ISSN 1682-8356
ansinet.org/ijps

INTERNATIONAL JOURNAL OF

POULTRY SCIENCE

ANSInet
308 Lasani Town, Sargodha Road, Faisalabad - Pakistan
Mob: +92 300 3008585, Fax: +92 41 8815544
E-mail: editorijps@gmail.com

International Journal of Poultry Science 4 (2): 85-90, 2005
ISSN 1682-8356
Asian Network for Scientific Information, 2005

Studies on the Efficacy of Twenty-Five-Hydroxycholecalciferol to Prevent Tibial Dyschondroplasia in Ross Broilers Fed Marginal Calcium to Market Age

K.D. Roberson[1], M.F. Ledwaba and R.A. Charbeneau
Department of Animal Science, Michigan State University, East Lansing, MI, 48824, USA
E-mail: robers22@msu.edu

Abstract: Two experiments were conducted to evaluate the effect of 25-hydroxycholecalciferol [25-(OH)D_3] on tibial dyschondroplasia (TD) in broilers grown to market age. In Experiment 1, Ross cockerels were fed a mash diet marginal in calcium and adequate in phosphorus and cholecalciferol to 35 days of age in battery cages. Dietary 25-(OH)D_3 was fed at 0 or 40 ug/kg during the starter (1-17 days) and grower (18-35 days) phases. During the grower phase, there was third treatment in which 25-(OH)D_3 was fed to 17 days of age and then the control diet was fed to 35 days of age. Phytate phosphorus retention was measured at the end of each phase. In Experiment 2, Ross straight-run broilers were grown on litter and fed marginal calcium diets that were either crumbled (starter, 1-16 days) or pelleted (grower, 17-42 days; finisher 43-49 days). Dietary 25-(OH)D_3 was fed at 0 or 69 ug/kg or a third treatment consisted of 69 ug/kg 25-(OH)D_3 to 16 days and the control diet to 49 days of age. Dietary 25-(OH)D_3 did not affect growth performance or incidence of TD in either experiment. In Experiment 1, phytate phosphorus retention was increased by feeding 25-(OH)D_3 only in the grower phase. Bone ash at 35 days in Experiment 1 and bone strength at 48 days in Experiment 2 were not affected. The results show that Ross broilers have a low incidence of TD when marginal calcium is fed and do not require dietary 25-(OH)D_3 to prevent TD or improve bone strength.

Key words: 25-cholecalciferol, calcium, phytate, Ross, tibial dyschondroplasia

Introduction

Tibial dyschondoplasia (TD) is a common abnormality found in rapidly growing meat-type poultry, is characterized by a mass of unvascularized growth plate cartilage, and is influenced by nutrition, management, and genetics (Leach and Nesheim, 1965; Edwards, 1984; Leach and Lilburn, 1992). The bone is more prone to deformities and breakage especially during processing, which can lead to downgrading of the carcass or condemnation (Burton *et al*., 1981). Although multiple factors that cause TD to occur have been identified, the exact etiology is still not understood.

Dietary supplementation with various cholecalciferol metabolites to low-calcium diets as well as to diets adequate in calcium can reduce the incidence and severity of TD (Edwards, 1989, 1990; Rennie *et al*., 1993; Rennie and Whitehead, 1996; Roberson and Edwards, 1996; Mitchell *et al*., 1997; Xu *et al*., 1997; Zhang *et al*., 1997). The cholecalciferol metabolite, 25-hydroxycholecalciferol [25-(OH)D_3], is available commercially and has been reported to alleviate the incidence and severity of TD (Mireles *et al*., 1996; Rennie and Whitehead, 1996). Others have reported that 25-(OH)D_3 reduces TD in broilers selected for low incidence of TD but not in broilers selected for high incidence of TD (Mitchell *et al*., 1997; Zhang *et al*., 1997). Ledwaba and Roberson (2003) reported that 25-(OH)D_3 reduced TD in Ross cockerels only when dietary calcium was 0.85% or lower in a broiler starter diet.

Recently, researchers have shown that 25-(OH)D_3 can improve phosphorus utilization in broiler chicks (Applegate *et al*., 2000; Angel *et al*., 2001; Edwards, 2002). Phosphorus pollution of water has been blamed partially on the poultry industry due to run-off from fields to which manure or litter has been used as fertilizer. However, the ability of 25-(OH)D_3 to improve phytate phosphorus retention has not been consistent (Edwards, 2002; Ledwaba and Roberson, 2003). Ledwaba and Roberson (2003) reported that phytate phosphorus retention was improved when a TD-inducing (low calcium) diet was fed, but not when calcium was fed at 0.85% or higher as would be expected in a broiler starter diet.

Previous studies on the requirement of broiler chicks for 25-(OH)D_3 demonstrated maximal dose-response effects on weight gain, feed efficiency, and breast meat yield between 50 and 70 ug/kg feed with no supplemental cholecalciferol in the diet (Yarger *et al*., 1995b). The basal dosage level of 69 ug/kg feed has been used in other studies (Yarger *et al*., 1995a; Mireles *et al*., 1996) because this level is the average of cholecalciferol fed in commercial broiler diets in the U.S. as reported by Yarger *et al*. (1995b). However, later studies investigating the effect of dietary 25-(OH)D_3 on TD incidence and severity suggest that the requirement for 25-(OH)D_3 may be lower (Mitchell *et al*., 1997) when adequate cholecalciferol is in the diet. Ledwaba and Roberson (2003) observed no difference between 40 or 70 ug/kg 25-(OH)D_3 on the incidence of TD in Ross broilers fed a low (0.67%) or marginal (0.85%) calcium

diet.

The objective of this study was to determine if dietary 25-(OH)D_3 needed to be fed during the entire grow-out as recommended by the manufacturer of the vitamin D metabolite to prevent TD and improve bone strength in broilers grown to various market ages. Dietary 25-(OH)D_3 was also provided during the starter phase only when the incidence of TD would be expected to be higher. The first experiment was conducted in cages to study further the ability of 25-(OH)D_3 to release phytate phosphorus in corn and soybean meal.

Materials and Methods

Experiment 1: A total of 240 1-day-old male broiler chicks were obtained from a commercial hatchery[2] for phase I of the experiment. The birds were of Ross X Ross strain and were placed at 10 chicks per pen in an electrically heated battery brooder[3] that contained fluorescent lights. The fluorescent bulbs were 15 W (F15T12) cool white[4] bulbs that provided 3.7% of the watts in the ultraviolet light range (260 to 400 nm). Fluorescent tubes in all pens were covered with plastic filters[5] to block emission of ultraviolet radiation as reported previously (Ledwaba and Roberson, 2003). The room in which the brooder was kept contained no windows and incandescent lighting was provided from the ceiling. Room temperature was maintained at 23°C and lighting was continuous. Feed and water were provided at all times. The compositions of the corn-soybean meal based diets are listed in Table 1. Dietary 25-(OH)D_3 premix was donated[6] and delivered to a commercial feed mill. Dietary treatments consisted of: 1) control diet with dietary calcium fed at a marginal level [compared to NRC (1994) recommendations] that may be used in the commercial U.S. broiler industry; 2) 25-(OH)D_3 fed at 40 ug/kg to 17 days of age, then fed control diet to 35 days of age; or 3) 40 ug/kg 25-(OH)D_3 fed 1 to 35 days of age. The control diet was fed to 8 pens of 10 chicks each during the starter phase and dietary 25-(OH)D_3 was fed to 16 pens of 10 chicks each. Chromic oxide was used as an external indicator at 0.10% of the basal diet to determine phytate phosphorus retention.

At 17 days of age, all birds were weighed individually and half the birds in each pen were killed by cervical dislocation. The right tibia of each bird killed was scored for incidence of TD as described by Edwards and Veltmann (1983). Two severity score indexes were used which have been previously described (Ledwaba and Roberson, 2003). Scoring index number 1 averages actual lesions found per pen (but counts a pen without lesions as a zero score) and scoring index number 2 takes into account all birds regardless of presence or absence of TD. The left tibia of each bird was saved for bone ash analysis (AOAC, 1995). Feed consumption was recorded to calculate conversion of feed to gain. Excreta were collected from each pen over a 24-hr period at the end of each phase for phytate phosphorus and chromic oxide determination. The samples were dried at 50 C in forced-air oven.

The remaining birds were housed in a non-heated grower cage and fed either 0 or 40 ug/kg 25-(OH)D_3 to 35 days of age. Pen-mates from two starter pens per treatment were mixed in a grower pen at 7 birds per pen. The control diet was fed to 8 pens and 40 ug/kg 25-(OH)D_3 was fed to four pens of birds. At 35 days of age, all birds were weighed and the same parameters were measured as for birds at 17 days of age.

Feed and excreta samples were ground through a 1-mm screen[7] and then through a 0.5-mm screen[8]. Samples were digested in a microwave[9] for nutrient analyses. Chromic oxide was measured by the method of Williams *et al.* (1962) and phytate phosphorus content was determined as described by Latta and Eskin (1980). Phytate phosphorus retention was calculated using the chromic oxide balance method (Edwards and Gillis, 1959). Feed calcium was determined by atomic absorption spectrophotometry[10] in 1.0% La solution (26.74 g $LaCl_3$/L of physiological saline) using the method described by the manufacturer of the atomic absorption spectrophotometer. Feed phosphorus was determined colorimetrically[11] using the method described by Gomori (1942).

Data were analyzed by ANOVA with the general linear models procedure of SAS software (SAS, 2003) using pen as the experimental unit. Treatment means were separated by the Student-Newman-Kuels test when treatment differences were significant ($p<0.05$). Because there were no differences in results for percentage data when arc sine transformations were used, analyses of actual percentage data are shown.

Experiment 2: A total of 2100 straight-run broiler chicks were obtained from a commercial hatchery[12] and placed at 1-day of age. Chicks were feather-sexed and males and females were placed evenly in each pen. Chicks were brooded in 16 pens (2.46 m X 3.08 m) at about 130 chicks per pen to 16 days of age. Each pen was split in half and spread to 32 pens at 16 days to provide approximately 65 birds per pen and birds were grown to 49 days of age. The composition of the control diet for each phase is listed in Table 1. Dietary treatments consisted of: 1) control diet with dietary calcium fed at a marginal level for 49 days; 2) 25-(OH)D_3 fed at 69 ug/kg to 16 days of age, then fed control diet to 49 days of age; or 3) 69 ug/kg 25-(OH)D_3 fed 1 to 35 days of age. There were 8 pens of birds each for Treatments 1 and 3 during the starter phase. During the grower and finisher phases, there were 16 pens of control birds, and 8 pens each of Treatments 2 and 3. Dietary 25-(OH)D_3 was fed at the manufacturer's recommended level for commercially grown broilers. The feed was manufactured at a commercial feed mill[13] with pelleting capability. The starter feed was delivered as crumbles and the grower and finisher feeds were provided as

Table 1: Composition (%) of the experimental diets

Ingredient	Experiment 1		Experiment 2		Finisher
	Starter	Grower	Starter	Grower	
Ground yellow corn	51.00	61.13	56.90	61.40	65.90
Soybean meal (dehulled)	39.00	31.12	32.00	27.10	22.15
Wheat middlings	0.00	0.00	2.50	3.00	3.50
Porcine meat and bone meal	0.00	0.00	3.00	3.00	3.00
Soybean oil	6.07	4.32	0.00	0.00	0.00
Animal/vegetable fat	0.00	0.00	3.00	3.13	3.25
Dicalcium phosphate	1.70	1.21	1.00	0.78	0.55
Limestone	0.98	1.06	0.68	0.78	0.88
Salt	0.45	0.40	0.40	0.40	0.40
DL-methionine	0.20	0.16	0.25	0.20	0.15
Vitamin premix[1,2]	0.25	0.25	0.12	0.12	0.12
Trace mineral premix[3,4]	0.25	0.25	0.10	0.10	0.10
Choline chloride	0.00	0.00	0.05	0.05	0.00
Chromic oxide	0.10	0.10	0.00	0.00	0.00
Calculated composition					
Crude protein	23.00	20.00	22.00	20.00	18.00
ME, kcal/kg	3200	3200	3100	3150	3200
Calcium	0.85	0.76	0.85	0.82	0.80
Nonphytate phosphorus	0.45	0.35	0.45	0.40	0.35
Analyzed composition					
Calcium	0.94	0.80	0.89	0.85	0.82
Phosphorus	0.70	0.62	0.73	0.67	0.61
Phytate phosphorus	0.27	0.25	0.26	0.25	0.22

[1]Vitamin premix provided per kg of diet in Experiment 1: vitamin A (all-trans-retinyl acetate), 5500 IU; cholecalciferol, 2200 ICU; vitamin E (all-rac-α-tocopherol acetate), 11 IU; menadione (as menadione sodium bisulfite), 1.1 mg; riboflavin, 4.4 mg; Ca pantothenate, 10 mg; nicotinic acid, 44 mg; choline chloride, 600 mg; vitamin B_{12}, 0.01 mg; vitamin B_6, 3 mg; thiamin (as thiamin mononitrate), 2.2 mg; folic acid, 3 mg; biotin, 0.3 mg; and ethoxyquin, 125 mg.

[2]Vitamin premix provided per kg of diet in Experiment 2: vitamin A (all-trans-retinyl acetate), 8800 IU; cholecalciferol, 4000 ICU; vitamin E (all-rac-α-tocopherol acetate), 28 IU; menadione (as menadione sodium bisulfite), 2.2 mg; riboflavin, 8.0 mg; Ca pantothenate, 16 mg; nicotinic acid, 64 mg; vitamin B_{12}, 0.02 mg; vitamin B_6, 3.4 mg; thiamin (as thiamin mononitrate), 2.3 mg; folic acid, 1.8 mg; biotin, 0.16 mg; selenium, 0.22 mg; and ethoxyquin, 100 mg.

[3]Mineral premix supplied per kg of diet in Experiment 1: manganese, 120 mg; zinc, 100 mg; iron, 60 mg; copper, 10 mg; iodine, 2.1 mg; and selenium, 0.1 mg.

[4]Mineral premix supplied per kg of diet in Experiment 2: manganese, 80 mg; zinc, 80 mg; iron, 40 mg; copper, 8 mg; and iodine, 0.9 mg.

3/16-inch pellets.

Lighting was from incandescent bulbs only (1 over each pen). The birds were given 24 hr light the first four days and then 23 hr L: 1 hr D the rest of the grow-out. Room temperature was set at 28.3°C the first week and heat lamps were available. After 7 days, the heat lamps were removed and room temperature was reduced by about 1°C each week until room temperature was set at 23°C at 35 days of age.

Body weight and feed consumption were measured at 16, 42 and 49 days of age and feed conversion was calculated. Feed conversion was expressed on a net basis (feed intake divided by actual gain of live birds) and on an adjusted basis in which body weight gain of birds that died or were culled was added to the sum of body weight gain for the pen. At 48 days of age, 8 males from 8 pens per treatment were randomly selected and killed by cervical dislocation to determine incidence and severity of TD. The right tibia was used to score for TD and the left tibia was saved to determine bone breaking strength. Fracture force was determined by the shear block method according to ASAE (1999) standards and the pieces were saved for bone ash determination. Cross sectional area of the shaft of the tibia was measured with calipers at the point of the break to calculate breaking strength. All other laboratory and statistical analyses for measurements in Experiment 2 were conducted the same as in Experiment 1.

Results and Discussion

There were no effects of dietary 25-(OH)D_3 on body weight or feed conversion (feed:gain) in Experiment 1 (Table 2). Body weight was also not affected by treatment in Experiment 2 (Table 3). Feed conversion was decreased at 42 days of age for birds fed 25-(OH)D_3 in the starter period only compared to the control treatment. However, feed conversion was not significantly different from the control group when

Table 2: Effect of 25-hydroxycholecalciferol [25-(OH)D_3] on growth performance, phosphorus utilization and bone parameters of broiler chickens (Experiment 1)

Parameter	Treatment				
	Control	25-(OH)D_3/Control	25-(OH)D_3	SEM	p<
Phase I (1-17 days)					
Body weight (g)	505	----	496	11	0.608
Feed:gain (g:g)	1.295	----	1.276	0.008	0.354
Tibial Dyschondroplasia (TD)					
Incidence (%)	12	----	12	3	0.997
Score Index No. 1[1]	1.38	----	1.19	0.36	0.704
Score Index No. 2[2]	0.22	----	0.24	0.08	0.880
Severe lesions (%)[3]	2	----	5	3	0.555
Bone ash (%)	44.2	----	43.7	0.2	0.187
Phytate phosphorus retention (%)	42.3	----	40.3	1.5	0.371
Phase II (18-35 days)					
Body weight (g)	1857	1809	1847	30	0.526
Feed:gain (g:g)	1.603	1.610	1.585	0.011	0.464
Tibial Dyschondroplasia (TD)					
Incidence (%)	0	0	7	1	0.405
Score Index No. 1[1]	0.00	0.00	0.75	0.43	0.405
Score Index No. 2[2]	0.00	0.00	0.22	0.06	0.405
Severe lesions (%)[3]	0	0	7	1	0.405
Bone ash (%)	43.4	43.6	43.4	0.3	0.846
Phytate phosphorus Retention (%)	43.0[b]	42.7[b]	47.1[a]	0.8	0.004

[a-b]Means with no common superscript are different at $p < 0.05$.

[1]Total score from birds having TD lesions scored 1 to 3 per number of birds with TD in the pen (includes 0 score for pens in which there was no TD). [2]Total score from birds having TD lesions scored 0 to 3 per number of birds in the pen.

[3]Percentage of birds that scored number 3 (severe lesion) at the end of the phase.

25-(OH)D_3 was fed the entire 42 days. There were no feed conversion effects at 16 or 49 days of age demonstrating that dietary 25-(OH)D_3 generally did not affect feed conversion in the experiment. The lack of a body weight or feed efficiency response to dietary 25-(OH)D_3 in young broilers is typical when adequate cholecalciferol has been provided in the diet (Edwards, 1989, 1990 ; Roberson, 1999; Ledwaba and Roberson, 2003). Growth responses to dietary 25-(OH)D_3 have been reported when 25-(OH)D_3 is substituted for cholecalciferol in broiler diets (McNutt and Haussler, 1973; Cantor and Bacon, 1978; Yarger *et al.*, 1995b).

Incidence of TD was low in both experiments and was not affected by dietary 25-(OH)D_3. The incidence of TD found in Experiment 1 at 17 days of age was 12% (Table 2). Analysis of dietary calcium showed that it was higher than the calculated level of 0.85% (Table 1). Ledwaba and Roberson (2003) previously reported a 13% incidence of TD in Ross cockerels at 20 days of age when 0.90% calcium was fed. Other studies have demonstrated that the incidence of TD in Ross broilers is low (<25%) when dietary calcium is greater than 0.85% in a starter diet (Roberson, 1999; Scheideler and Ferket, 2000). There were no control birds with TD at 35 days of age in Experiment 1 and there was an 8% incidence of TD was observed in Experiment 2 in the 48-day-old control birds (Table 3). The incidence of TD in broilers is expected to decrease as the bird's age (Roberson and Edwards, 1996). There was also no evidence of a difference in bone ash in Experiment 1 (Table 2) or bone strength in Experiment 2 (Table 3), or when 25-(OH)D_3 was supplemented to the diet.

There was no effect of dietary 25-(OH)D_3 on phytate phosphorus retention at 17 days of age in Experiment 1 (Table 2). However, phytate phosphorus retention was increased at 35 days of age when 25-(OH)D_3 was fed the entire experiment. Dietary addition of 25-(OH)D_3 to broiler diets have previously been shown to improve phytate phosphorus utilization (Angel *et al.*, 2001; Edwards, 2002; Ledwaba and Roberson, 2003), but the response is inconsistent (Edwards, 2002; Ledwaba and Roberson, 2003).

The results of this study showed that dietary 25-(OH)D_3 did not affect growth or bone parameters in broilers fed to market age with marginal dietary calcium. However, there was some evidence that phosphorus utilization could be improved by adding 25-(OH)D_3 to broiler diets adequate in cholecalciferol and marginal in calcium. Further studies should be conducted to investigate the

Table 3: Effect of 25-hydroxycholecalciferol [25-(OH)D_3] on growth performance and bone parameters of broiler chickens (Experiment 2)

Parameter	Treatment				
	Control	25-(OH)D_3/Control	25-(OH)D_3	SEM	p<
16 days					
Body weight (g)	585	----	581	4	0.512
Feed:gain (g:g)	1.266	----	1.287	0.007	0.195
Feed:gain-adj.1 (g:g)	1.255	----	1.269	0.005	0.059
42 days					
Body weight (g)	2730	2734	2778	28	0.454
Feed:gain (g:g)	1.733[a]	1.703[b]	1.706[ab]	0.009	0.044
Feed:gain-adj.1(g:g)	1.689	1.679	1.677	0.011	0.667
48 days					
Tibial Dyschondroplasia (TD) Incidence (%)	8	2	6	3	0.365
Score Index No. 1[2]	0.94	0.25	1.12	0.44	0.353
Score Index No. 2[3]	0.20	0.03	0.19	0.09	0.331
Severe lesions (%)[4]	6	0	6	3	0.198
Fracture force (N)[5]	1063	1061	1078	27	0.883
Breaking strength (Mpa)[6]	11.86	12.35	11.62	0.63	0.368
49 days					
Body weight (g)	3155	3162	3216	30	0.344
Feed:gain (g:g)	1.914	1.930	1.883	0.021	0.391
Feed:gain-adj.(g:g)	1.829	1.833	1.825	0.010	0.858

[a-b]Means with no common superscript are different at $p < 0.05$. [1]Feed:gain-adj. = feed conversion adjusted for mortality.
[2]Total score from birds having TD lesions scored 1 to 3 per number of birds with TD in the pen (includes 0 score for pens in which there was no TD). [3]Total score from birds having TD lesions scored 0 to 3 per number of birds in the pen.
[4]Percentage of birds that scored number 3 (severe lesion) at the end of the phase
[5]N = Newtons; N = 0.102 kilograms of force = 9.8 kilograms of mass. [6]MPa = megapascals = N/m^2 (fracture force/cross sectional area).

factors involved in the phytate phosphorus response to dietary 25-(OH)D_3.

References

American Society of Agricultural Engineers (ASAE), 1999. Shear and three-point bending test of animal bone. Pages 584-586 in: ASAE Standards.

Angel, R., A.S. Dhandu, T.J. Applegate and M. Christman, 2001. Phosphorus sparing effect of phytase, 25-hydroxycholecalciferol, and citric acid when fed to broiler chicks. Poult. Sci., 80 (Suppl.1):134. (Abstr.).

Applegate, T.J., R. Angel, H.L. Classen, R.W. Newkirk and D.D. Maenz, 2000. Effect of dietary calcium concentration and 25-hydroxycholecalciferol on phytate hydrolysis and intestinal phytase activity in broilers. Poult. Sci., 79(Suppl.1): 21 (Abstr.).

Association of Official Analytical Chemists (AOAC), 1995. Pages 57-58 in Official Methods of Analysis of the Association of Official Analytical Chemists, 16th rev. ed. Vol. 2., A.O.A.C., Washington, DC.

Burton, R., A. Sheridan and C. Howlett, 1981. The incidence and importance of tibial dyschondroplasia to the commercial broiler industry in Austrailia. Br. Poult. Sci., 22: 153-160.

Cantor, A. and W. Bacon, 1978. Performance of caged broilers fed vitamin D_3 and 25-(OH) vitamin D_3. Poult. Sci., 57: 1123-1124.

Edwards, H.M., Jr., 1984. Studies on the etiology of tibial dyschondroplasia. J. Nutr., 114: 1001-1013.

Edwards, H.M., Jr., 1989. The effect of dietary cholecalciferol, 25-hydroxycholecalciferol and 1, 25-dihydroxycholecalciferol on the development of tibial dyschondroplasia in broiler chickens in the absence and presence and disulfiram. J. Nutr., 119: 647-652.

Edwards, H.M., Jr., 1990. Efficacy of several vitamin D compounds in the prevention of tibial dyschondroplasia in broiler chickens. J. Nutr., 120: 1054-1061.

Edwards, H.M., Jr., 2002. Studies on the efficacy of cholecalciferol and derivatives for stimulating phytate utilization in broilers. Poult. Sci., 81: 1026-1031.

Edwards, H.M., Jr. and J.R. Veltmann, Jr., 1983. The role of calcium and phosphorus in the etiology of tibial dyschondroplasia in young chicks. J. Nutr., 113: 1568-1575.

Edwards, H.M., Jr. and M.B. Gillis, 1959. A chromic oxide balance method for determining phosphate availability. Poult. Sci., 38: 569-574.

Gomori, G., 1942. A modification of the colorimetric phosphorus determination for the use with the photoelectric colorimeter. J. Clin. Lab. Med., 27: 955-960.

Latta, M. and M. Eskin, 1980. Phytate phosphorus determination. J. Agri. Food Chem., 28: 1313-1315.

Leach, R.M., Jr. and M.S. Lilburn, 1992. Current knowledge on the etiology of tibial dyschondroplasia in the avian species. Poult. Sci. Rev., 4: 57-65.

Leach, R.M., Jr. and M.C. Nesheim, 1965. Nutritional, genetic and morphological studies of an abnormal cartilage formation in young chicks. J. Nutr., 86: 236-244.

Ledwaba, M.F. and K.D. Roberson, 2003. Effectiveness of twenty-five-hydroxycholecalciferol in the prevention of tibial dyschondroplasia in Ross cockerels depends on dietary calcium level. Poult. Sci., 82: 1769-1777.

McNutt, K. and M. Haussler, 1973. Nutritional effectiveness of 1,25-dihydroxycholecalciferol in preventing rickets in chicks. J. Nutr., 103: 681-689.

Mireles, A.M., Jr., S. Kim, B. Krautmann, J. Yarger and L. Stark, 1996. Effect of 25 hydroxycholecalciferol (25-OH-D_3) on broiler field performance and incidence of tibial dyschondroplasia (TD): Minimum D_3 metabolite consumption period. Poult. Sci., 75(Suppl.1): 280. (Abstr.).

Mitchell, R.D., H.M. Edwards, Jr. and G.R. McDaniel, 1997. The effects of ultraviolet light and cholecalciferol and its metabolites on the development of leg abnormalities in chickens genetically selected for a high and low incidence of tibial dyschondroplasia. Poult. Sci., 76: 346-354.

National Research Council, 1994. Nutrient Requirements of Poultry. 9th rev. ed. National Academy Press, Washington, DC.

Rennie, J.S. and C.C. Whitehead, 1996. Effectiveness of dietary 25- and 1-hydroxycholecalciferol in combating tibial dyschondroplasia in broiler chickens. Br. Poult. Sci., 37: 413-421.

Rennie, J.S., C.C. Whitehead and B.H. Thorp, 1993. 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: 809-816.

Roberson, K.D., 1999. 25-hydroxycholecalciferol fails to prevent tibial dyschondroplasia in broiler chicks raised in battery brooders. J. Appl. Poult. Res., 8: 54-61.

Roberson, K.D. and H.M. Edwards, Jr., 1996. Effect of dietary 1,25-dihydroxycholecalciferol level on broiler performance. Poult. Sci., 75: 90-94.

SAS Institute, 2003. Release 8.2 Edition. SAS Institute Inc., Cary, NC.

Scheideler, S.E. and P.R. Ferket, 2000. Phytase in broiler rations. Effects on carcass yields and incidence of tibial dyschondroplasia. J. Appl. Poult. Res., 9: 468-475.

Shafey, T.M., M.W.M. McDonald and R.A. Pym, 1990. Effects of dietary calcium, available phosphorus and vitamin D on growth rate, food utilization, plasma and bone constituents and calcium and phosphorus retention of commercial broiler strains. Br. Poult. Sci., 31: 587-602.

Williams, C., D. David and O. Iisma, 1962. The determination of chromic oxide in faeces samples by atomic absorption spectrophotometry. J. Agri. Sci., 59: 381-385.

Xu, T., R.M. Leach, Jr., B. Hollis and J.H. Soares, 1997. Evidence of increased cholecalciferol requirement in chicks with tibial dyschondroplasia. Poult. Sci., 76: 47-53.

Yarger, J.G., C.L. Quarles, B.W. Hollis and R.W. Gray, 1995a. Safety of 25-hydroxycholecalciferol as a source of cholecalciferol in poultry rations. Poult. Sci., 74: 1437-1446.

Yarger, J.G., C.A. Saunders, J.L. McNaughton, C.L. Quarles, B.W. Hollis and R.W. Gray, 1995b. Comparison of dietary 25-hydroxycholecalciferol and cholecalciferol in broiler chickens. Poult. Sci., 74: 1159-1167.

Zhang, X., G. Liu, G.R. McDaniel and D. Roland, 1997. Responses of broiler lines selected for tibial dyschondroplasia incidence to supplementary 25-hydroxycholecalciferol. J. Appl. Poult. Res., 6: 410-416.

---

[1]To whom correspondence should be addressed: robers22@msu.edu

[2]Townline Poultry Farm, Zeeland, MI 49464, USA

[3]Petersime Incubator Co., Gettyburg, OH 45328, USA

[4]OSRAM Sylvania, Danvers, MA 01923, USA

[5]University Products, Inc., Holyoke, MA 01041, USA

[6]Monsanto Animal Nutrition, Naperville, IL 60563, USA

[7]Wiley Mill, Arthur H. Thomas Co., Philadelphia, PA 19019, USA

[8]Cyclotech Model 1093 Sample Mill, Foss Tecator, AB Hoganas, Sweden

[9]Microwave Accelerated Reaction System 5 Model 907005, CEM Corp., Matthews, NC 28106, USA

[10]UNICAM 989, Thermo Electron Corporation, Franklin, MA 02038, USA

[11]DU 7400, Beckman Instruments, Inc., Fullerton, CA 92634, USA

[12]Pine Manor, Inc., Goshen, IN 46526, USA

[13]Hamilton Farm Bureau, Hamilton, MI 49419, USA