Some Observations on the Dietary Vitamin D Requirement of Weanling Pups

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ABSTRACT A study was conducted to test the hypothesis that growing dogs do not require supplementation with cholecalciferol (vitamin D-3) in a nonpurified extruded diet. Twenty-eight weanling pups from four litters of English pointers and two litters of German Shepherd dogs were allotted equally to two groups by gender and weight. Dogs were raised in indoor-outdoor kennel runs for the 102-wk evaluation. One group was fed a diet that contained no added vitamin D while the other group was fed the same diet to which had been added 60.5 μg cholecalciferol/kg diet. Both diets contained ~1.4% calcium and 1.0% phosphorus. Growth as measured by body weight and length, serum calcium, phosphorus and alkaline phosphatase and urine hydroxyproline were not significantly different (P > 0.05) between the two treatment groups. There was no relationship between dietary treatment and nutritional secondary hyperparathyroidism (NSH), rickets or other skeletal abnormalities. The data support the concept that addition of vitamin D to typical commercial dog foods for purposes of prevention or amelioration of rickets, NSH or other skeletal diseases is unnecessary. J. Nutr. 121: S66-S69, 1991.

INDEXING KEY WORDS:
• symposium • dogs • vitamin D

Vitamin D rickets has been produced experimentally in the dog (1–5). Generally, there was an accompanying deficiency in dietary calcium and/or phosphorus, or the diets were imbalanced with respect to calcium/phosphorus ratio (where percent calcium in the diet was less than percent phosphorus). Vitamin-D–deficiency rickets has also been reported in dogs fed a diet reported to be adequate in calcium and phosphorus [6]. In most studies, the amount of ultraviolet light provided was not reported (1–5).

It has been suggested that the dog may not possess the mechanisms for vitamin D synthesis in the skin [7]. In one study, rickets did not occur when 6-wk-old pups were housed in total darkness for 8 wk while fed nonpurified diets without added vitamin D [R. D. Kealy, unpublished data]. The results of another study indicated that rickets in dogs cannot be prevented or treated by ultraviolet radiation [6].

Nevertheless, the need for 1,25-dihydroxycholecalciferol for intestinal calcium absorption in the dog has been demonstrated [4]. Also, 1,25-dihydroxycholecalciferol is produced by the kidney of the dog, as in other mammalian species [8].

The purpose of this study was to investigate whether pups of large breeds of dogs fed a nonpurified diet for 2 y to which no vitamin D was added developed rickets when housed in indoor/outdoor runs.

METHODS AND MATERIALS

Four litters of English pointers and two litters of German Shepherd dogs were allotted to two groups by gender and body weight, with 14 dogs per treatment group. Pups were weaned at ~6 wk of age. They received routine vaccinations and antiparasite treatments. All dogs were housed in indoor/outdoor kennel runs (~2 × 19 m) with concrete floors. Food and water were provided ad libitum.

Dry extruded nonpurified diets were prepared for the study using a vitamin-mineral supplement (without added cholecalciferol) plus corn, corn gluten meal, soybean meal, meat and bone meal, rice, animal fat, corn gluten feed, calcium chloride, dicalcium phosphate, sodium chloride and L-lysine hydrochloride. For the treatment group of dogs, the base diet was supplemented with 60.5 μg synthetic cholecalciferol/kg diet.3

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TABLE 1
Average composition of dry, extruded diets (average of laboratory analyses)

<table>
<thead>
<tr>
<th>Added cholecalciferol (μg/kg diet)</th>
<th>0</th>
<th>60.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (sample size)</td>
<td>15</td>
<td>19</td>
</tr>
<tr>
<td>Moisture, g/100 g</td>
<td>8.5</td>
<td>8.2</td>
</tr>
<tr>
<td>Protein, g/100 g</td>
<td>27.0</td>
<td>27.0</td>
</tr>
<tr>
<td>Fat, g/100 g</td>
<td>12.1</td>
<td>11.9</td>
</tr>
<tr>
<td>Calcium, g/100 g</td>
<td>1.35</td>
<td>1.44</td>
</tr>
<tr>
<td>Phosphorus, g/100 g</td>
<td>1.00</td>
<td>1.03</td>
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</tbody>
</table>

1 The vitamin premix used in both diets included retinyl palmitate, riboflavin, vitamin E, biotin, vitamin B-12, calcium pantothenate, folic acid, choline chloride, menadione sodium bisulfite complex, niacin, pyridoxine hydrochloride and thiamine hydrochloride. Trace minerals included cobalt carbonate, copper oxide, ferrous sulfate, manganese oxide, calcium iodate and zinc oxide. Nutrient levels met or exceeded standards established by The National Research Council (11).

Multiple batches of each diet were produced. Representative samples from each batch were analyzed using methods of the Association of Official Analytical Chemists (Table 1). Dietary calcium contents were determined by atomic-absorption spectrophotometry, and phosphorus was determined by colorimetry. Vitamin D analyses of diets were not obtained.

Physical examinations were conducted on all dogs. Blood was collected from all dogs at 11, 16, 30, 54, 80 and 108 wk of age. Blood was collected by jugular venipuncture, allowed to clot and centrifuged at 1325 × g for 12 min. Urine was collected by catheterization using aseptic techniques at all intervals except 80 and 108 wk of age. Analyses for serum calcium, phosphorus, alkaline phosphatase (EC 3.1.3.1) and urine creatinine were conducted on an autoanalyzer. Analysis of urine for hydroxyproline was done by a previously described procedure (9). The body weight and length of the dogs were measured at intervals. Food consumption was measured by pen.

Development of the pelvis, hip joints and femurs was examined by radiography at 30, 42, 54, 80 and 108 wk of age. Data were analyzed by analyses of variance (10). Mean results plotted include all data available at the time measurements were taken.

RESULTS

With one exception, all dogs remained healthy. One German Shepherd dog was removed from the study because of symptomatic hip dysplasia. This dog had been fed the nonvitamin-D–supplemented diet. Two other English pointers, one per treatment, were removed due to space/behavioral problems.

Regardless of breed, the dogs from the two treatments exhibited similar body weight, body length and daily food consumption (Table 2). The weight trends of dogs on both treatments were typical of their respective breeds [Fig. 1].

Serum calcium, serum phosphorus and serum alkaline phosphatase were analyzed statistically to detect differences between treatment groups in either or both breeds studied. Over time, no significant differences attributable to treatment were observed (P > 0.05).

Serum phosphorus and serum alkaline phosphatase significantly decreased with time in each group (P < 0.05) [Fig. 2]. All blood serum values were within reference intervals.

Urine hydroxyproline was expressed as hydroxyproline/creatinine ratio to adjust for variation in urine output. Urine hydroxyproline:creatinine did not differ significantly (P > 0.05) by treatment (Fig. 1). However, the ratio declined markedly during the first year of growth.

Observations from radiographs of pelvis, hip joints and femurs did not reveal any apparent dietary treatment effects.

DISCUSSION

The results of this study suggest that large breed dogs housed in indoor-outdoor kennel runs do not require added cholecalciferol when nonpurified diets are fed during the first 2 y of life. The breeds in this study were selected because developmental bone disorders

<table>
<thead>
<tr>
<th>Added cholecalciferol (μg/kg diet)</th>
<th>0</th>
<th>60.5</th>
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<tbody>
<tr>
<td>German Shepherd dogs:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>31.7 ± 1.21</td>
<td>30.2 ± 1.1</td>
</tr>
<tr>
<td>Length, cm²</td>
<td>113.7 ± 1.3</td>
<td>111.0 ± 1.1</td>
</tr>
<tr>
<td>Food intake/d, kg</td>
<td>0.63</td>
<td>0.64</td>
</tr>
<tr>
<td>English pointers:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>24.5 ± 0.90</td>
<td>24.9 ± 0.88</td>
</tr>
<tr>
<td>Length, cm²</td>
<td>91.6 ± 0.97</td>
<td>93.2 ± 0.94</td>
</tr>
<tr>
<td>Food intake/d, kg</td>
<td>0.58</td>
<td>0.58</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM.
2 Body length was only taken until 54 wk of age.
FIGURE 1 Weight change for eight English pointers (upper panel) and four or five German Shepherds (lower panel) dogs fed diets with or without added cholecalciferol. Values are means ± SEM.

occur more frequently in the larger breeds of dogs compared with many of the smaller breeds. The single case of symptomatic hip dysplasia occurred in a dog fed the nonvitamin-D-supplemented diet and was apparently unrelated to treatment.

Dogs fed diets with and without supplemental cholecalciferol did not differ in growth rate, food consumption or selected serum or urine values. Likewise, there were no differences between the two groups in the results of physical examination and radiographic evaluation. The lack of response to added cholecalciferol was probably due to adequate levels of calcium and phosphorus in the base diet and possible synthesis of vitamin D. However, it is possible that some vitamin D could have been present in one or more of the ingredients of the basal diet. It has been suggested that dogs may only require additional vitamin D when there is a mineral deficiency or imbalance in the diet [11]. However, one study reported canine rickets in diets containing 1.2% calcium and 1% phosphorus [6].

It has been reported that carnivores may not possess the mechanism of vitamin D synthesis in the skin [7]. Another study demonstrated that dogs fed a nonpurified diet without added vitamin D under conditions of total darkness did not exhibit bone defects [Kealy, unpublished data]. Previous reports did not record the ultraviolet light status of the environment. It is not

FIGURE 2 Serum calcium (a) phosphorus (b) and alkaline phosphatase (c) values and urine hydroxyproline:creatinine ratios (d) for dogs fed diets with or without added cholecalciferol (vitamin D). Urine hydroxyproline was corrected against urine creatinine in the same urine sample (μg hydroxyproline/mg creatinine). Values are means ± SEM for 12 or 13 dogs.
understood at this time how the dog acquires sufficient vitamin D for metabolism. Part of the explanation appears to be related to a very low vitamin D requirement in the presence of adequate dietary mineral balance.

The observation that large breed dogs raised in indoor-outdoor kennel runs do not require added vitamin D is important because supplemental vitamin D, calcium and phosphorus are frequently recommended and used at levels in excess of the nutritional requirements of the dog, presumably to enhance bone growth and development. The data reported here suggest that supplementation of nonpurified, commercially available dog foods with vitamin D may not be necessary.

LITERATURE CITED