Effect of 1,25-Dihydroxycholecalciferol, Cholecalciferol, and Fluorescent Lights on the Development of Tibial Dyschondroplasia and Rickets in Broiler Chickens[1]

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ABSTRACT Experiments were conducted to determine whether dietary 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] will alleviate a cholecalciferol deficiency induced by low dietary cholecalciferol and no fluorescent lighting and to determine cholecalciferol requirements as influenced by fluorescent lighting or 1,25-$(OH)_2D_3$. In each study, nutritionally complete basal diets were fed to broiler cockerels from 1 to 16 d of age. Experiment 1 had a 2 × 2 × 2 factorial arrangement of treatments with 1,25-$(OH)_2D_3$ at 0 and 10 μg/kg, cholecalciferol at 2.75 and 27.5 μg/kg, and fluorescent lights on or off. Experiments 2 to 4 had four levels of dietary cholecalciferol (0, 5.0, 27.5, and 50.0 μg/kg) and fluorescent lights on or off (Experiment 2) or 1,25-$(OH)_2D_3$ at 0 and 10 μg/kg (Experiments 3 and 4). In Experiment 1, fluorescent lighting increased bone ash, and decreased the incidence and severity of rickets at 2.75 μg/kg cholecalciferol and 0 μg/kg 1,25-$(OH)_2D_3$ and reduced the severity of TD at both levels of cholecalciferol and 0 μg/kg 1,25-$(OH)_2D_3$. In all cases 1,25-$(OH)_2D_3$ improved bone ash. The metabolite also decreased the incidence and severity of TD at both cholecalciferol levels with lights off and decreased the incidence and severity of rickets at 2.75 μg/kg cholecalciferol and lights off.

In the absence of fluorescent lighting and 1,25-$(OH)_2D_3$ 27.5 μg/kg cholecalciferol reduced the incidence and severity of rickets to levels equivalent to those produced by either fluorescent lighting or 1,25-$(OH)_2D_3$ alone (Experiments 2, 3, and 4). However, even 50.0 μg/kg cholecalciferol was not as effective as fluorescent lights or 1,25-$(OH)_2D_3$ in reducing the incidence and severity of TD.

(*Key words*: 1,25-dihydroxycholecalciferol, fluorescent lighting, tibial dyschondroplasia, rickets, cholecalciferol)

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INTRODUCTION

Edwards (1989a, 1990) has reported that dietary supplementation of a low Ca diet containing 27.5 μg/kg cholecalciferol with 10 μg/kg 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] significantly decreased the incidence and severity of tibial dyschondroplasia (TD) and increased tibia bone ash. These studies were the first to suggest that TD may be characteristic of a cholecalciferol deficiency in the broiler chicken. Edwards made no attempt to limit maternal cholecalciferol reserves of the chicks or the amount of ultraviolet light received by the chickens from the battery brooder fluorescent lights or sunlight. Presumably, the birds were able to meet much of their requirement for cholecalciferol via ultraviolet irradiation of cholecalciferol precursors in the skin.

Under the experimental conditions utilized by Edwards (1989a, 1990), very low incidences of TD occur in birds fed diets adequate in Ca and cholecalciferol. However, in modern broiler production, in order to achieve maximum feed efficiency and weight gain, growers are raising the birds in closed poultry houses under extremely low incandescent light intensity. The birds frequently move only to eat and drink. This management technique makes the diet the sole source of the nutrient.

When exposure of birds to fluorescent lighting or sunlight was prevented, dietary supplementation of a basal diet containing no cholecalciferol and 0.65% Ca with 27.5 μg/kg cholecalciferol [5.5 times the National Research Council, (1994) recommendation], only reduced the incidence of TD from 92 to 73% and only improved tibia bone ash from 27 to 34% (Edwards, 1989b). The values obtained with 27.5 μg/kg cholecalciferol indicate that the birds were still in a cholecalciferol-deficient state. In two experiments in which the birds were fed 0.65% Ca and 27.5 μg/kg cholecalciferol and no attempt was made to limit the exposure to ultraviolet light from sunlight or fluorescent

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lighting, TD levels of 46 and 56% were observed (Elliot and Edwards, 1992). In the same report, three other experiments were conducted in which a severe cholecalciferol deficiency was desired, so the birds were prevented from exposure to ultraviolet light. In the treatments corresponding to the treatments in the two experiments in which the birds were exposed to fluorescent lights (low Ca and 27.5 μg/kg cholecalciferol), the incidences of TD observed were 92, 91, and 95% respectively. Thus, the absence of fluorescent lighting caused the incidence of TD to increase by 80, 78, and 86%, respectively (Elliot and Edwards, 1992). When the Ca level was increased to an adequate level (0.95%) and fed with 27.5 μg/kg cholecalciferol, the average incidence of TD observed in these three experiments was 73, 78, and 78% (Elliot and Edwards, 1992). The only difference between these studies and those in which a TD incidence of 5 to 15% is expected (Edwards and Veltmann, 1983) is exposure to fluorescent lighting. These studies concur with Edwards (1989a, 1990), who hypothesized that the young, rapidly growing broiler chicken is not able to produce 1,25-$(OH)_2D_3$ from dietary cholecalciferol rapidly and efficiently enough to meet its needs for maximum Ca absorption and bone development and that this inability makes the broiler chicken more susceptible to TD.

The present studies were conducted to determine whether dietary supplementation with 1,25-$(OH)_2D_3$ will alleviate a cholecalciferol deficiency induced by low dietary cholecalciferol and a lack of fluorescent lighting. Studies were conducted to determine the level of cholecalciferol required for optimal performance in both the presence and absence of fluorescent lighting and to determine the level of cholecalciferol required for optimal performance in both the presence and absence of 1,25-$(OH)_2D_3$ when the fluorescent lights are either off or equipped with Arm-a-lite [3] ultraviolet filter tubes. The effect of dietary cholecalciferol and dietary 1,25-$(OH)_2D_3$ on plasma 1,25-$(OH)_2D_3$ was measured in one experiment.

MATERIALS AND METHODS

General Procedures

Four experiments were conducted using 240 day-old Peterson × Arbor Acres broiler cockerels. The birds were randomly wing-banded and placed in electrically heated Petersime[4] wire-floored battery brooders at 10 birds per cage. The chicks were raised on a continuous illumination schedule and feed and water were provided for *ad libitum* consumption throughout the 16-d experimental period.

Each experiment was conducted with a corn and soybean meal practical diet (Table 1). All dietary modifications were made at the expense of corn. The basal diet contained by calculation 0.90% Ca, 0.72% total P, 0.46% nonphytate P, and 0.34% Cl.

At the end of the experimental period, the birds were weighed by pen and feed intake recorded for feed efficiency computation. One bird was randomly selected from each pen and a blood sample removed by cardiac puncture for subsequent determination of plasma Ca[5] and plasma dialyzable P[6] in Experiments 3 and 4. All birds were then killed by asphyxiation and randomly inspected for the presence and severity of TD (Edwards and Veltmann, 1983) and rickets (Long *et al.*, 1984). The left tibia was removed for bone ash determination on a dry fat-free basis (Association of Official Agricultural Chemists, 1955).

Experiment 1

This experiment was conducted to determine whether dietary supplementation with 1,25-$(OH)_2D_3$ will alleviate a cholecalciferol deficiency induced by low dietary cholecalciferol and a lack of fluorescent lighting. This experiment had a 2 × 2 × 2 factorial arrangement of treatments with fluorescent lights on and off, cholecalciferol at 2.75 and 27.5 μg/kg, and 1,25-$(OH)_2D_3$ at 0 and 10 μg/kg. Three pens of 10 broiler cockerels were randomly assigned to each of the eight dietary treatments. To prevent exposure to ultraviolet light, the battery brooder fluorescent lights were turned off (in those treatments which required no exposure to ultraviolet light) and the room fluorescent lights and windows were covered with clear plastic.

TABLE 1. Composition of the basal diet

Ingredients and analysis	Percentage
Yellow corn	56.30
Soybean meal (dehulled)	35.00
Poultry fat (stabilized)	5.00
Iodized sodium chloride	0.42
Dicalcium phosphate (feed grade)	1.86
Limestone	1.01
Vitamin premix[1]	0.25
DL-methionine	0.20
Trace mineral premix[2]	0.10
Se concentrate (0.02% from sodium selenite)	0.05
Calculated analysis	
Protein	22.10
Calcium	0.90
Total phosphorus	0.72
Nonphytate phosphorus	0.46
Chlorine	0.34
Metabolizable energy, kcal/kg	3,227

[1]Supplied per kilogram of diet: vitamin A (as all-*trans*-retinyl acetate), 1,892; vitamin E (all-*rac*-α-tocopheryl acetate), 11; menadione (as menadione sodium bisulfite), 1.1; cholecalciferol, 27.5 μg; riboflavin, 4.4; Ca pantothenate, 12; nicotinic acid, 44; choline chloride, 220; vitamin B_{12}, 9 μg; vitamin B_6, 3.0; thiamin (as thiamin mononitrate), 2.2; folic acid, 3; biotin, 0.3; and ethoxyquin, 125.

[2]Supplied per kilogram of diet: manganese, 120; zinc, 100; iron, 60; copper, 10; iodine, 2.1; Ca, 150 (min), 180 (max).

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Experiment 1 and 2 of this paper were conducted before we had available the Arm-a-lite ultraviolet filter tubes, which were proven to be excellent filters of ultraviolet light (Edwards *et al*., 1994). However, the results obtained in Experiment 1 and 2 of this paper compared to Experiments 3 and 4 of this paper would indicate that the 10-mil polyethylene plastic used to cover the fluorescent lights in Experiment 1 and 2 was very effective in screening out the ultraviolet light.

Experiment 2

This experiment was designed to determine what level of cholecalciferol is required for optimal performance in both the presence and absence of fluorescent lighting. The room lighting in this experiment was identical to that used in Experiment 1. The basal diet contained 0 μg/kg cholecalciferol. The experiment had a 2 × 4 factorial arrangement of treatments with cholecalciferol at 0, 5.0, 27.5, and 50.0 μg/kg, and fluorescent lights on and off. Three pens of 10 broiler cockerels were randomly assigned to each of the eight dietary treatments.

Experiments 3 and 4

These experiments were conducted to determine the level of cholecalciferol required for optimal performance in both the presence and absence of 1,25-$(OH)_2D_3$. It was necessary in these two experiments to limit the exposure of the birds to ultraviolet light from either sunlight or fluorescent lights. In Experiment 3, this limitation of exposure was accomplished by covering the room fluorescent lights and windows with clear plastic and turning the battery brooder fluorescent lights off. In Experiment 4, the battery brooder and room fluorescent lights were equipped with Arm-a-lite ultraviolet filter tubes, which exclude light in the ultraviolet range (260 to 400 nm). Research in this laboratory has shown these ultraviolet filter tubes to be effective in excluding light in the ultraviolet range (Edwards *et al*., 1994). Also in Experiment 4 plasma 1,25-$(OH)_2D_3$ was measured to determine the effect of dietary cholecalciferol and dietary 1,25-$(OH)_2D_3$ on plasma 1,25-$(OH)_2D_3$. Plasma 1,25-$(OH)_2D_3$ was measured by a calf thymus radioreceptor assay developed by Reinhardt and Hollis (1986). In this procedure, an ammonium sulfate precipitate of calf thymus 1,25-$(OH)_2D_3$ cytosol receptor is used. Plasma samples were extracted by two sequential SEP-PAK[7] chromatography steps (Reinhardt and Hollis, 1986). This extraction eliminates the time-consuming HPLC purification steps. As with Experiment 2, the basal diet contained 0 μg/kg cholecalciferol. Experiments 3 and 4 had a 2 × 4 factorial arrangement of treatments with cholecalciferol at 0, 5.0, 27.5, and 50.0 μg/kg, and 1,25-$(OH)_2D_3$ at 0 and 10 μg/kg. Three pens of 10 broiler cockerels were randomly assigned to each of the eight dietary treatments.

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Statistical Analysis

The data were analyzed within experiments using the General Linear Models procedure for analysis of variance and regression analysis. When appropriate, mean differences were separated by Duncan's new multiple range test. Unless otherwise stated, statements of statistical significance are based on a probability of ($P \leq 0.05$) (Steel and Torrie, 1980).

RESULTS

Experiment 1

There was no treatment effect on weight gain or feed efficiency. Dietary supplementation with 27.5 μg/kg cholecalciferol only improved tibia bone ash in the absence of fluorescent lighting and 1,25-$(OH)_2D_3$ (Table 2). In the absence of 1,25-$(OH)_2D_3$, fluorescent lighting improved bone ash at 2.75 μg/kg cholecalciferol, but had no effect at 27.5 μg/kg cholecalciferol. In all cases 1,25-$(OH)_2D_3$ significantly improved tibia bone ash (Table 2). Dietary supplementation with 27.5 μg/kg cholecalciferol failed to decrease the incidence of TD and the average lesion score and only decreased the percentage number 3 scores in the absence of 1,25-$(OH)_2D_3$ and fluorescent lighting (Table 2). Fluorescent lights decreased the incidence of TD and the percentage number 3 scores at both levels of cholecalciferol and with no 1,25-$(OH)_2D_3$. With the fluorescent lights off, 1,25-$(OH)_2D_3$ decreased the incidence of TD and percentage number 3 scores at both levels of cholecalciferol (Table 2). In this experiment, fluorescent lighting was nearly as effective as 1,25-$(OH)_2D_3$ in reducing the incidence and severity of TD. The response to fluorescent lighting and 1,25-$(OH)_2D_3$ was greater than that achieved by 5.5 times the National Research Council (1994) level of cholecalciferol recommended as a minimum requirement for growing chickens. Fluorescent lighting, 27.5 μg/kg cholecalciferol, and 10 μg/kg 1,25-$(OH)_2D_3$ were all equally effective in reducing the incidence and severity of rickets (Table 2).

Experiment 2

In the presence of fluorescent lighting, increasing levels of cholecalciferol had no effect on weight gain, TD score, percentage number 3 TD scores, incidence of rickets, and percentage number 3 rickets scores (Table 3). Percentage bone ash was significantly increased by 5.0 and 27.5μg/kg cholecalciferol in the presence of fluorescent lighting. Feed efficiency, the incidence of TD, and the average rickets lesion score were decreased by 50.0 μg/kg cholecalciferol

TABLE 2. Effect of dietary cholecalciferol (D_3), 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$], and fluorescent lighting on growth, feed efficiency, and the incidence and severity of tibial dyschondroplasia and rickets in broiler cockerels, Experiment 1

Treatments			16-d	Gain:feed	Bone	Tibial dyschondroplasia			Rickets		
D_3	1,25-$(OH)_2D_3$	Lights	BW[1]	ratio[1]	ash[1]	Incidence[1,2]	Score[1]	No. 3[1,2]	Incidence[1,2]	Score[1]	No. 3[1,3]
(μg/kg)			(g)	(g:g)		(%)			(%)		(%)
2.75	0	Off	379	0.704	29.6^{de}	63^{a}	3.00^{a}	63^{a}	84^{a}	2.95^{a}	79^{b}
2.75	0	On	433	0.709	37.5^{c}	31^{ab}	1.50^{ab}	12^{c}	20^{bc}	1.67^{ab}	0^{b}
2.75	10	Off	382	0.678	38.8^{abc}	18^{b}	2.33^{ab}	7^{c}	11^{bc}	0.67^{bc}	0^{b}
2.75	10	On	422	0.705	39.7^{a}	26^{b}	1.92^{ab}	11^{c}	0^{c}	0^{c}	0^{b}
27.50	0	Off	433	0.721	37.8^{c}	63^{a}	2.39^{ab}	37^{b}	11^{bc}	1.33^{bc}	0^{b}
27.50	0	On	441	0.716	38.0^{bc}	38^{ab}	2.29^{ab}	15^{c}	24^{b}	1.50^{b}	4^{b}
27.50	10	Off	429	0.713	39.3^{ab}	22^{b}	1.33^{b}	0^{c}	4^{bc}	0.33^{b}	0^{b}
27.50	10	On	406	0.689	39.5^{a}	12^{b}	2.00^{ab}	4^{c}	4^{bc}	1.00^{bc}	4^{b}
$\bar{x}$ ± SEM			415 ± 6	0.704 ± 0.014	37.5 ± 5	34 ± 11	2.09 ± 0.46	18 ± 5	19 ± 7	1.18 ± 0.44	11 ± 3
ANOVA			Probability								
Source of variation		df									
D_3		1	0.937	0.291	<0.001	0.924	0.573	0.028	0.001	0.379	<0.001
1,25-$(OH)_2D_3$		1	0.605	0.118	<0.001	0.002	0.236	<0.001	<0.001	<0.001	<0.001
D_3 × 1,25-$(OH)_2D_3$		1	0.203	0.867	<0.001	0.575	0.411	0.610	0.003	0.065	<0.001
Lights		1	0.836	0.946	<0.001	0.077	0.311	0.001	0.005	0.379	<0.001
D_3 × Lights		1	0.931	0.132	<0.001	0.710	0.072	0.075	<0.001	0.039	<0.001
1,25-$(OH)_2D_3$ × Lights		1	0.161	0.920	<0.001	0.093	0.171	<0.001	0.044	0.379	<0.001
D_3 × 1,25-$(OH)_2D_3$ × Lights		1	0.410	0.314	<0.001	0.454	0.809	0.075	0.003	0.924	<0.001
			Main effect means								
D_3											
2.75 μg/kg			429	0.699	36.4^{b}	35	2.2	23^{a}	29^{a}	1.3	20^{a}
27.5 μg/kg			427	0.710	38.6^{a}	34	2.0	14^{b}	11^{b}	1.0	2^{b}
1,25-$(OH)_2D_3$											
0 μg/kg			422	0.712	35.7^{b}	49^{a}	2.3	32^{a}	35^{a}	1.9^{a}	21^{a}
10 μg/kg			435	0.696	39.3^{a}	20^{b}	1.9	6^{b}	5^{b}	0.5^{b}	1^{b}
Lights											
On			431	0.704	36.4^{b}	42	2.3	27^{a}	27^{a}	1.3	20^{a}
Off			426	0.705	38.7^{a}	27	1.9	11^{b}	12^{b}	1.0	2^{b}

$^{a-e}$Values of the same variable with no common superscript differ significantly ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Percentage of birds scored number 3 (large mass of cartilage in the proximal end of the tibiotarsus).

[3]Percentage of birds scored number 3 (very wide growth plate proliferating zone with no calcification).

TABLE 3. Effect of dietary cholecalciferol supplementation and light on growth, feed efficiency, tibia bone ash, and on the incidence and severity of tibial dyschondroplasia and rickets in broiler males, Experiment 2

Treatments		16-d	Gain:feed	Bone	Tibial dyschondroplasia			Rickets		
Cholecalciferol	Lights	BW[1]	ratio[1]	ash[1]	Incidence[1]	Score[1]	No. 3[1,2]	Incidence[1]	Score[1]	No. 3[1,3]
(μg/kg)		(g)	(g:g)	(%)				(%)		(%)
0	Off	306[c]	0.681[c]	25.5[e]	48[b]	2.92[a]	45[bc]	97[a]	2.84[a]	81[a]
5	Off	354[b]	0.701[abc]	29.1[d]	72[a]	2.96[a]	69[a]	86[a]	2.42[ab]	56[b]
27.5	Off	415[a]	0.728[ab]	37.0[b]	70[a]	2.58[ab]	50[b]	20[b]	1.00[b]	7[c]
50	Off	387[ab]	0.709[abc]	38.1[abc]	50[ab]	2.48[abc]	30[c]	7[bc]	1.33[abc]	7[c]
0	On	405[a]	0.737[a]	37.4[bc]	37[b]	1.72[bcd]	10[d]	10[bc]	1.33[abc]	3[c]
5	On	399[a]	0.721[abc]	39.0[a]	38[a]	1.61[bcd]	4[d]	7[bc]	0.33[c]	0[c]
27.5	On	392[a]	0.706[abc]	39.1[a]	23[bc]	1.56[cd]	3[d]	3[bc]	0.33[c]	0[c]
50	On	407[a]	0.696[bc]	38.6[ab]	7[c]	1.33[d]	0[d]	0[c]	0.00[d]	0[c]
$\bar{x}$ ± SEM		383 ± 11	0.710 ± 0.012	35.5 ± 5	43 ± 10	2.15 ± 0.31	26 ± 6	29 ± 6	1.20 ± 0.48	19 ± 6
ANOVA		Probability								
Source of variation	df									
Cholecalciferol	3	0.002	0.687	<0.001	<0.076	0.521	0.015	<0.001	0.027	<0.001
Lights	1	<0.001	0.230	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001
Lights × cholecalciferol	3	0.001	0.022	<0.001	0.267	0.961	0.031	<0.001	0.546	<0.001
Regression	df									
Lights off										
Linear	1	<0.001	0.028	<0.001	0.155	0.124	0.456	<0.001	0.060	0.001
Quadratic	1	0.002	0.052	<0.001	0.123	0.485	0.199	0.008	0.146	0.007
Lights on										
Linear	1	0.286	0.252	0.035	0.676	0.938	0.507	0.561	0.344	0.337
Quadratic	1	0.256	0.559	0.055	0.751	0.930	0.768	0.836	0.577	0.543
		Main effect means								
Lights off		365[b]	0.705	32.4[b]	60[a]	2.7[a]	48[a]	52[a]	1.9[a]	38[a]
Lights on		401[a]	0.715	38.5[a]	26[b]	1.6[b]	4[b]	5[b]	0.5[b]	1[b]
Cholecalciferol										
0 μg/kg		356[c]	0.709	31.5[c]	42[ab]	2.3	27[ab]	53[a]	2.1[a]	42[a]
5 μg/kg		376[bc]	0.711	34.0[b]	55[a]	2.3	36[a]	47[a]	1.4[ab]	28[b]
27.5 μg/kg		403[a]	0.717	38.1[a]	47[ab]	2.1	27[ab]	12[b]	0.7[b]	3[c]
50 μg/kg		397[ab]	0.702	38.4[a]	28[b]	1.9	15[b]	3[b]	0.7[b]	3[c]

[a-e]Values of the same variable with no common superscript differ significantly ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Percentage of birds scored number 3 (large mass of cartilage in the proximal end of the tibiotarsus).

[3]Percentage of birds scored number 3 (very wide growth plate proliferating zone with no calcification).

in the presence of fluorescent lighting (Table 3). In the absence of fluorescent lighting, supplementation with 27.5 μg/kg cholecalciferol resulted in increased weight gain equivalent to that produced by fluorescent lighting and 0 μg/kg cholecalciferol. In the absence of fluorescent lighting, 27.5 μg/kg cholecalciferol resulted in increased feed efficiency and 50.0 μg/kg stimulated tibia bone ash values equivalent to those produced by fluorescent lighting and 0 μg/kg cholecalciferol. In the absence of fluorescent lighting, 27.5 μg/kg cholecalciferol reduced the incidence of rickets and percentage number 3 rickets scores to the same levels produced by fluorescent lighting and 0 μg/kg cholecalciferol. The average rickets lesion score was not reduced by 50.0 μg/kg cholecalciferol in the absence of fluorescent lighting. The incidence and severity of TD was not completely reduced by cholecalciferol in the absence of fluorescent lighting (Table 3). At 50.0 μg/kg and no fluorescent lights, the incidence of TD was 50%, the TD score 2.48, and the number 3 TD score was 30%. In the presence of fluorescent lighting, the corresponding values were 7, 1.33, and 0%. In the absence of fluorescent lighting, increasing levels of dietary cholecalciferol affected weight gain, feed efficiency, and tibia bone ash in both a linear and quadratic manner, with 5.0 and 27.5 μg/kg improving the parameters and 50.0 μg/kg adversely influencing body weight and feed efficiency (Table 3). The incidence and severity of rickets was decreased linearly with increasing levels of dietary cholecalciferol in the absence of fluorescent lighting (Table 3).

Experiments 3 and 4

An attempt was made to combine the data from Experiments 3 and 4, but due to numerous experiment treatment interactions it was not possible. In both experiments, a significant linear increase in 16-d weight, bone ash, and plasma Ca, and a linear decrease in the incidence and severity of rickets were obtained from increasing cholecalciferol levels in the absence of 1,25-$(OH)_2D_3$ supplementation. Increasing cholecalciferol levels in the absence of 1,25-$(OH)_2D_3$ also linearly decreased the incidence and severity of TD in Experiment 4 (Tables 4, 5, 6, and 7). Conversely, in both experiments, in the presence of dietary 1,25-$(OH)_2D_3$, there were no linear or quadratic effects of cholecalciferol levels on any of the parameters measured (Tables 4, 5, 6, and 7). In the presence of 1,25-$(OH)_2D_3$, in both experiments, increasing levels of cholecalciferol had no effect on tibia bone ash, plasma Ca, plasma dialyzable P, incidence and severity of TD, and rickets (Tables 4, 5, 6, and 7). In the absence of 1,25-$(OH)_2D_3$ 50.0 μg/kg cholecalciferol produced bone ash equivalent to that produced by dietary 1,25-$(OH)_2D_3$ and 0 μg/kg cholecalciferol (Tables 4 and 6). With the exception of the incidence of rickets in Experiment 3, which was not reduced to a level equivalent to that achieved by 1,25-$(OH)_2D_3$ alone until 50.0 μg/kg cholecalciferol was fed, plasma Ca and rickets incidence and severity were equivalent at 27.5 μg/kg cholecalciferol (Tables 4, 5, 6, and 7). Plasma dialyzable P was unaffected by dietary treatment in Experiment 4 (Table 6) and significantly depressed by the absence of both cholecalciferol and 1,25-$(OH)_2D_3$ in Experiment 3 (Table 4). The incidence and severity of TD was not completely reduced by cholecalciferol in the absence of 1,25-$(OH)_2D_3$ in Experiment 3 (Table 5). At 50.0 μg/kg without 1,25-$(OH)_2D_3$ the incidence of TD was 50%, the TD score 2.73, and the number 3 TD score was 40%. In the presence of 1,25-$(OH)_2D_3$ the corresponding values were 27, 1.69, and 3% (Table 5). Supplementary 1,25-$(OH)_2D_3$ decreased weight gain and feed efficiency in Experiment 4 (Table 6). In Experiment 4, increasing levels of dietary cholecalciferol increased numerically, but not significantly, plasma 1,25-$(OH)_2D_3$ in the presence and absence of dietary 1,25-$(OH)_2D_3$ (Table 6). When the diet contained no 1,25-$(OH)_2D_3$ increasing levels of cholecalciferol linearly increased plasma 1,25-$(OH)_2D_3$ levels (Table 7). However, there was a 1,25-$(OH)_2D_3$ effect on plasma 1,25-$(OH)_2D_3$ ($P \leq 0.045$) and no cholecalciferol by 1,25-$(OH)_2D_3$ effect, indicating that dietary 1,25-$(OH)_2D_3$ had the same effect across all dietary cholecalciferol levels (Table 6). The average plasma 1,25-$(OH)_2D_3$ level was 86 pg/mL at 0 supplementation and 115 pg/mL when the diet was supplemented with 10 μg/kg 1,25-$(OH)_2D_3$ (Table 6). The plasma 1,25-$(OH)_2D_3$ level was increased when the dietary cholecalciferol level was increased from 0 to 5.0 μg/kg when the diet contained 10μg/kg 1,25-$(OH)_2D_3$ (Table 6). At 0 and 5.0 μg/kg cholecalciferol and 0 μg/kg 1,25-$(OH)_2D_3$ the plasma 1,25-$(OH)_2D_3$ was very low 70 and 68 pg/mL vs 103 and 104 pg/mL when the diet contained 10μg/kg 1,25-$(OH)_2D_3$ (Table 6).

DISCUSSION

These results show that when young rapidly growing broilers are fed diets adequate in both Ca and P and are not exposed to fluorescent lighting or sunlight they will have a high incidence and severity of TD, which cannot be reduced by dietary supplementation of up to 10 times the National Research Council (1994) recommended level of cholecalciferol. This finding demonstrates that the cholecalciferol requirement of birds not exposed to ultraviolet light is considerably higher than the National Research Council (1994) requirement of 5.0 μg/kg. In experiments conducted to quantify the cholecalciferol requirement of broiler chicks in the absence of fluorescent lighting, Edwards *et al.* (1994) estimated that General Electric F15T8-CW fluorescent lights were providing the equivalent of 20.0 to 40.0 μg cholecalciferol/kg.

The development of TD appears to be more sensitive to a cholecalciferol deficiency than rickets. Long *et al.* (1984) reported that both rachitic and dyschondroplastic lesions were observed in 3- and 4-wk-old birds. The Ca-deficient bird will rapidly develop rickets. As birds become Ca-deficient they will also become more efficient at assimilating Ca from the intestinal tract, presumably

TABLE 4. Effect of cholecalciferol and 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] on growth, feed efficiency, bone ash, and plasma minerals in birds not exposed to ultraviolet light[2], Experiment 3

Treatments			16-d BW[1]	Gain:feed ratio[1]	Bone ash[1]	Plasma mineral	
Cholecalciferol	1,25-$(OH)_2D_3$					Ca[1]	P[1,3]
(μg/ kg)			(g)	(g:g)	(%)	(μg/ dL)	
0	0		367[ab]	0.776	28.5[e]	8.3[b]	3.7[b]
5	0		367[ab]	0.745	31.3[d]	8.1[b]	5.3[a]
27.5	0		409[a]	0.810	38.2[c]	11.0[a]	5.0[a]
50	0		407[ab]	0.774	38.5[bc]	11.5[a]	5.4[a]
0	10		369[ab]	0.780	39.7[ab]	11.8[a]	5.2[a]
5	10		350[b]	0.739	39.7[ab]	12.1[a]	5.6[a]
27.5	10		390[ab]	0.774	39.9[a]	12.8[a]	5.4[a]
50	10		364[ab]	0.748	39.5[ab]	12.9[a]	4.8[a]
$\bar{x}$ ± SEM			378 ± 17	0.768 ± 0.25	36.9 ± 4	11.0 ± 6	5.0 ± 3
ANOVA			Probability				
Source of variation		df					
Cholecalciferol		3	0.130	0.255	<0.001	0.002	0.062
1,25-$(OH)_2D_3$		1	0.128	0.387	<0.001	<0.001	0.131
1,25-$(OH)_2D_3$ × cholecalciferol		3	0.639	0.858	<0.001	0.113	0.055
Regression No 1,25-$(OH)_2D_3$		df					
Linear		1	0.033	0.537	<0.001	0.001	0.150
Quadratic Plus 1,25-$(OH)_2D_3$		1	0.311	0.316	<0.001	0.263	0.485
Linear		1	0.724	0.749	0.758	0.132	0.096
Quadratic		1	0.371	0.781	0.526	0.486	0.171
			Main effect means				
1,25-$(OH)_2D_3$							
0 μg/ kg			388	0.776	34.1[b]	9.7[b]	4.9
10 μg/ kg			368	0.760	39.7[a]	12.4[a]	5.2
Cholecalciferol							
0 μg/ kg			368[ab]	0.778	34.1[c]	10.0[b]	4.5[b]
50 μg/ kg			359[b]	0.742	35.5[b]	10.1[b]	5.4[a]
27.5 μg/ kg			400[a]	0.792	39.0[a]	11.9[a]	5.2[ab]
50 μg/ kg			386[ab]	0.761	39.1[a]	12.2[a]	5.1[ab]

[a–e]Values of the same variable with no common superscript differ significantly ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Exposure to ultraviolet light prevented by covering room fluorescent lights and windows with clear plastic and turning off battery fluorescent lights.

[3]Plasma dialyzable P.

due to increased production of parathyroid hormone leading to increased conversion of 25-hydroxycholecalciferol [25-$(OH)D_3$] to 1,25-$(OH)_2D_3$. This increased efficiency will then lead to increased plasma Ca levels, making more Ca available to the bone. In Ca-deficient rickets, the proliferating prehypertrophy zone is lengthened due to a delay in chondrocyte hypertrophy. Long *et al.* (1984) observed a lengthening of the hypertrophy zone in 3-and 4-wk-old birds, similar to the TD lesion. They hypothesized that this was due to increased numbers of chondrocytes from the lengthened proliferating prehypertrophy zone (rachitic lesion) suddenly entering the hypertrophied stage of development due to increased Ca absorption. Tibial dyschondroplasia in the Ca- or cholecalciferol-deficient bird may be an attempt by the bird to repair its impaired endochondral ossification. Riddell and Pass (1987) have also reported that rickets appears to precede the development of TD in 2-wk-old chickens. Leach and Burdette (1987) observed lesions containing both rickets and TD in birds fed diets containing normal levels of P and intermediate levels of Ca (0.45 and 0.55%). The higher incidences of TD observed in the present studies may be due to the rachitic lesions developing into TD as the bird tries to heal the rachitic lesion. Variability between experiments indicates that factors such as parent flock age and cholecalciferol body stores at hatch may affect the cholecalciferol requirement of the young broiler chicken.

Experiment 1 showed that when broilers are not exposed to fluorescent lighting, dietary supplementation with 1,25-$(OH)_2D_3$ will reduce the incidence and severity of both TD and rickets to levels equal to or below those obtained in birds exposed to fluorescent lighting and fed a basal diet containing adequate cholecalciferol and Ca. The present studies indicate that fluorescent lighting and dietary supplementation with 1,25-$(OH)_2D_3$ are equally effective in reducing the development of TD and rickets in broiler chicks. Edwards (1989a, 1990) has previously reported that dietary supplementation of a low Ca diet containing 27.5 μg/ kg cholecalciferol with 10 μg/ kg 1,25-$(OH)_2D_3$ significantly decreases the incidence and severity of TD and increases tibia bone ash. The present report is the first to show that supplementation of a diet adequate in

TABLE 5. Effect of dietary cholecalciferol and 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] on the incidence and severity of tibial dyschondroplasia (TD) and rickets in broiler cockerels not exposed to fluorescent lighting[2], Experiment 3

Treatments			Tibial dyschondroplasia			Rickets		
Cholecalciferol	1,25-$(OH)_2D_3$		Incidence[1]	Score[1]	No. 3[1,3]	Incidence[1]	Score[1]	No. 3[1,4]
(μg/kg)			(%)		(%)			(%)
0	0		30[c]	2.83[a]	20[b]	97[a]	2.82[a]	87[a]
5	0		67[ab]	2.82[a]	56[a]	82[a]	2.66[a]	70[b]
27.5	0		72[a]	2.57[a]	54[a]	4[b]	1.88[ab]	15[c]
50	0		50[b]	2.73[a]	40[a]	2[bc]	0.83[b]	3[c]
0	10		23[c]	1.33[b]	3[b]	1[c]	0.67[b]	0[c]
5	10		27[c]	1.67[b]	3[b]	7[c]	0.67[b]	0[c]
27.5	10		14[c]	1.33[b]	4[b]	4[c]	1.00[b]	4[c]
50	10		27[c]	1.69[b]	3[b]	10[c]	1.00[b]	0[c]
$\bar{x}$ ± SEM			39 ± 6	2.12 ± 0.22	23 ± 7	26 ± 7	1.44 ± 0.48	22 ± 5
ANOVA			Probability					
Source of variation		df						
Cholecalciferol		3	0.028	0.537	0.053	<0.001	0.340	<0.001
1,25-$(OH)_2D_3$		1	<0.001	<0.001	<0.001	<0.001	0.003	<0.001
1,25-$(OH)_2D_3$ × cholecalciferol		3	0.006	0.756	0.056	<0.001	0.093	<0.001
Regression No 1,25-$(OH)_2D_3$		df						
Linear		1	0.521	0.454	0.591	<0.001	<0.001	<0.001
Quadratic Plus 1,25-$(OH)_2D_3$		1	0.011	0.283	0.091	0.068	0.649	0.007
Linear		1	0.923	0.616	0.984	0.987	0.599	0.747
Quadratic		1	0.232	0.602	0.929	0.285	0.845	0.130
			Main effect means					
1,25-$(OH)_2D_3$								
0 μg/kg			55[a]	2.74[a]	43[a]	60[a]	2.05[a]	44[a]
10 μg/kg			23[b]	1.51[b]	3[b]	8[b]	0.83[b]	1[b]
Cholecalciferol								
0 μg/kg			27[b]	2.08	12[b]	53[a]	1.75	43[a]
5 μg/kg			47[a]	2.25	30[a]	44[a]	1.67	35[a]
27.5 μg/kg			43[a]	1.95	29[a]	20[b]	1.44	9[b]
50 μg/kg			38[ab]	2.21	22[ab]	17[b]	0.92	2[b]

[a–c]Values of the same variable with no common superscript differ significantly ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Exposure to ultraviolet light prevented by covering room fluorescent lights and windows with clear plastic and turning off battery fluorescent lights.

[3]Percentage of birds scored number 3 (large mass of cartilage in the proximal tibiotarsus).

[4]Percentage of birds scored number 3 (very wide growth plate proliferating zone).

both Ca and cholecalciferol with 1,25-$(OH)_2D_3$ will reduce the development of both rickets and TD.

We had hypothesized that the young, fast-growing bird is unable to produce 1,25-$(OH)_2D_3$ from dietary cholecalciferol rapidly or efficiently enough to meet its needs. This theory was neither substantiated or refuted by the plasma data in Experiment 4. In the absence of 1,25-$(OH)_2D_3$, 27.5 μg/kg cholecalciferol stimulated plasma 1,25-$(OH)_2D_3$, plasma Ca, and bone ash values equivalent to those obtained by 1,25-$(OH)_2D_3$. However, the incidence of TD and rickets was still quite high even though the plasma levels of 1,25-$(OH)_2D_3$ were similar with or without 1,25-$(OH)_2D_3$ supplementation. In Experiments 2 and 3 conducted with either 1,25-$(OH)_2D_3$ or fluorescent lighting as treatments, great differences existed in the incidence and severity of TD and percentage bone ash at 50.0 μg/kg cholecalciferol. It is unfortunate that we were not able to measure plasma 1,25-$(OH)_2D_3$ in these experiments. More experiments need to be conducted in this area.

It is obvious that the National Research Council (1994) requirement of 5.0 μg/kg is inadequate for young broiler chickens not exposed to fluorescent lighting and sunlight and that the requirement varies depending on the criteria measured. Indeed, because 50.0 μg/kg cholecalciferol was unable to reduce the incidence and severity of TD to levels achieved by fluorescent lighting and 0 μg/kg cholecalciferol, a question remains whether dietary supplementation with cholecalciferol alone can reduce the apparent cholecalciferol deficiency induced by the absence of fluorescent lighting or sunlight. Hidiroglou and Karpinski (1989) conducted a study in which confined, shorn sheep were either fed 50 μg/d cholecalciferol via bolus for 75 d or exposed for 10 h daily to ultraviolet light from two Westinghouse FS 40 fluorescent light tubes (providing ultraviolet rays in the 280 to 320 nm range) mounted 1.5 cm from the sheep's back for 60 d. In the sheep administered cholecalciferol orally, plasma cholecalciferol increased until a plateau of 0.94 ng/mL was reached at 56 d. Plasma 25-(OH)D_3

TABLE 6. Effect of cholecalciferol and 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] on growth, feed efficiency, bone ash, plasma Ca, plasma dialyzable P, and plasma 1,25-$(OH)_2D_3$ in broiler chickens not exposed to ultraviolet light[2], Experiment 4

Treatments					Plasma mineral		
Cholecalciferol	1,25-$(OH)_2D_3$	16-d BW[1]	Gain:feed ratio[1]	Bone ash[1]	Ca[1]	P[1,3]	1,25-$(OH)_2D_3$[1]
(μg/kg)		(g)	(g:g)	(%)	(mg/dL)		(pg/mL)
0	0	399^{b}	0.705^{a}	28.1^{c}	7.0^{b}	7.3	70^{b}
5	0	427^{ab}	0.712^{a}	31.5^{b}	8.2^{b}	6.2	68^{b}
27.5	0	455^{a}	0.719^{a}	39.6^{a}	11.1^{a}	8.5	103^{ab}
50	0	457^{a}	0.730^{a}	40.5^{a}	11.3^{a}	7.9	104^{ab}
0	10	409^{ab}	0.700^{ab}	40.4^{a}	12.3^{a}	7.4	101^{ab}
5	10	379^{b}	0.650^{b}	39.7^{a}	12.1^{a}	6.1	116^{ab}
27.5	10	414^{ab}	0.691^{ab}	39.9^{a}	12.0^{a}	6.9	110^{ab}
50	10	423^{ab}	0.688^{ab}	40.2^{a}	12.6^{a}	7.8	133^{a}
$\bar{x}$ ± SEM		420 ± 16	0.699 ± 0.016	37.5 ± 5	10.8 ± 6	7.3 ± 11	100 ± 19
ANOVA		Probability					
Source of variation	df						
Cholecalciferol	3	0.056	0.341	<0.001	0.003	0.419	0.328
1,25-$(OH)_2D_3$	1	0.021	0.007	<0.001	<0.001	0.598	0.045
1,25-$(OH)_2D_3$ × cholecalciferol	3	0.279	0.411	<0.001	0.004	0.841	0.747
Regression No 1,25-$(OH)_2D_3$	df						
Linear	1	0.025	0.301	<0.001	<0.001	0.323	0.037
Quadratic Plus 1,25-$(OH)_2D_3$	1	0.224	0.993	<0.001	0.004	0.544	0.390
Linear	1	0.180	0.537	0.932	0.721	0.513	0.381
Quadratic	1	0.819	0.862	0.171	0.567	0.572	0.772
		Main effect means					
1,25-$(OH)_2D_3$							
0 μg/kg		435^{a}	0.717^{a}	34.9^{b}	9.4^{b}	7.5	86^{b}
10 μg/kg		406^{b}	0.681^{b}	40.1^{a}	12.2^{a}	7.1	115^{a}
Cholecalciferol							
0 μg/kg		404^{b}	0.701	34.3^{c}	9.6^{b}	7.4	85
5 μg/kg		403^{b}	0.681	35.6^{b}	10.2^{b}	6.2	92
27.5 μg/kg		434^{ab}	0.705	39.7^{a}	11.5^{a}	7.7	107
50 μg/kg		440^{a}	0.709	40.3^{a}	11.9^{a}	7.8	118

$^{a-c}$Values of the same variable with no common superscript are significantly different ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Exposure to ultraviolet light prevented by covering room fluorescent lights and battery fluorescent lights with Arm-a-Lite ultraviolet light filter tubes (Thermoplastic Processes, Stirling, NJ) and covering windows with clear plastic.

[3]Plasma dialyzable P.

increased and reached a plateau of 21 ng/mL after 70 d. In the animals exposed to ultraviolet lights, plasma cholecalciferol reached a plateau at 2.03 ng/mL and plasma 25-(OH)D_3 plateaued at 29.6 ng/mL after 49 d. Plasma cholecalciferol and plasma 25-(OH)D_3 levels were significantly higher in the animals exposed to ultraviolet light by 7 d of treatment and remained so throughout the duration of the experiment (Hidiroglou and Karpinski, 1989). The results indicate that animals exposed to fluorescent lighting have a higher physiological set point that is attained earlier than that of animals fed daily boluses of cholecalciferol and that in sheared, confined sheep ultraviolet light is a more effective way of improving low cholecalciferol status than oral supplementation with cholecalciferol (Hidiroglou and Karpinski, 1989). Hidiroglou (1987) obtained similar results with sheep only exposed to the fluorescent lights for 2 h/d and Davie and Lawson (1980) reported similar results in elderly humans. The data from the present studies suggest that this may also be true in broiler chickens.

A summary of the significant three-way interactions observed in Experiment 1 might be more easily understood by first considering the significant two-way interactions in Experiments 2, 3, and 4. For bone ash, there is a significant effect of light at low cholecalciferol levels but no effect at high cholecalciferol levels in Experiment 2. In Experiments 3 and 4, there is a significant effect of 1,25-$(OH)_2D_3$ at low cholecalciferol but no effect at high cholecalciferol levels. In Experiment 1, a significant increase in bone ash was obtained at high cholecalciferol levels from 1,25-$(OH)_2D_3$ supplementation in the presence or absence of light. The significant response to 1,25-$(OH)_2D_3$ supplementation in this experiment at high levels of cholecalciferol in the presence or absence of light results in the three-way interaction. This response to 1,25-$(OH)_2D_3$ at high levels of cholecalciferol was also obtained in Experiments 3 and 4, but light was not a variable in these studies. The significant three-way interaction for the incidence of rickets was primarily due to the fact that having the light on chickens receiving the high cholecalciferol diet, but no 1,25-$(OH)_2D_3$, did not significantly decrease rickets as it had when chicks received the low cholecalciferol diet.

There was some indication in the present studies that both 50.0 μg/kg cholecalciferol and 10 μg/kg

TABLE 7. Effect of dietary cholecalciferol and 1,25-dihydroxycholecalciferol [1,25-$(OH)_2D_3$] on the incidence and severity of TD and rickets in broiler cockerels not exposed to ultraviolet light[2], Experiment 4

Treatments			Tibial dyschondroplasia			Rickets		
Cholecalciferol	1,25-$(OH)_2D_3$		Incidence[1]	Score[1]	No. 3[1,3]	Incidence[1]	Score[1]	No. 3[1,4]
(μg/kg)			(%)		(%)			(%)
0	0		52^{ab}	2.93	48^{b}	93^{a}	2.68^{a}	67^{a}
5	0		76^{a}	2.93	69^{a}	89^{a}	2.25^{a}	48^{a}
27.5	0		30^{bc}	2.17	13^{c}	17^{b}	0.83^{b}	0^{b}
50	0		37^{bc}	1.83	10^{cd}	20^{b}	1.00^{b}	0^{b}
0	10		20^{c}	1.11	0^{e}	3^{b}	0.33^{b}	0^{b}
5	10		20^{c}	2.25	7^{cde}	10^{b}	0.56^{b}	0^{b}
27.5	10		17^{c}	3.92	3de	3^{b}	0.67^{b}	0^{b}
50	10		24^{bc}	1.08	0de	4^{b}	0.67^{b}	0^{b}
$\bar{x}$ ± SEM			34 ± 9	2.28 ± 1.11	19 ± 3	30 ± 6	1.12 ± 0.39	14 ± 7
ANOVA			Probability					
Source of variation		df						
Cholecalciferol		3	0.079	0.531	<0.001	<0.001	0.161	<0.001
1,25-$(OH)_2D_3$		1	<0.001	0.639	<0.001	<0.001	0.001	<0.001
1,25-$(OH)_2D_3$ × cholecalciferol		3	0.097	0.451	<0.001	<0.001	0.035	<0.001
Regression No 1,25-$(OH)_2D_3$		df						
Linear		1	0.049	0.002	<0.001	<0.001	0.002	0.001
Quadratic Plus 1,25-$(OH)_2D_3$		1	0.395	0.602	0.256	0.004	0.011	0.027
Linear		1	0.819	0.991	0.513	0.669	0.638	0.147
Quadratic		1	0.638	0.150	0.315	0.982	0.790	0.371
			Main effect means					
1,25-$(OH)_2D_3$								
0 μg/kg			49^{a}	2.5	35^{a}	55^{a}	1.7^{a}	29^{a}
10 μg/kg			20^{b}	2.1	3^{b}	5^{b}	0.6^{b}	0^{b}
Cholecalciferol								
0 μg/kg			36^{ab}	2.0	24^{b}	48^{a}	1.5	34^{a}
5 μg/kg			48^{a}	2.6	38^{a}	50^{a}	1.4	24^{a}
27.5 μg/kg			23^{b}	3.0	8^{c}	10^{b}	0.8	0^{b}
50 μg/kg			30^{ab}	1.5	5^{c}	12^{b}	0.8	2^{b}

$^{a-e}$Values of the same variable with no common superscript are significantly different ($P \leq 0.05$); results of Duncan's new multiple range test.

[1]Means of three pens per treatment.

[2]Exposure to ultraviolet light prevented by covering fluorescent lights with Arm-a-Lite ultraviolet light filter tubes (Thermoplastic Processes, NJ) and windows with plastic.

[3]Percentages of birds scored number 3 (large mass of cartilage in the proximal tibiotarsus).

[4]Percentage of birds scored number 3 (wide growth plate proliferating zone with no calcification).

1,25-$(OH)_2D_3$ may be toxic to chickens. Edwards (1989a, 1990) has observed some depression in weight gain and feed efficiency as a result of 1,25-$(OH)_2D_3$ supplementation in broiler chicks. In cholecalciferol-replete humans, exposure to ultraviolet light increases plasma cholecalciferol and plasma 1,25-$(OH)_2D_3$ levels after 1 or 2 d. If exposure to ultraviolet light continues, the levels then decline to their original level (Adams *et al.*, 1982). In cholecalciferol-deficient humans, exposure to ultraviolet light will also increase plasma cholecalciferol and plasma 1,25-$(OH)_2D_3$ levels, but with continued exposure the levels will decline to a new set point well above that which existed in their cholecalciferol-deficient state (Adams *et al.*, 1982). This information suggests that when cholecalciferol is provided by the skin due to exposure to ultraviolet light, it is very difficult to induce a cholecalciferol toxicity. Upon exposure, the ultraviolet light penetrates the skin and the high energy photons between 290 and 315 nm interact with the 5,7-diene of 7-dehydrocholesterol (procholecalciferol) in the epidermis. The 5,7-diene absorbs these photons and undergoes a photochemical reaction to produce the thermally labile precholecalciferol, which isomerizes to the more thermodynamically stable cholecalciferol. Cholecalciferol is translocated to the circulation by the vitamin D binding protein (Holick *et al.*, 1980, 1981). Adams *et al.* (1982) and Holick *et al.* (1980) have demonstrated that prolonged exposure to ultraviolet light does not further increase plasma levels of cholecalciferol and its metabolites. With prolonged exposure 7-dehydrocholesterol levels decrease, and precholecalciferol, plasma cholecalciferol, and plasma cholecalciferol metabolites remain constant. This condition occurs because precholecalciferol is also photolabile. With continued photolysis of 7-dehydrocholesterol to precholecalciferol, the precholecalciferol begins to photolyze to two additional photoproducts, lumisterol and tachysterol, which have no effect on intestinal Ca absorption or bone Ca mobilization. The result is that prolonged exposure to ultraviolet light does not increase precholecalciferol concentration above 15% of the initial 7-dehydrocholesterol concentration (Holick, 1987). It would appear that the risk of cholecalciferol toxicity is lower when the cholecalciferol needs are met primarily

with ultraviolet light. Further research is needed to determine whether it might be economically feasible for poultry producers to use fluorescent lights in broiler houses during initial brooding to reduce the incidence of leg abnormalities in commercial broiler flocks.

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