1974), 2211-2219.
- [9] Sanders, A.M. and Edwards, H.M. Jr. "The Effect of 1,25-dihydroxycholecalciferol on Performance and Bone Development in the Turkey Poult". *Poultry Sci.*, 70 (1991), 853-866.
- [10] Edwards, H. M. Jr., Elliot, M.A. and Sooncharemying, S. "Quantitative Substitution of 1,25-dihydroxycholecalciferol and 1- hydroxy-cholecalciferol for Cholecalciferol in Broiler Diets". *Proceeding XIX World's Poultry Congress*, Amsterdam, Netherlands, Vol. 1 (1992), 567-571.
- [11] Weiser, H. and Schlachter, M. *Combined Use of Vitamin D<sub>3</sub>, Vitamin D<sub>3</sub> Metabolites and Vitamin C in Bone Metabolism. Generalized Bone Diseases*. Berlin – Heidelberg, Gemany: Springer Verlag, 1987. pp. 71-76.
- [12] Weiser, H., Schlachter, M. and Bachmann, H. The Importance of Vitamin C for Hydroxylation of Vitamin D<sub>3</sub> to 1 α, 25 (OH)<sub>2</sub>D<sub>3</sub> and 24, 25 (OH)<sub>2</sub>D<sub>3</sub> to a More Active Metabolite. *Molecular, Cellular and Clinical Endocrinology*. Berlin, Germany: Walter de Gruyter and Co., 1988. P.644- 653.
- [13] Bains, B.S. "Broilers Suffer from Dyschondroplasia and Femoral Necrosis". *World Poultry Misset*, 10 (1994), 109-111.
- [14] Atia, Faten. A.M. "Effect of Feeding Different Levels of Phytase Enzyme and Vitamin D<sub>3</sub> on Growing Turkey." *Ph.D. Thesis*, College of Agric., Suez Canal Univ., (1998).
- [15] Bliss, C.I. " The Combined Slope in Comparative Tests of Tibia and Toe Ash in the Chick Assay for Vitamin D". *Poultry Sci.*, 24 (1945), 534-541.
- [16] *SAS User's Guide. Basics, Statistics*. Version 5. SAS Institute, Inc., Cary, NC, USA, (1985).
- [17] Duncan, D.B.; " Multiple Range and Multiple F Tests". *Biometrics*, 11 (1955), 1-42.
- [18] Stevens, V. I.; Blair, R., and Salmon, R.E. " Influence of Maternal Vitamin D<sub>3</sub> carryover on Kidney 25-hydroxyvitamin D<sub>3</sub>-1-hydroxylase Activity of Poults". *Poultry Sci.*, 63 (1984), 765.
- [19] Hedstrom, O.R. " Pathology of Vitamin D Deficiency in Growing Turkeys". *Dissertation Abstracts International, B.*, 47, 4 (1986), 1446.

- [20] Perry, R.W.; Rowland, G.N., and Britton, W.M. : “ Pathology of Experimental Vitamin D Deficiency in Turkeys and the Effects of Various Vitamin D Supplements”. *Avian Diseases*, 35 (1991), 542-553.
- [21] Elliot, M.A. Effect of Fluorescent Lighting, Strain, Cholecalciferol and 1,25-dihydroxycholecalciferol on the Development of Tibial Dyschondroplasia in Chickens.” *Ph. D. Thesis*, University of Georgia, Athens, G.A., (1992).
- [22] Rennie, J.S.; Whitehead, C.C. and Thorp, B. H. “ The Effect of Dietary 1,25- dihydroxycholecalciferol in Preventing Tibial Dyschondroplasia in Broilers Fed on Diets Imbalanced in Calcium and Phosphorus”. *Br. J. Nutr.*, 69 (1993), 809-816.
- [23] Edwards, H. M.Jr., Elliot, M.A., Sooncharenying, S. and Britton, W.M. “ Quantitative Requirement for Cholecalciferol in the Absence of Ultraviolet Light”. *Poultry Sci.*, 73 (1994), 288-294.
- [24] Mitchell, R.D., Edwards, H.M. Jr., McDaniel, G.R. and Rowland, G.N. “ Dietary 1,25-dihydroxycholecalciferol has Variable Effects on the Incidences of Leg Abnormalities, Plasma Vitamin D Metabolites and Vitamin D Receptors in Chicken Divergently Selected for Tibial Dyschondroplasia”. *Poultry Sci.*, 76 (1997), 338-345.
- [25] Roberson, K.D. and Edwards, H. M. Jr. “ Effect of Dietary 1,25- dihydroxycholecalciferol Level on Broiler Performance”. *Poultry Sci.*, 75 (1996), 90-94.
- [26] Norman, A.W. and Wong, R.G. “ Biological Activity of the Vitamin D Metabolite 1,25-dihydroxycholecalciferol in Chickens and Rats”. *J. Nutr.*, 102 (1972), 1709-1718.
- [27] Boris, A.; Hurley, J. F. and Trmal, T. “Relative Activities of Some Metabolites and Analogs of Cholecalciferol in Stimulation of Tibia Ash Weight in Chicks Otherwise Deprived of Vitamin D.” *J. Nutr.*, 107 (1977), 194-198.
- [28] Elliot, M.A. and Edwards, H.M. Jr. “ Effect of Genetic Strain, Calcium and Feed Withdrawal on Growth, Tibial Dyschondroplasia, Plasma 1,25- dihydroxycholecalciferol, and Plasma 25-hydroxycholecalciferol in Sixteen – Day – Old Chickens”. *Poultry Sci.*, 73 (1994), 509-519.
- [29] Thorp, B.H., Ducro, B., Farquharson, C., Sorensen P., and Whitehead, C.C. “ Avian Tibial Dyschondroplasia. “The Interaction of Genetic Selection and Dietary 1,25- dihydroxycholecalciferol”. *Avian Pathol.*, 22 (1993), 311-324.
- [30] Xu, T., Soares, J.H. Leach, R.M., Kerr, Jr., J. and Hollis, B. “ Evidence of Vitamin D Resistance in Chicks with Tibial Dyschondroplasia”. *Poultry Sci.*, 72, Suppl. I (1993), 109.
- [31] Mitchell, R.D., Edwards, H.M. Jr. and McDaniel, G.R. “Effect of Dietary 1,25-dihydroxycholecalciferol and Genetic Tendency for Tibial Dyschondroplasia on Leg Abnormalities, Plasma Vitamin D Metabolites and Vitamin D Receptors”. *Poultry Sci.*, 74: Suppl., I (1995), 315.
- [32] Mitchell, R.D., and Edwards, H. M. Jr. “ Effect of Phytase and 1,25-dihydroxycholecalciferol on Phytate Utilization and Quantitative Requirement for Calcium and Phosphorus in Young Broiler Chickens”. *Poultry Sci.*, 75 (1996), 95-110.
- [33] Spivey – Fox, M.R., Fry, B.E., Harland, B.F., Shertel, M.E., and Week, C.E. “ Effect of Ascorbic Acid on Cadmium Toxicity in the Young Coturnix”. *J. Nutr.*, 101 (1971), 1295-1305.
- [34] Edwards, H. M. Jr. “ Effect o Vitamin C, Environmental Temperature, Chlortetracycline and Vitamin D<sub>3</sub> on the Development of Tibial Dyschondroplasia in Chickens”. *Poultry Sci.*, 68 (1989), 1527-1543.
- [35] Farquharson, C., Rennie, J. S., Loveridge, N., and Whitehead, C.C. “ Effect of Ascorbic Acid and 1,25-(OH)<sub>2</sub>D<sub>3</sub> on Bone Cell Metabolism in Relation to the Development of Tibial Dyschondroplasia”. *World Poultry Sci.*, Association UK Branch Spring Meeting, (1993), 23-24.
- [36] Mos, D.W. and Henderson, A.R. Enzymes Chapter 19 in Tietz Fundamentals of Clinical Chemistry. 4<sup>th</sup> ed., Editors: Burtis, C.A. and E.R. Ashwood. W. B. Saunders Company, Philadelphia, USA. 1996.
- [37] Rennie, J.S. “ Vitamin D Metabolites and the Prevention of Tibial Dyschondroplasia”. *Proc. 9<sup>th</sup> European Poultry Conference Glasgow 7-12<sup>th</sup> Aug.* (1994), 207-210.
- [38] Roberson, K. D., and Edwards, H.M. Jr. “ Effects of Ascorbic Acid and 1,25- dihydroxycholecalciferol on Alkaline Phosphatase and Tibial Dyschondroplasia in Broiler Chickens”. *Br. Poultry Sci.*, 73 (1994), 763-773.

## تأثير التغذية على فيتامين د<sub>3</sub> ، ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل وحمض الأسكوربيك على النمو وتكلس العظم في الرومي النامي

إسماعيل حافظ هرمس<sup>(١)</sup>، بول أ وييل<sup>(٢)</sup>، فاتن عبد العزيز عطية<sup>(٣)</sup>، هاني محمد صبري<sup>(٤)</sup>

<sup>(١)</sup> قسم إنتاج وتربية الحيوان - كلية الزراعة والطب البيطري، جامعة الملك سعود القصيم، بريدة،  
المملكة العربية السعودية.

<sup>(٢)</sup> قسم علم الحيوان، جامعة مينسوتا، الولايات المتحدة الأمريكية.

<sup>(٣)</sup> قسم الإنتاج الحيواني، كلية الزراعة، جامعة قناة السويس، الإسماعيلية، جمهورية مصر العربية.

(قدم للنشر في ١/١/١٤٢٠هـ؛ وقبل للنشر في ١٢ / ٨ / ١٤٢٠هـ)

**ملخص البحث.** أجريت هذه التجربة لدراسة إضافة فيتامين د<sub>3</sub> ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل (الصورة النشطة من فيتامين د<sub>3</sub>) وحمض الاسكوربيك على أداء النمو وتطور العظم في ذكور الرومي الأبيض كبير الحجم وذلك من عمر ٣ أيام إلى ٢١ يوم. تم تربية صغار الرومي في بطاريات مزودة بتدفئة كهربائية وكذلك تم تغطية الإضاءة الفوريستت بأغطية لحجز الأشعة فوق البنفسجية. صممت التجربة من ١٣ معاملة بالإضافة إلى الكنترول الموجب، وكل معاملة بها أربعة مكررات بكل مكررة ثمانية طيور. تكونت العليقة الأساسية من الذرة الصفراء وكسب فول الصويا، احتوت على ٠.٨٣، كالسيوم، ٠.٦٦، فسفور مستفاد. تمت التغذية على فيتامين د<sub>3</sub> بتركيزات ٣٠٠، ٦٠٠، ٩٠٠، ١٨٠٠ وحدة دولية لكل كيلو جرام عليقه بمفرده أو مع إضافة ٥٠٠ ملجم حامض الاسكوربيك لكل كيلو جرام عليقه، كذلك تمت التغذية على ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل بمفرده بتركيزات ٥، ١٠، ٢٠ ميكروجرام لكل كيلو جرام عليقه وأيضاً التركيز ١٠ ميكرو جرام ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل مع ٩٠٠ وحدة دولية من فيتامين د<sub>3</sub> بمفرده أو مع ٥٠٠ ملجم حمض الاسكوربيك لكل كيلو جرام عليقه. أظهرت النتائج أن التغذية على ٩٠٠ وحدة دولية من فيتامين د<sub>3</sub> بمفرده أو مع إضافة حمض الاسكوربيك أدى إلى الزيادة في الوزن وكانت قريبة من أعلى زيادة متحصل عليها. بينما كانت أعلى زيادة في الوزن مع عدم ظهور كساح عند التغذية على جميع مستويات ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل بمفرده أو عند إضافة المستوى ١٠ ميكروجرامات مع ٩٠٠ وحدة دولية من فيتامين د<sub>3</sub>. عند التغذية على ٩٠٠ وحدة دولية من فيتامين د<sub>3</sub> أو ٥ ميكروجرامات من ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل لكل كيلو جرام عليقه انخفض رماد العظم (عظم الفخذ والإصبع) وقوة كسر العظم. لم يعطي حامض الاسكوربيك أي تأثير معنوي على أي من القياسات.

وتوصى هذه الدراسة بالآتي: ١- إضافة خمسة ميكروجرام من ١-٢٥ كولي كالسيفرول ثنائي الهيدروكسيل أو ٩٠٠ وحدة دولية من فيتامين د<sub>3</sub> لكل كيلو جرام عليقه غطت احتياجات الرومي الأبيض كبير الحجم وذلك من عمر ٣-٢١ يوم بالنسبة لزيادة الوزن وعدم ظهور الكساح ولكن لم تكن كافية للحصول على أقصى رماد بالعظم وأقصى قوة كسر للعظم.

٢- حامض الاسكوربيك لم يكن له أي تأثير على الاحتياجات من فيتامين د<sub>3</sub>.