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J ANIM SCI 2000, 78:2706-2712.

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A kinetic model of phosphorus metabolism in growing goats[1]

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ABSTRACT: The effect of increasing phosphorus (P) intake on P utilization was investigated in balance experiments using 12 Saanen goats, 4 to 5 mo of age and weighing 20 to 30 kg. The goats were given similar diets with various concentrations of P, and ^{32}P was injected to trace the movement of P in the body. A P metabolism model with four pools was developed to compute P exchanges in the system. The results showed that P absorption, bone resorption, and excretion of urinary P and endogenous and fecal P all play a part in the homeostatic control of P. Endogenous fecal output was positively correlated to P intake ($P < .01$). Bone resorption of P was not influenced by intake of P, and P recycling from tissues to the blood pool was lesser for low P intake. Endogenous P loss occurred even in animals fed an inadequate P diet, resulting in a negative P balance. The extrapolated minimum endogenous loss in feces was .067 g of P/d. The minimum P intake for maintenance in Saanen goats was calculated to be .61 g of P/d or .055 g of P/(kg$^{.75}$·d) at 25 kg BW. Model outputs indicate greater P flow from the blood pool to the gut and vice versa as P intake increased. Intake of P did not significantly affect P flow from bone and soft tissue to blood. The kinetic model and regressions could be used to estimate P requirement and the fate of P in goats and could also be extrapolated to both sheep and cattle.

Key Words: Goats, Metabolism, Mineral Absorption, Phosphorus, Simulation Models

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J. Anim. Sci. 2000. 78:2706–2712

Introduction

Phosphorus is an essential nutrient and, as phosphate, is involved in most of the metabolic activities of the body as well as in bone formation (Symonds and Forbes, 1993). However, there is a controversy over the exact mechanisms involved in P homeostasis (Challa et al., 1989). It is not clear whether homeostasis is regulated by P absorption, salivary P, excretion of P in urine, or a combination of two or all factors.

Work toward improving the understanding of P metabolism in ruminants has been carried out for some time. A number of workers have used isotope dilution and balance methods to study the absorption of P in sheep and cattle (Braithwaite, 1983; Schneider et al., 1985; Salviano and Vitti, 1998). Radioisotopes have been used to study the distribution of P by applying compartmental models (Grace, 1981; Schneider et al., 1987), and the kinetics of ^{32}P following intravenous injection have been studied using a compartmental analysis computer program (Boston et al., 1981). However, the studies had limited application in calculating P requirement and extrapolating to other ruminants because only one level of P concentration was considered. In addition, the existing published information related to P metabolism in the goat is relatively little and inconsistent (e.g., Akinsoyinu, 1986; Muschen et al., 1988).

Little is known, for example, about the blood and soft tissue P pools of the body and their rates of P inflow and outflow in goats and other ruminants. Quantitative information on many indices of P metabolism, such as fecal endogenous P losses and true absorption, is needed. Variation in P intake might affect P utilization and homeostasis, and low P intakes might result in low P levels in the blood and soft tissue P pools of the body. The

[1]The authors would like to thank Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP Proc. 98/13406-3) and Conselho Nacional de Desenvolvimento Científico e Tecnológico for financial support.

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Received November 29, 1999.
Accepted April 25, 2000.

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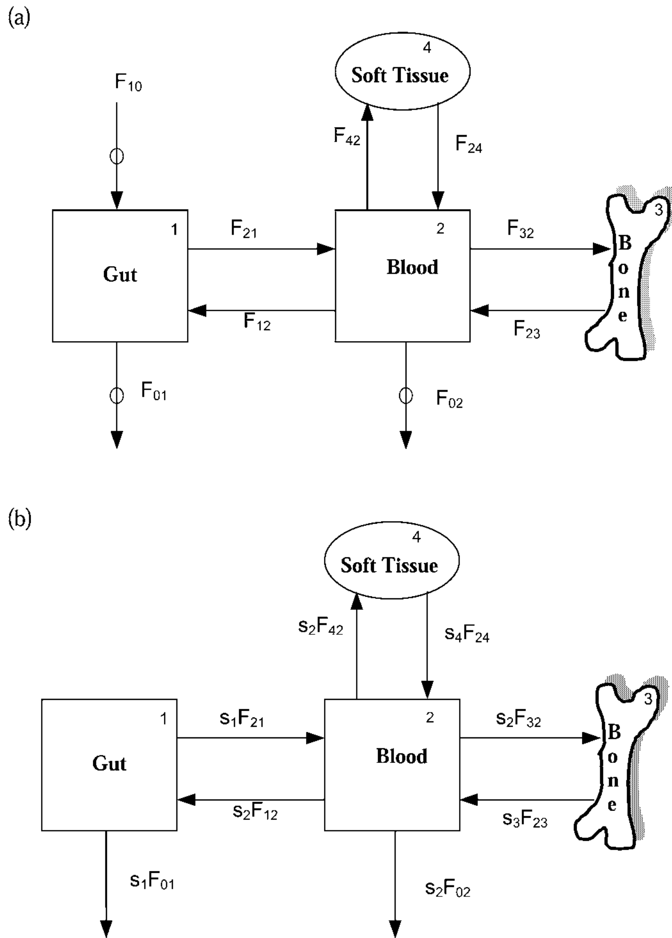

Figure 1. Schematic representation of the model of phosphorus metabolism in goats: (a) unlabeled P and (b) labeled P; F_{ij} is the total flux of pool i from j, F_{i0} is an external flux into pool i, and F_{0j} a flux from pool j out of the system. Specific activity of pool i is represented by s_i, and circles denote fluxes measured experimentally.

purpose of this research was to use data from balance and kinetic studies to propose and solve a model of P metabolism in growing goats fed increasing levels of P. The nutritional and metabolic implications of increasing P intake are evaluated.

Materials and Methods

Model Development

The proposed model of whole-body P metabolism is the simplified scheme based on P flows shown in Figure 1a. It contains four pools of P: 1) gut lumen, 2) blood, 3) bone, and 4) soft tissue. The fluxes of P between pools and into and out of the system are shown as arrowed lines. The gut lumen, bone, and soft tissue pools interchange in two directions with the blood pool, with fluxes F_{21} and F_{12}, F_{23} and F_{32}, and F_{24} and F_{42}, respectively. Phosphorus entry to the system is via intake, F_{10}, and exit via feces, F_{01}, and urine, F_{02}. The scheme adopted for the movement of label is shown in Figure 1b. Labeled ^{32}P was administered intravenously as a single dose, D, at time zero, and the size and specific activity of the blood, bone, and soft tissue pools measured after 8 d. The scheme assumes there is no reentry of label from external sources.

Conservation of mass principles can be applied to each pool in Figure 1 to generate differential equations that describe the dynamic behavior of the system. For unlabeled P, these differential equations are (the mathematical notation is defined in Table 1):

$$dQ_1/dt = F_{10} + F_{12} - F_{01} - F_{21} \quad [1]$$

$$dQ_2/dt = F_{21} + F_{23} + F_{24} - F_{02} - F_{12} - F_{32} - F_{42} \quad [2]$$

$$dQ_3/dt = F_{32} - F_{23} \quad [3]$$

$$dQ_4/dt = F_{42} - F_{24} \quad [4]$$

and for label:

$$dq_1/dt = s_2F_{12} - s_1(F_{01} + F_{21}) \quad [5]$$

$$dq_2/dt = s_1F_{21} + s_3F_{23} + s_4F_{24} - s_2(F_{02} + F_{12} + F_{32} + F_{42}) \quad [6]$$

$$dq_3/dt = s_2F_{32} - s_3F_{23} \quad [7]$$

$$dq_4/dt = s_2F_{42} - s_4F_{24} \quad [8]$$

Consider the differential coefficient of s_3 with respect to time and differentiating $q_3Q_3^{-1}$ as a product:

$$ds_3/dt = d(q_3Q_3^{-1})/dt = [dq_3/dt - (q_3/Q_3)dQ_3/dt]/Q_3 \quad [9]$$

Rearranging gives

$$dq_3/dt = Q_3ds_3/dt + s_3dQ_3/dt \quad [10]$$

Using Eq. [3] and [7] to substitute for dQ_3/dt and dq_3/dt, respectively, and approximating ds_3/dt by $[s_3 - s_3(0)]/[t - 0]$, Eq. [10] becomes

$$s_3/t = (s_2 - s_3)F_{32}/Q_3 \quad [11]$$

as $s_3(0)$, the value of s_3 at time zero, is zero. Similarly, consideration of ds_4/dt yields

$$s_4/t = (s_2 - s_4)F_{42}/Q_4 \quad [12]$$

After 8 d, we assumed that Pool 1 (gut lumen) was in complete steady state (i.e., both dQ_1/dt and dq_1/dt are zero) and Pool 2 (blood) was in nonisotopic steady state (i.e., dQ_2/dt is zero). Equations [1], [2], [5], [11], and [12] now become, respectively

$$F_{10} + F_{12} - F_{01} - F_{21} = 0 \quad [13]$$

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Table 1. Principal symbols used in the model

Symbol	Meaning
F_{ij}	Total flux of P to pool i from j; F_{i0} denotes an external flux into pool i and F_{0j} a flux from pool j out of the system; a tilde indicates a flux that can be measured experimentally. Units are g/d.
D	Dose of ^{32}P administered to blood at time zero: dpm
Q_i	Total quantity of P in pool i: g
q_i	Quantity of ^{32}P in pool i: dpm
s_i	Specific activity of pool i (= q_i/Q_i): dpm/g
t	Time: d

$$F_{21} + F_{23} + F_{24} - F_{02} - F_{12} - F_{32} - F_{42} = 0 \quad [14]$$

$$s_2F_{12} - s_1(F_{01} + F_{21}) = 0 \quad [15]$$

$$(s_2 - s_3)F_{32}/Q_3 = s_3/8 \quad [16]$$

$$(s_2 - s_4)F_{42}/Q_4 = s_4/8 \quad [17]$$

Algebraic manipulation of Eq. [13] to [17] gives

$$F_{12} = s_1\tilde{F}_{10}/(s_2 - s_1) \quad [18]$$

$$F_{21} = \tilde{F}_{10} + F_{12} - \tilde{F}_{01} \quad [19]$$

$$F_{32} = s_3Q_3/[8(s_2 - s_3)] \quad [20]$$

$$F_{42} = s_4Q_4/[8(s_2 - s_4)] \quad [21]$$

$$|F_{23} + F_{24}| = \tilde{F}_{02} + F_{12} + F_{32} + F_{42} - F_{21} \quad [22]$$

where the tilde indicates an experimentally measured flux. The combined flux $|F_{23} + F_{24}|$, which denotes the sum of outflow from Pool 3 and outflow from Pool 4,

$$|F_{23} + F_{24}| = F_{23} + F_{24} \quad [23]$$

can be partitioned by combining Pools 3 and 4. Let s^* denote the specific activity of the combined pool. This is calculated as

$$s^* = (s_3Q_3 + s_4Q_4)/(Q_3 + Q_4) \quad [24]$$

The outflow of label from the combined pool is the sum of the outflow of label from Pool 3 and the outflow of label from Pool 4:

$$s^* \times |F_{23} + F_{24}| = s_3F_{23} + s_4F_{24} \quad [25]$$

Algebraic manipulation of Eq. [23] and [25] gives

$$F_{24} = (s^* - s_3) \times |F_{23} + F_{24}|/(s_4 - s^*) \quad [26]$$

$$F_{23} = |F_{23} + F_{24}| - F_{24} \quad [27]$$

The model is applied by using Eq. [18] to [22], [24], and [26] to [27] to compute the unknown fluxes.

Experimental Procedure

Twelve Saanen goats, 4 to 5 mo of age and weighing 20 to 30 kg, were housed indoors in metabolism crates designed for isotope studies and handling of feces and urine at the Faculty of Animal Science and Veterinary of Jaboticabal (UNESP). After 15 d, the goats were transferred to the Center for Nuclear Energy in Agriculture (CENA), University of São Paulo, for ^{32}P injection and collection of data. The goats received a diet consisting of a concentrate mixture and *Brachiaria decumbens* hay (Table 2). The hay was offered ad libitum, and P supplementation was offered as dicalcium phosphate to give .42 (low P level, **L**), 1.36 (medium P level, **M**) and 3.63 g P/d (high P level, **H**) (Agricultural and Food Research Council, 1991). For L, the diet was not supplemented with any additional P. Feed was given twice a day for one 28-d period. All animals received vitamins A, D, and E by intramuscular injection (Table 2).

Table 2. Feed ingredients and composition of the diet fed to goats containing low (L), medium (M), and high (H) P concentrations[a]

Item	L	M	H
Ingredient, g/kg DM			
Hay	601	602	576
Cassava meal	380	292	292
Ground corn	0	86.1	85.8
Dicalcium phosphate	0	2.39	15.3
White salt	2.89	2.69	2.93
Mineral mixture[b]	3.01	3.02	3.15
Urea	13.1	9.90	12.1
Calcium carbonate	0	2.02	12.76
Chemical composition			
Dry matter, g/kg as is	90.6	90.7	90.9
Crude protein, g/kg	91.6	91.8	92.4
NDF, g/kg	650	632	610
ADF, g/kg	266	265	254
Ca, g/kg	2.20	3.70	10.7
P, g/kg	.80	1.50	3.80
ME, MJ/kg DM	2.20	2.21	2.16
Ca:P	2.70	2.54	2.81
DMI, g/d	562	919	999
BW, kg	26.4	28.3	28.8

[a]The goats received 20,000 IU vitamin A, 5,000 IU vitamin D, and 6 mg vitamin E.
[b]The mineral mixture contained, in milligrams per kilogram DM, 8.73 Fe, 7.64 Cu, 44.4 Mn, 58.7 Zn, .11 Co, .21 I, and .032 mg Se.

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After 21 d, each animal was given, as a single dose via the right jugular vein, 200 μCi of ^{32}P in 1 mL of sterile isotonic saline (9 g/L NaCl). Blood samples (10 mL) were taken by Vacutainer from the left jugular vein after isotope administration at 24-h intervals for 7 d. Because most of the mobile P in blood is found in plasma, the blood was centrifuged and plasma removed for analysis. Nine milliliters of trichloroacetic acid (100 g/L) was added to 1 mL of plasma for protein precipitation. After centrifugation ($1,100 \times g$), inorganic P was determined by colorimetric analyses (Fiske and Subbarow, 1925).

Phosphorus intake and excretion in feces and urine were recorded daily for 7 d, and subsamples (10% of total outputs) were stored for further analysis. Feces samples (1 g) were dried overnight (105°C) and ashed (500°C for 8 h). The ash was dissolved in concentrated HCl, and P content was determined by a colorimetric method (Sarruge and Haag, 1974). A similar procedure was used to determine P content of food intake. Urine samples (30 mL) were acidified during collection using 100 mL of 12 *N* HCl, which were then dried (55°C) and ashed (500°C). Ashed samples were diluted (3 *N* HCl), and volume was made up to 10 mL (Morse et al., 1992). Inorganic P was determined using vanadate-molybdate reagents (Sarruge and Haag, 1974).

For radioactivity measurements, 1-mL plasma and urine samples were added to 19 mL of distilled water in counting vials. Ashed fecal samples (1 g) were dissolved in 18 *N* H_2SO_4 and placed in counting vials. Radioactivity of ^{32}P was measured in a Packard Liquid Scintillation Spectrometer (model 2450B, A. Canberra Company) using Cerenkov radiation. Specific activities in plasma and feces were determined according to Lofgreen and Kleiber (1953).

After the end of collection period, the goats were killed by intravenous injection of pentobarbital (200 mg/mL), and tissues (liver, heart, kidney, and muscles) and bone samples (12th rib) were collected once. The material was cleaned, weighed, and autoclaved. Samples were ground and dissolved in 18 *N* H_2SO_4. The extract was transferred to vials for radioactivity determination. For mineral determination, bone samples were dissolved in concentrated HCl (Sarruge and Haag, 1974). Bone specific activity in 1 g DM and ^{32}P incorporation in bone were calculated according to Lofgreen and Kleiber (1953).

Statistical Analyses

Experimental measurements (model inputs) and model outputs were analyzed as a completely randomized design. The data used in the analysis were from nine animals, with three from each level of P intake due to incomplete results for the other animals. A comparison of means between each level of P intake was carried out using the General Linear Models procedure (SAS, 1990), with the sources of variation being P concentrations (degrees of freedom of the error = 6). The SEM was calculated as $S/\sqrt{3}$. Treatment means were assessed using the least significant difference method when overall treatment effects were $P < .05$. Regression analysis was carried out using the PROC REG procedure (SAS, 1990), and the SE of coefficients was reported.

Results

Daily intake and daily excretion of P and specific activities and P contents in bone, blood, and soft tissues are summarized in Table 3. The values are the means of three goats grouped according to P intake level. Diet L supplied only .42 g P/d and it was largely deficient in P. This resulted in a negative P balance (−.081 g/d) for goats fed diet L. Diet M supplied 1.36 g P/d, which was considered moderately deficient, but the H diet supplied 3.63 g P/d, which was adequate according to ARC (1980).

Total P excreted in feces (F_{01}) increased with P intake (F_{10}), and there was a highly significant linear relationship between these fluxes. Total P excreted in feces was about 74 and 44% higher for goats on Diet H compared with L and, on M compared with L, respectively. Urinary loss of P was low for all treatments, representing .95, .29, and .17% of dietary P for L, M, and H diets, respectively. There were no significant differences in losses of P in urine between the three diets.

Specific activities and P contents in blood and soft tissues were affected by P intake. The values for specific activities were greater ($P < .05$) and blood and soft tissue P were lower ($P < .05$) in goats on L diet.

The endogenous fecal loss (F_{e01}, where $F_{e01} = F_{12}\tilde{F}_{01}/(F_{12} + \tilde{F}_{10}) = s_1\tilde{F}_{10}/s_2$) was .15, .35, and .89 g P/d for Diets L, M, and H, respectively. The endogenous fecal loss of P increased significantly with increased P intake as given by the following equation:

$$F_{e01} = .067 + .21F_{10} \qquad [28]$$

(SE = .034 and .014 for intercept and slope, respectively; n = 9; $r^2 = .97$)

Total endogenous losses represented 36% of P intake for unsupplemented animals (L). In goats fed Diets M and H, endogenous P represented about 25% of P intake. Truly absorbed P, which is the amount of dietary P absorbed by the goats (F_{d21}, where $F_{d21} = F_{10} - [F_{01} - F_{e01}]$) was .04, .77, and 2.49 g of P/d in L, M, and H diets respectively. Whereas P was truly absorbed from the L diet at only 10% efficiency (true absorption/intake), true absorption from M and H diets was at 57 and 69% efficiency, respectively. There was a significant linear relationship between the truly absorbed P and P intake, which was similar to reported values of .64 to .70 (Agricultural and Food Research Council, 1998):

$$F_{d21} = -.18 + .70F_{10} \qquad [29]$$

(SE = .14 and .062 for intercept and slope, respectively; n = 9; $r^2 = .95$)

The retention of P was negative on the deficient P diet (−.11 g/d) when the absorption was low but became

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Table 3. Phosphorus intake levels, specific radioactivities (SRA), and other inputs used in the model. The values shown are mean values for each treatment

Item	Symbol[ab]	Level of P intake			SEM
		Low	Medium	High	
Input fluxes, g of P/d					
Intake	F_{10}	.42[z]	1.36[y]	3.63[x]	.27
Feces	F_{01}	.53[y]	.94[y]	2.03[x]	.29
Urine	F_{02}	.0041	.0043	.0057	.0016
SRA, dpm/g					
Feces	s_1	19.3[y]	12.6[xy]	10.4[x]	2.3
Blood	s_2	69.3[y]	34.0[x]	23.7[x]	4.8
Bone	s_3	2.30	3.07	2.80	.40
Soft tissue	s_4	25.4[y]	16.5[x]	13.7[x]	.89
Combined bone/soft tissue	s^*	4.17	4.47	3.86	.43
Other inputs, g					
Blood P	Q_2	.040[z]	.073[y]	.13[x]	.0043
Bone P	Q_3	147	143	152	8.0
Soft tissue P	Q_4	12.8[y]	16.6[x]	16.4[x]	.88

[a]Symbols are according to Figure 1 and are defined in Table 1.
[b]*s = combined SRA of bone and soft tissues.
[x,y,z]Means within row and treatment category with different superscripts differ ($P < .05$).

positive on the moderately deficient (.42 g/d) and adequate (1.6 g P/d) diets. Retention on the H diet was almost four times greater ($P < .01$) than on the M diet.

Phosphorus flow from the digestive tract into the blood pool (F_{21}) increased with P intake ($P < .001$) (Table 4). A highly significant positive relationship also existed for P secretion from blood into gut lumen (F_{12}).

$$F_{21} = -.76 + 1.63F_{10} \quad [30]$$
(SE = .25 and .15 for intercept and slope respectively; n = 9; $r^2 = .94$)

$$F_{12} = -.39 + 1.02F_{10} \quad [31]$$
(SE = .21 and .12 for intercept and slope respectively; n = 9; $r^2 = .90$)

The P fluxes from blood to soft tissue (F_{42}) and from blood to bone (F_{32}) were lesser in goats fed Diet L ($P < .05$). Phosphorus recycling from tissue to the blood pool (F_{24}) was also lesser for animals fed low P levels. Bone resorption (F_{23}) was not influenced by P intake, despite the lesser value observed for P in Diet L. However, if the amounts of P taken up by bone, in relation to total P absorbed (F_{32}/F_{21}), are compared (12.6, 1.55, and .58 in L, M, and H diets, respectively), goats receiving Diet L transferred far greater amounts of P from blood to bone. Similarly, comparing P mobilization from bone to blood (F_{23}/F_{21}) (30.80, 2.82, and .80 for Diets L, M, and H, respectively), the values are greater ($P < .05$) in goats fed the L diet. Phosphorus flow from the soft tissue to blood pools in relation to total P absorbed was not significantly different between the treatments, although goats fed Diet L had a 32% lower value than those on Diet M (Table 4).

Discussion

Ruminants usually excrete negligible amounts of P in urine, but considerable variation may be observed between animals, with some animals excreting a quite

Table 4. Comparison of kinetic model outputs for the different levels of P intake. The values shown are mean values for each treatment

Item	Symbol[a]	Level of P intake			SEM
		Low	Medium	High	
Model outputs, g/d					
P from blood to gut	F_{12}	.16[z]	.90[y]	2.80[x]	.21
P from gut to blood	F_{21}	.05[z]	1.32[y]	4.40[x]	.24
P from blood to bone	F_{32}	.63[y]	2.04[xy]	2.57[x]	.46
P from blood to soft tissue	F_{42}	.93[y]	2.52[xy]	2.90[x]	.49
P from bone to blood	F_{23}	1.54	3.72	3.50	.83
P from soft tissue to blood	F_{24}	.14	.43	.37	.094

[a]Symbols are according to Figure 1 and are defined in Table 1.
[x,y,z]Means within row and treatment category with different superscripts differ ($P < .05$).

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high proportion of P intake in urine (Manston and Vaag, 1970). Urinary loss of P normally is not directly related to P intake, but it is associated with a greater efficiency of absorption and when plasma P concentration exceeds a renal threshold (between 2.0 to 3.0 m*M* of P) (Challa et al., 1989). Normally, the salivary glands clear plasma P at amounts below the threshold quite efficiently, and the majority of P is excreted in saliva. However, when the capacity of the salivary glands is saturated, renal excretion of P is increased. When P requirements for maintenance and growth are met, P is eliminated in urine (Challa et al., 1989). In the present experiment, dietary P levels were not sufficient to provide an excess of P, so P in urine was low for all treatments.

Endogenous fecal P has been reported to constitute more than 66% of total fecal P in cattle and sheep (Coates and Ternouth, 1992; Bortolussi et al., 1996). In the present study, this loss represented about 28, 37, and 44% of total P in feces in goats given Diets L, M, and H, respectively. This lower value could be due to the relatively lower P supply given to the animals, which does not far exceed the P requirement for maintenance.

Previous studies with sheep have shown that the endogenous P in feces is directly related to P intake (Braithwaite, 1985; Challa et al., 1989; Louvandini and Vitti, 1994). In this study, despite a need to retain P, especially from P-deficient diets, the growing goats were unable to avoid the inevitable endogenous loss through feces. At maintenance, no net gain or loss of P in bone and soft tissue is expected, so P flow from the digestive tract into the blood pool (F_{21}) should be equal to P flow into the gut (F_{12}) and P excretion through urine (F_{02}). The relationships between P intake and F_{21} and F_{12} are given by Eq. [29] and [30], and urine loss was assumed to be .004, which was the amount observed in goats fed Diets L and M. Therefore, maintenance intake was calculated to be .61 g of P/d, which is approximately equivalent to .055 g of P/($kg^{.75}$·d), assuming an average of 25 kg BW for goats in this study. The value of .61 is slightly lower than the previously reported value of .90 g of P/d (Kessler, 1991; Agricultural and Food Research Council, 1998 for 20-kg goats growing at a rate of 50 g/d). Animals fed Diet M, or 1.36 g of P/d, were in positive balance. However, this does not mean that this level is the adequate level of P for these goats, because P requirement for growth varies from 0.9 to 4.1 g/d for goats gaining 0 to 200 g/d (Agricultural and Food Research Council, 1998).

The improvement in efficiency of absorption obtained in this study, when Diet L was supplemented with P (Diets M and H), was also observed by Braithwaite (1985) for growing lambs fed with deficient and adequate diets. The reason for the low P absorption on Diet L may have been due to the low availability of P, mainly present in the organic form. Absorbed P is a mixture of dietary and digestive juices, mainly salivary P. The increased total P absorption was a result of increasing both dietary and endogenous P absorption (Braithwaite, 1984, 1985; Ternouth and Sevilla 1990). However, some authors report that the net absorption efficiency remains constant, irrespective of the dietary intake of P. According to Braithwaite (1984), the efficiency of P absorption depends on both P intake and P demands. The efficiency should be maximal in animals with high demand and receiving low to moderate P diets, and in such animals the active transport mechanism should be stimulated to the maximum. In the present study, the animals had a high demand because they were growing goats, and it was observed that the increase in efficiency was lower from the moderate to high levels than from the deficient to moderate levels. Probably, if greater levels of P were offered to the animals, the efficiency of P absorption would have decreased. It appears that a level between M and H should have been adequate for the goats in this experiment.

The high specific activity values in goats fed Diet L mainly reflected the lesser total P present in blood and soft tissue. In M and H, there was a dilution of radioactive P due to increased P concentration of the diets. The nonsignificant differences for bone resorption could be due to the high variation observed for this flux.

Animals fed at the highest P level were able to retain more P in soft tissue. The results show that bone P balance was negative for all treatments. However, in relation to the total P absorbed, goats fed Diet H had lesser rates of P resorption. Goats fed Diet L had greater amounts of P mobilized from tissue and bone to maintain P metabolism. The animals used in the present experiment were growing goats with a high P demand, which could explain the negative P balances observed. Fernandez (1995) observed that P bone accretion in pigs was not influenced by P intake, and bone resorption decreased from medium to high P intake levels.

In terms of regulation of P metabolism, we postulated that, with low P intakes insufficient to meet maintenance requirements, the input of P to the blood pool is maintained by an increased bone resorption of P and by P mobilization from soft tissues. Despite the low P intake leading to a negative P balance, an inevitable endogenous fecal loss of P occurs.

When P intake is increased to meet maintenance requirements (zero P balance), the rate of absorption is increased in direct relation to P supply, so endogenous secretion in the tract is increased. In the present study, the endogenous loss in feces did not show a significant increase between goats fed Diets L and M. This could be due to P intake just meeting P requirements for maintenance on Diet M. In goats fed an adequate P level (Diet H), the efficiency of absorption increased significantly, suggesting that P intake did not exceed the requirements and the P absorption mechanism was not saturated.

Implications

The model developed in this study could be used to investigate P metabolism in other ruminants as well as goats. The model showed that bone resorption, fecal and endogenous P excretion, and P absorption all play a part in P homeostasis in growing goats. Urinary P excretion

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did not significantly influence the control of P metabolism even in goats fed diets with P above requirement. At low P intakes, bone and tissue mobilization represented a vital process to maintain P levels in blood. A minimum endogenous loss of P from the animal was identified and needs to be supplied to avoid being in negative P balance. The kinetic P model developed here for goats could also be extrapolated to other ruminants.

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