PHOSPHORUS NUTRITION OF RUMINANTS

R.D.H. COHEN*

Summary

The role of phosphorus in ruminant nutrition is discussed with reference to factors influencing phosphorus turnover. Recent information leading to a re-estimation of P requirements is presented.

Particular reference is made to recent information on the hormonal control of P homeostasis and its relationship to the Ca-P interaction. Emphasis is placed on the interaction of P with other nutrients, particularly Ca, protein and energy and it is stressed that phosphorus cannot be considered as an isolated nutrient and panacea for ill-thrift and infertility.

I. INTRODUCTION

Most Australian soils are derived from parent material which is low in phosphorus. Soils of low phosphorus status support early maturing, grass dominant pastures with a short summer growth period and hence lengthy period of senescence. Phosphorus in these grasses may be as low as 0.02 g P/100 g OM during senescence (Robinson and Sageman 1967) and may only reach 0.15 g P/100 g OM during active growth. These are the pastures which are grazed by many of Australia's livestock.

Phosphorus has long been recognised as a major essential nutrient for ruminants and is probably the nutrient most frequently given as a supplement to ruminants in northern Australia. This discussion will therefore be most applicable to the beef cattle industry, because the undeveloped north supports more than 50% of Australia's beef cattle but only about 10% of Australia's sheep (Australian Meat Board Annual Report 1977).

II. PHOSPHORUS TURNOVER

Up-to-date findings in the phosphorus metabolism of ruminants have been reviewed recently (Wadsworth and Cohen 1976) but it is appropriate to summarise some of that information here.

(a) Salivary P

The daily turn-over of P in ruminant saliva is similar to or greater than daily P intake. For example, total daily P secretion from both parotid glands of sheep exceeds P intake by a factor of 7.2-1.3 for intakes from 0.4 to 4.0 g P/d (Tomas, Moir and Somers 1967). Similarly it may be calculated from the data of Poutiainen (1971) that a mean of 34 g P/d, or 77% of the P intake, entered the rumen of cows via the saliva.

The phosphorus in saliva is mostly inorganic phosphate (Pi) and its concentration exceeds that of serum by a factor of 5-19 for sheep and 4.5 for cattle; comparable levels of salivary P in cattle being lower and the number of estimates fewer than in sheep. Although few direct estimates of

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daily P secretion in saliva have been made, some generalisations can be stated. First, for any given diet, P output in saliva is dependant on P intake. Second, salivary P concentration is directly related to plasma P concentration and varies inversely with saliva flow rate which tends to keep the daily output of salivary P constant. Third, in the absence of significant fluxes of P across the rumen wall, the only sources of P in the rumen are diet and saliva and salivary P is the most important.

It has been reported (Clark 1953) that, even during clinical hypophosphorosis, concentrations of P in rumen fluid do not fall below 20 mg P/100 ml. However, concentrations of rumen Pi between 15 and 18.3 mg P/100 ml rumen fluid have been recorded recently (Cohen 1978) for heifers with low intakes of dietary P (see table 2). There are few reports on the requirements for P of rumen micro-organisms, but these levels are in excess of the minimum requirement of 6 mg P/100 ml for Bacteroides succinogens (Bryant, Robinson and Chu 1959). This is consistent with the large number of findings that P supplementation of low P diets does not affect digestion in the ruminant (See Cohen 1975).

(b) P absorption

Absorption of P from the rumen and omasum is negligible and it is not known if P is absorbed from the abomasum. The main site of P absorption is the small intestine and its transport consists of both active and passive diffusion. Passive diffusion predominates at high luminal concentrations of P. Thus in P deficiency, P absorption will have a higher energy cost. Active diffusion of P across the small intestinal wall is independent of Ca transport and by a different path. There is only a small absorption of P from the large intestine.

(c) P excretion

Ruminant animals usually excrete negligible amounts of P in urine. This major difference between ruminants and non-ruminants is due to a greater ability of the ruminant kidney to reabsorb phosphate.

In the non-lactating ruminant, the faeces are the major vehicle for the excretion of P. Organic P excretion remains relatively constant and independent of the amount of P ingested, whereas faecal Pi varies directly with P intake. On the other hand the P content of milk is relatively constant at 0.95 g P/kg milk (Agricultural Research Council 1965), independent of P intake and is thus a major source of loss of endogenous P.

(d) Accretion and resorption of P in bone

The rate of bone deposition is adjusted by young animals in response to changes in nutrition and by adult females also in response to the onset of lactation. In the normal, well nourished animal the accretion and resorption of P in bone is in dynamic equilibrium. During P deficiency the young animal appears to meet its changing mineral needs by changing the rate of bone resorption and also by changing the rate of bone accretion, The mature animal, however, appears to lose its ability to adjust its rate of bone accretion. Thus there is a higher rate of P turnover in the mature animal when it eats a low P diet.

About 40% of bone mineral can be mobilised to meet the demands of severe P depletion (Benzie et al. 1959) but net retention of P in bone (accretion minus resorption) is only a small fraction of P intake. It
has been estimated that as little as 8.7% of fertilizer P applied to pasture is recovered in animal products other than milk (Williams 1974).

III. PHOSPHORUS REQUIREMENTS

Phosphorus requirements for livestock are calculated as the sum of the P retained by the animal, P lost in animal products (conceptus and milk) and endogenous P losses (faeces and urine). This value is adjusted for the availability of dietary P. Phosphorus retention during growth is estimated from data on the P content of animals of different weights and stages of pregnancy.

Since the Agricultural Research Council published estimates of P requirements of livestock (A.R.C. 1965), more data have become available. In making their estimates, the Agricultural Research Council assumed that the P content of liveweight gain in sheep was constant, that endogenous losses of P from cattle were proportional to live weight and that the availability of P was a simple function of live weight for cattle and age for sheep. These assumptions are now unacceptable (Langlands and Sutherland 1969; Playne 1976) and the requirements have been recalculated (Wadsworth and Cohen 1976). Some recalculated estimates of P requirements for maintenance and growth are presented in Table 1 and compared with estimates made by the Agricultural Research Council (1965) and the National Academy of Sciences (1970).

Table 1. Phosphorus requirements of sheep and cattle

<table>
<thead>
<tr>
<th>Liveweight (kg)</th>
<th>Age (Mo)</th>
<th>Growth rate (kg/d)</th>
<th>Dietary P requirement (g/d)</th>
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<tbody>
<tr>
<td></td>
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<td>ARC¹</td>
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SHEEP

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<tbody>
<tr>
<td>5</td>
<td>0.5</td>
<td>0.2</td>
<td>1.4</td>
<td>nd</td>
<td>1.3</td>
</tr>
<tr>
<td>45</td>
<td>12</td>
<td>0.05</td>
<td>2.8</td>
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</tr>
<tr>
<td>48</td>
<td>0.05</td>
<td>3.7</td>
<td></td>
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<td>2.7</td>
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<tr>
<td>Mature</td>
<td>0</td>
<td>3.3</td>
<td></td>
<td></td>
<td>2.4</td>
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CATTLE

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<tr>
<td>50</td>
<td>1</td>
<td>0.5</td>
<td>6.2</td>
<td>nd</td>
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<tr>
<td>100</td>
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<td>0.5</td>
<td>7.3</td>
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<td>9.8</td>
<td>10.0</td>
<td>13.1</td>
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<tr>
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<td>8.0</td>
<td>10.1</td>
</tr>
<tr>
<td>300</td>
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<td>0.5</td>
<td>14.6</td>
<td>14.0</td>
<td>15.4</td>
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<tr>
<td>400</td>
<td>na</td>
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<td>10.0</td>
<td>13.5</td>
</tr>
<tr>
<td>400</td>
<td>na</td>
<td>0.5</td>
<td>23.7</td>
<td>17.0</td>
<td>17.8</td>
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COWS (8 months pregnant)

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<tbody>
<tr>
<td>300</td>
<td>na</td>
<td>0</td>
<td>19.3</td>
<td>9.0</td>
<td>16.8a</td>
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<tr>
<td>400</td>
<td>na</td>
<td>0</td>
<td>29.8</td>
<td>10.0</td>
<td>20.2a</td>
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COWS LACTATING

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<tr>
<td>5 kg milk/d</td>
<td>350</td>
<td>na</td>
<td>23.0</td>
<td>20.0*</td>
<td>19.3b</td>
</tr>
<tr>
<td>500</td>
<td>na</td>
<td>0</td>
<td>60.0</td>
<td>50.0§</td>
<td>41.9b</td>
</tr>
</tbody>
</table>

1. Agricultural Research Council (1965)
   a. Assumes a P requirement of 4.3 g P/d for growth of foetus and placenta of a cow producing a 45 kg calf (A.R.C. 1965)
   b. Assumes an endogenous P loss of 0.95 g P/kg milk (A.R.C. 1965)
   nd Not determined
   † No age distinction made
   na Not applicable
   * Milk yield not stated
It can be seen that the estimated requirements for sheep over 12 months of age are considerably less than those estimated by the A.R.C. and that estimated requirements for cattle up to 300 kg live weight are greater than those estimated by the A.R.C. or N.A.S. but estimates for growing, pregnant and lactating cows over 300 kg live weight are intermediate to A.R.C. and N.A.S. estimates.

IV. INTERACTION OF OTHER NUTRIENTS WITH P

The latest information on the interaction of other nutrients with P has been fully discussed in two recent reviews (Cohen 1975; Wadsworth and Cohen 1976) and only a brief summary will be included here.

(a) Calcium

A significant recent advance of relevance to the Ca-P interaction has been the demonstration that parathyroid hormone (PTH) increases the P concentration in saliva (Tomas 1974). PTH is the major regulator of Ca metabolism and maintains stable plasma Ca levels by stimulating bone resorption (Kronfield, Hager and Ramberg 1976). Its secretion is inversely related to Ca levels in the blood perfusing the parathyroid gland and is unaffected by changes in plasma P levels (Sherwood et al. 1968). Thus a major regulatory influence on P turnover is dependent largely on the Ca status of the animal.

The action of PTH on bone resorption is dependent on the presence of the \( 1,25 \text{dihydroxy-metabolite of vitamin D}_3 \) (Omdahl and DeLuca 1973), and is inhibited by the hormone, calcitonin, which is secreted by the thyroid gland (Rasmussen and Tenhouse 1967). Oestrogens may also be involved. Absorption of P from the intestine is enhanced by \( 1,25 \text{-OH}_2 \text{D}_3 \) which is produced by sequential hydroxylation of D3 in the liver and kidney. This hydroxylation is stimulated by low serum (and intracellular) Pi levels and depressed by high serum Ca levels (De Luca and Schones 1976).

Thus when Ca intake is high, there is a depression of production of \( 1,25 \text{-OH}_2 \text{D}_3 \) with a consequent reduction in absorption of P; a reduction in PTH secretion with a consequent reduction in salivary P concentration and mobilisation of P from bone. Add to this a low P intake and clinical symptoms of hypophosphorosis such as low blood Pi levels may develop and growth responses to P supplements may occur to a level no longer dictated by dietary P but by dietary digestible energy and protein.

Conversely, when dietary Ca is low there is an increase in production of \( 1,25 \text{-OH}_2 \text{D}_3 \), absorption of P, secretion of PTH, salivary P concentration, and mobilisation of P from bone resorption and hence an appreciable increase in P available for soft tissue growth. Under these conditions it is not surprising to find that a growth response to supplements of P does not occur. For example Theiler, duToit and Malan (1937a) reported that heifers which consumed low P diets but which received adequate or excessive levels of Ca showed depressed food intake, low rates of liveweight gain and marked clinical signs of hypophosphorosis whereas heifers which consumed a ration low in Ca and P, but adequate in other nutrients, ate well, grew normally and showed no clinical signs of hypophosphorosis for 18 months. At autopsy however, animals on a low Ca and P diet showed severe rickets and demineralisation of bone and low blood P values. This experiment was repeated with identical results using steers (Theiler, duToit and Malan 1937a; Otto 1938) and pigs (Theiler, duToit
and Malan 1937b). Similar results have also been found with sheep
(Field, Suttle and Nisbet 1975).

In fact visual evidence of bone disorders (Stiff gait, "bent leg")
may well indicate low intakes of both Ca and P (Theiler, duToit
and Malan 1937a) and contravene any production loss until ultimately the animal
breaks a leg and dies. The desirable restoration of bone health in these
circumstances may therefore only occur when a Ca-P supplement is given.
It is worth noting that the literature abounds with examples of responses
to bone meal and Ca\(_x\)(PO\(_4\))\(_y\) salts and attributes the responses to the P
alone. In those cases when production responses have been recorded, the
response may well be associated with more extensive grazing as a result of
improved bone health rather than any direct improvement in the utiliza-
tion of P for growth.

(b) Energy and protein

It is apparent that P deficiency restricts energy intake rather than
biochemical energy transformations (Wadsworth and Cohen 1976) so that a
response to P supplements can only be anticipated up to a level where
other nutrients, for example protein, restrict energy intake. Since the
phosphorus and protein contents of pasture are usually correlated (see
Cohen 1975) it follows that supplementation of low protein pastures with
P alone is likely to provide little stimulus to animal production unless
there is a strong Ca-P imbalance. Conversely responses from feeding low
P-high protein supplements (for example oil-seed meals) or the use of low
P tolerant legumes (for example Stylosanthes humilis; Aeschynomene
falcata) may only be maximised if supplements of P are also provided.

Despite normal P intakes, bone mineralisation may be restricted by
inadequate intakes of energy (Benzie et al. 1960) and protein (Stewart
1965; Sykes, Nisbet and Field 1973; Siebert et al. 1975) through a restr-
iction of energy and protein which is available for the formation of
organic matrix for normal mineralisation.

(c) Other minerals

Several other dietary elements are known to influence P absorption
or utilization. These are Fe, Be, Al, Cu, Mn, Mo, Mg, Zn and S and their
effects have been discussed by Cohen (1975) and Wadsworth and Cohen
(1976) and do not warrant further discussion here.

v. EFFECTS OF ADDITIONAL DIETARY PHOSPHORUS

(a) Species of animal

The provision of supplemental phosphorus for grazing cattle has pro-

bably been the most widely accepted management practice in Australia's
beef cattle industry. This stems from an acceptance, at all levels of
the industry, of research results from South Africa and U.S.A. Through-
out the industry there has been an acceptance that phosphorus supplements
will increase growth rates and fertility, regardless of the adequacy or
otherwise of other nutrients (particularly protein, digestible energy and
calcium) and despite the small or non existent responses reported in the
published Australian research (See Cohen 1975).

'The acceptance of the role of phosphorus supplements in increasing
animal production in the dairy industry is better founded because of the
general adequacy of dairy cattle diets in respect to other nutrients. Further, P deficiency in dairy cows, particularly high yielding cows, is generally expressed as an acute metabolic disease because of the sudden large increase in P requirements for lactation (table 1). Phosphorus supplements have not been used extensively in the sheep industry, probably because there have been fewer overseas reports which have indicated a response. This lower sensitivity of sheep compared with cattle to P supplements may be due to one or more of the following explanations (See Cohen 1975): (i) the ability of sheep to select a diet containing more P than cattle when the P content of forage on offer is low; (ii) sheep need to consume 1.5 to 2 times as much feed per unit body weight and could therefore be expected to tolerate a lower concentration of phosphorus in the feed or (iii) when P intake is low sheep may retain P more efficiently than cattle. For these reasons the remainder of this discussion will concentrate on the provision of additional phosphorus for beef cattle.

The review by Cohen (1975) concluded that in Australia the provision of additional dietary P is, at best, likely to be accompanied by small increases in growth and fertility of grazing beef cattle which could be attributed to the additional dietary P alone. I would like to briefly illustrate this point with some further examples.

(b) Direct supplementation of beef cattle

The data of Cohen (1976) indicated no effect of a P supplement on liveweight change of cows and calves or fertility of cows which grazed a low P pasture. However the number of cows used was small and a further experiment was commenced in 1975 with 45 Hereford cows in each of two groups: control and supplemented (Want and Mears - unpublished data). The supplement was a commercial mineral mix advertised principally as a phosphorus supplement with the following dietary components: P (9%); Ca (16%); NaCl (10%); Cu (0.03%); Co (0.003%); I (0.003%); Urea (8%); Feed grade biuret (2%); Crude protein (2%, source unspecified). The cows were given the supplement at the rate of 100 g/d (9 g P/d) and between calving and weaning 120 g/d (10.8 g P/d). The results after three years indicated no significant effect of the supplement on cow liveweight or fertility (66.4% v 67.3% for control and supplemented cows respectively) but a small increase in calf weaning weights following supplementation of the cows (84 v 95 kg respectively). This weight advantage may however have been due to a stimulation of milk production from the nitrogen and crude protein in the supplement (Cohen 1976).

(c) Provision of additional P via pasture

A major advance in recent years in the P nutrition of ruminants has been the development of techniques for the estimation of P intake of grazing animals (Cohen 1974; Langlands 1976; Little, McLean and Winter 1977). This development allows animal production responses to superphosphate to be partitioned between increases in dietary protein, digestible organic matter, P and other nutrients. This has been done in an experiment in which Hereford heifers grazed pastures which were unfertilized or fertilized with superphosphate (Cohen 1978). The experiment was conducted for five years with fertilizer applications of 500 kg/ha in the first year and 250 kg/ha in the subsequent four years. Hereford heifers entered the experiment annually as 6-7 month old weaners (153 kg mean live weight) and were turned off 12 months later following the detection of oestrus in the final six weeks using vasectomised bulls wearing chin-
Table 2. The influence of pasture dressings of superphosphate alone or with oversown legumes on liveweight change, incidence of oestrus, phosphorus consumed and stored and dietary digestibility, nitrogen content and digestible organic matter intake of grazing Hereford heifers.

<table>
<thead>
<tr>
<th></th>
<th>Native pasture</th>
<th>Native pasture dressed with superphosphate</th>
<th>Native pasture Superphosphate and Legumes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean liv. change (kg/d)†</td>
<td>0.04c</td>
<td>0.11b</td>
<td>0.37a</td>
</tr>
<tr>
<td>Mean oestrus (%)†</td>
<td>4.5c</td>
<td>20.7b</td>
<td>90.5a</td>
</tr>
<tr>
<td>Mean diet P (g/100 g OM)†</td>
<td>0.15b</td>
<td>0.36a</td>
<td>0.38a</td>
</tr>
<tr>
<td>Mean P intake (g/d)†</td>
<td>5.0c</td>
<td>15.3b</td>
<td>22.6a</td>
</tr>
<tr>
<td>Mean carcase P Bone (g)††</td>
<td>1033c</td>
<td>1156b</td>
<td>1681a</td>
</tr>
<tr>
<td>Total carcase P (g)††</td>
<td>1177c</td>
<td>157b</td>
<td>214a</td>
</tr>
<tr>
<td>Mean diet digestibility (% OM)†</td>
<td>42.4c</td>
<td>45.7b</td>
<td>52.6a</td>
</tr>
<tr>
<td>Mean digestible organic matter intake (kg/d)†</td>
<td>1.38c</td>
<td>1.90b</td>
<td>3.10a</td>
</tr>
</tbody>
</table>

† Data from Cohen (1978) †† Data from Langlands and Cohen (1978)
Unlike superscripts within rows indicate differences significant (P<0.05)

It is obvious from table 2 that there was only a small, although significant (P<0.05), increase in the growth and incidence of oestrus of heifers which grazed fertilized compared to unfertilized pasture, despite a very substantial increase in P intake and dietary content; for example P intake was sufficient for liveweight gains of 0.5 kg/d. This small increase in animal production was also associated with small but significant increases in diet digestibility and digestible organic matter intake (DOMI) and the response was therefore probably associated with secondary rather than primary effects of the fertilizer (See Rees and Minson 1976). The inclusion of legumes in the pasture had little effect on dietary P content but a substantial effect on dietary digestibility, nitrogen content, DOMI, liveweight change and oestrus. Furthermore, despite an increase in P intake of 10.3 g/d (206%) following the application of superphosphate, there was only an increase of 0.37 g P/d stored in the body (11.6%) compared with an increase of 1.96 g P/d (61%) for heifers grazing fertilized pasture which included legumes. This represents recoveries in animal products of 3.6 and 11.2% respectively of the P consumed and highlights the interaction of protein, energy and phosphorus nutrition.

In conclusion, it is stressed that the consideration of phosphorus in ruminant nutrition as a single isolated nutrient and panacea for ill-thrift and infertility is no longer acceptable.

VI. REFERENCES


