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SYMPOSIUM ON 'MINERAL NUTRITION OF FARM LIVESTOCK'

Control of mineral absorption in ruminants

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Studies in recent years have suggested that the absorption of many of the major mineral elements from the gut may be under greater regulatory control than has hitherto been suspected. In this paper we consider the evidence for such control in ruminants with particular reference to calcium and phosphorus absorption.

Ca absorption

There is now clear evidence that both sheep and cattle absorb Ca from their gut according to need and that they can alter the efficiency of absorption to meet a change in requirement. For example, Braithwaite & Riazuddin (1971) have shown that young sheep with a high Ca requirement absorb Ca at a higher rate and with greater efficiency than mature animals with a low requirement. An increase in absorption and an increased efficiency of absorption also occurs in mature sheep when their requirement for Ca is increased through pregnancy or lactation (Braithwaite *et al.* 1969; 1970) or after a period of Ca deficiency (Braithwaite, 1974).

Studies in cattle have given similar results. Thus the efficiency of absorption of Ca in the small intestine of the dairy cow has been shown to rise in response to a reduction in dietary Ca intake and to the onset of lactation (Ramberg *et al.* 1970; van't Klooster, 1976). The amount of Ca absorbed has also been shown to be directly related to milk production (van't Klooster, 1976) though in early lactation when the demand for Ca is greatest the increase in absorption falls short of requirement (Symonds *et al.* 1966; van't Klooster, 1976), the deficit being met by increased bone resorption (Braithwaite *et al.* 1969).

The mechanism by which Ca absorption is adjusted in response to requirement has also received much attention and Fig. 1 summarizes the main features of the

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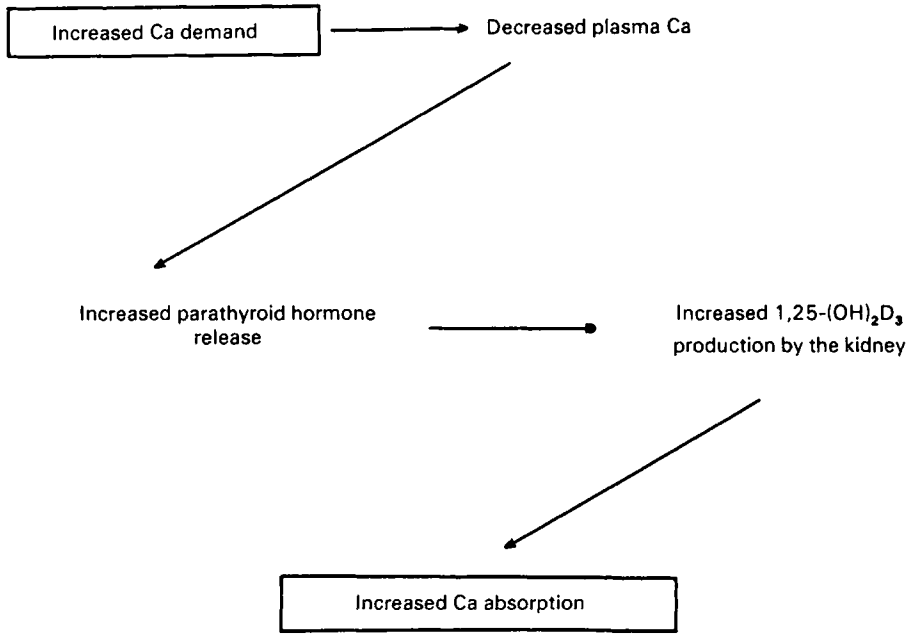

Fig. 1. Schematic representation of the role of 1,25 dihydroxycholecalciferol (1,25-$(OH)_2D_3$) in the control of Ca absorption.

system most generally accepted. In this a fall in plasma Ca concentration resulting from an increase in demand leads in turn to an increase in parathyroid hormone release. This then stimulates the increased production by the kidney of 1,25 dihydroxycholecalciferol (1,25 $(OH)_2D_3$) which acts on the gut to increase the production of Ca-binding protein and so accelerates Ca absorption (De Luca, 1979). In a reverse manner, an increase in plasma Ca concentration causes a suppression of parathyroid hormone release, a reduction in 1,25 $(OH)_2D_3$ production and reduced Ca absorption.

Although all aspects of this system have not as yet been fully examined in ruminants it does appear that the same mechanism operates in that an increase in circulatory 1,25 $(OH)_2D_3$ level has been found to precede the increase in Ca absorption that occurs in cattle soon after parturition (Horst *et al.* 1978). In addition, the administration of synthetic analogues of this hormone will reduce the fall in plasma Ca concentration that usually occurs at this time (Wittwer & Ford, 1980). In sheep, injection of such analogues in early lactation stimulates Ca absorption and thereby reduces the loss of Ca from bone that otherwise occurs (Braithwaite, 1978).

P absorption

Sheep that are fed on roughage diets usually excrete little P in their urine (Scott, 1969, 1972; Stacy, 1969; Clark *et al.* 1973; Tomas, 1975; Towns *et al.* 1978). Control of P balance must therefore be achieved within the gut either through

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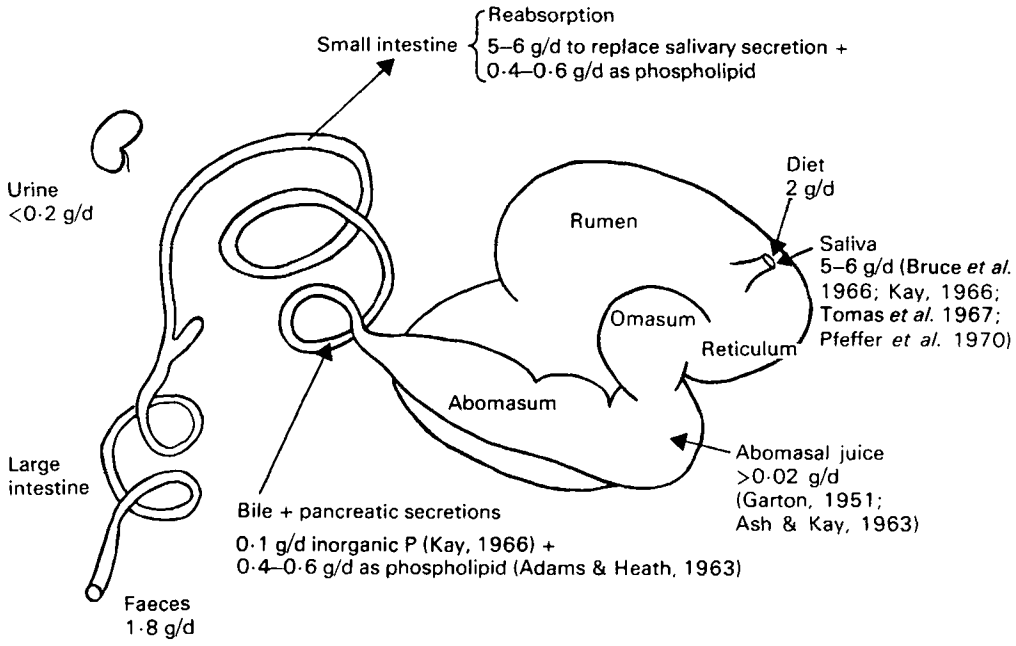

Fig. 2. Schematic representation of movement of P through the gut of sheep fed on a roughage diet.

control of absorption or secretion or both. Fig. 2 summarizes the major components contributing to the movement of P in the gut of the sheep. As can be seen, saliva is the main contributor. Little or no net absorption of P appears to occur from either the forestomach (Scarisbrick & Ewer, 1951; Sperber & Hyden, 1952; Parthasarathy *et al.* 1952; Engelhardt & Hauffe, 1975) or the large intestine (Bruce *et al.* 1966; Pfeffer *et al.* 1970) and it is generally accepted that the upper small intestine is the major absorptive site (Kay & Pfeffer, 1970). Using sheep fitted with re-entrant cannulas, several workers have shown that the net secretion of P before the pylorus (salivary) is closely matched by net absorption in the small intestine (Bruce *et al.* 1966; Kay & Pfeffer, 1970; Pfeffer *et al.* 1970). This ability to balance absorption against secretion has been shown to be unaffected by quite wide variations in dietary Ca:P value (Pfeffer, 1968).

Until recently, secretion of P in saliva has usually been viewed in the context of its role as a buffer against the volatile fatty acids produced in the rumen. Studies by Australian workers (Tomas, 1974*a*,*b*; Tomas & Somers, 1974) have, however, added an extra dimension in that they have suggested that the salivary glands apart from their role as a source of buffer may, in addition, by controlling the amount of P secreted into the gut, play an important role in P homoeostasis. Evidence for this function was first provided by studies in sheep in which both parotid salivary ducts were ligated, a procedure which led to a small increase in urinary P excretion and a proportional reduction in faecal P excretion (Tomas &

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Somers, 1974). Similar changes in the pathway of P excretion were also seen in sheep in which part of the parotid salivary flow was collected and returned directly to the circulation (Tomas, 1974*b*).

In separate studies Clark *et al.* (1973) infused a hypertonic solution of KH_2PO_4 intravenously over a 2 h period into sheep fed on a roughage diet. An average of 3·38 g P was given via this route and most was recovered in the faeces during the next 4–5 d. By adding a small amount of Cr-EDTA as a marker to the rumen at the outset of the infusion, they showed that most of the increase in faecal P excretion occurred at or after the appearance of this marker, which suggests that the saliva was the main pathway by which this extra P entered the gut.

To what extent the salivary glands determine the relative importance of the kidney or gut as pathways for phosphorus excretion in response to variations in dietary phosphorus intake is, however, not clear from these studies and it is this aspect that we have recently been examining.

In our initial experiments we used two groups of three mature crossbred sheep fed on either pelleted hay or grass (800 g/d) from a continuous belt feeder. In addition, to provide variation in P intake they were given either 3 or 6 g P/d as Na_2HPO_4 by continuous infusion into the rumen.

When pelleted hay was fed, very little P was excreted in the urine in the control period and no increase was seen during infusion; the faeces being the major pathway for excretion (Table 1). The faeces were also the major pathway for P excretion when the pelleted grass was fed, though here urine phosphorus excretion did show a small increase during infusion, the response being particularly marked in one of the three sheep.

The sheep fed on the pelleted grass diet were fitted with both rumen and duodenal cannulas and by continuously infusing polyethylene glycol (10 g/d) into the rumen and taking digesta samples at the duodenum, we were able to estimate the total flow of P entering the small intestine. Flow of P into the duodenum increased during P infusion by amounts equal to the infusion rate (Fig. 3), all of the extra P being accounted for as inorganic P in the liquid phase of the digesta. The added P thus entered the small intestine in a potentially highly available form and yet little was absorbed. The amount of P estimated to have been added to the gut

Table 1. *The effects of intra-ruminal infusions of Na_2HPO_4 on the pathway of phosphorus excretion in sheep fed either pelleted hay or grass diets*

(Values are means with their standard deviations)

	Intake (g/d)			Excretion (g/d)			
Diet	Diet	Infusion	Total	Urine	Faeces	Total	Faeces: Total (%)
Hay	1·24	—	1·24	0·08±0·01	1·18±0·03	1·26±0·03	93·7
	1·24	3·00	4·24	0·09±0·01	3·87±0·16	3·96±0·17	97·7
Grass	2·05	—	2·05	0·01±0·01	1·77±0·24	1·78±0·23	99·4
	2·05	3·00	5·05	0·22±0·42	4·38±0·54	4·67±0·16	93·8
	2·05	6·00	8·05	0·49±0·59	7·00±0·83	7·49±0·36	93·5

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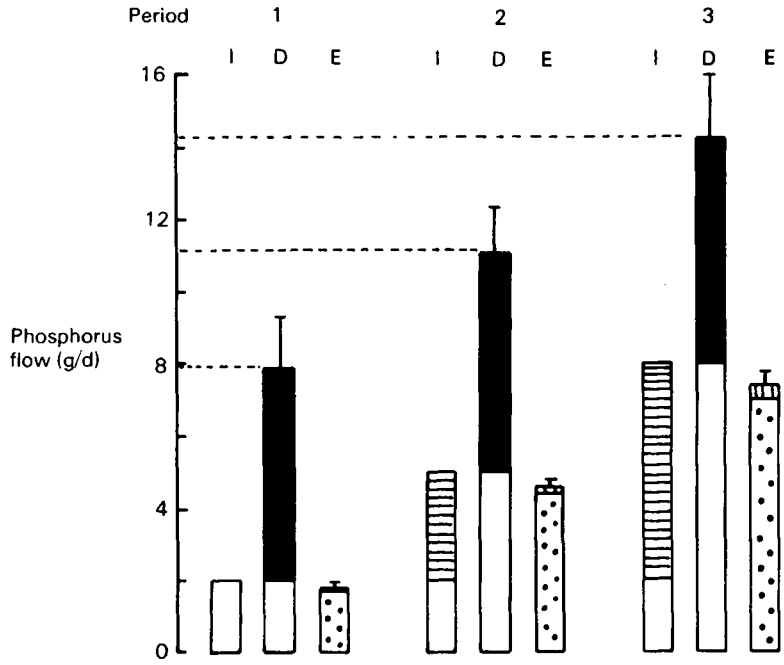

Fig. 3. The effects of intraruminal phosphate infusions on the flow of P through the gut of sheep fed on a pelleted grass diet (800 g/d). I, intake (▤ infusion, □ diet); D, duodenal (■ salivary); E, excretion (▥ urine, ⊡ faeces). Values are means for three sheep with their standard deviations shown as vertical bars.

via the saliva (duodenal–(dietary+infusion)) showed only a marginal increase which suggests that control of absorption was the major factor determining the amount of P excreted in the faeces under these conditions. These observations have since been repeated using a further five sheep and similar results were obtained, in that again, little or no increase in urinary P excretion occurred during intraruminal phosphate infusion when pelleted hay was fed, while when pelleted grass was fed some sheep showed an increase in urine P excretion and some did not.

Similar variation in renal response to phosphate loading has been reported by other workers. Thus, for example, Towns *et al.* (1978) infused 1·5 g P/d intravenously into four sheep for an 11 d period and in three of the sheep they recovered all of this extra P in the faeces while in the fourth sheep about a third was recovered in the urine and the rest in the faeces.

In order to account for the response seen in such individuals we have been looking at the renal threshold for P excretion in sheep fed on either pelleted hay or grass. Mature crossbred ewes were used and they were fitted with bladder catheters and were given intravenous infusions of a hypertonic phosphate (Na_2HPO_4) solution so as to increase plasma inorganic P concentration. Inulin was used as a marker to measure glomerular filtration rate (GFR). The relationship between urinary P excretion and plasma inorganic P concentration is shown in

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Fig. 4. When the grass diet was fed, little P was excreted in the urine until the concentration of inorganic P in the plasma was raised above about 2·0 mmol/l while for the hay diet the threshold position appeared, if anything, to be a little higher.

When these results were considered in relation to our earlier observations the reason for the variation between individuals in their renal response to intra-ruminal phosphate infusion became clearer. Thus, for example, Fig. 5 shows the mean daily excretion of P in the urine in relation to plasma inorganic P concentration for the three sheep fed on a pelleted grass diet (Table 1), two of which showed only a small increase in urine P excretion in response to intra-ruminal phosphate loading and one in which the renal response was much more marked. As can be seen in those sheep in which P absorption was no greater than salivary P secretion, plasma inorganic P concentration was well below the renal threshold for P excretion in the control period and remained below this value during infusion and little P was excreted in the urine. In the one sheep (2311), which during phosphate infusion regularly absorbed rather more P than was required to replace its salivary loss, plasma inorganic P concentration was already high in the control period and rose during infusion to exceed the renal threshold value and as a result more P was excreted in the urine. In sheep fed on the pelleted hay, plasma inorganic P concentration during control periods was always well below the renal threshold value seen in Fig. 5 and remained below this level during infusion and no increase in urine P excretion occurred. It would thus appear from our observations that when sheep are fed on roughage diets, they can control intestinal P absorption very finely to the amount needed to replace that secreted in the saliva and in so doing they favour the gut rather than the kidney as a pathway for excretion of excess dietary P. Some individuals do, however, appear to be less adept at exercising this control than others though why this is so is at present unknown.

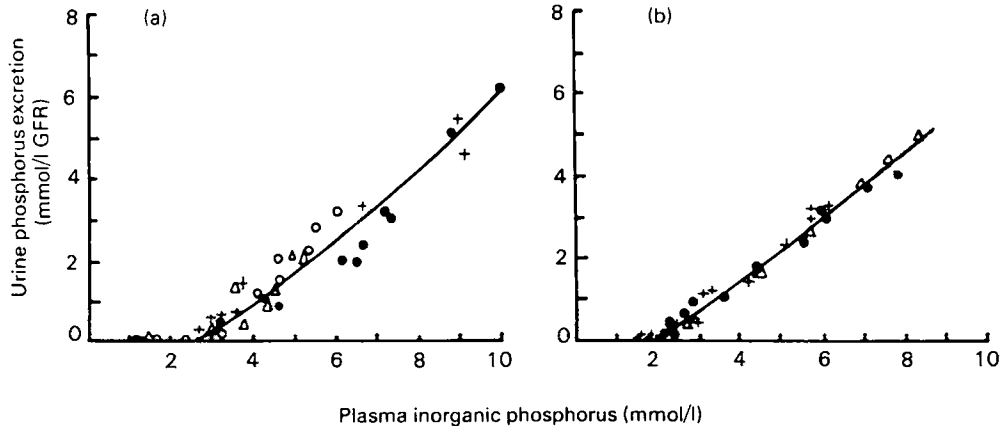

Fig. 4. The relationship between the concentration of inorganic P in the plasma and urine P excretion seen in sheep fed on either (a) pelleted hay or (b) grass diets.

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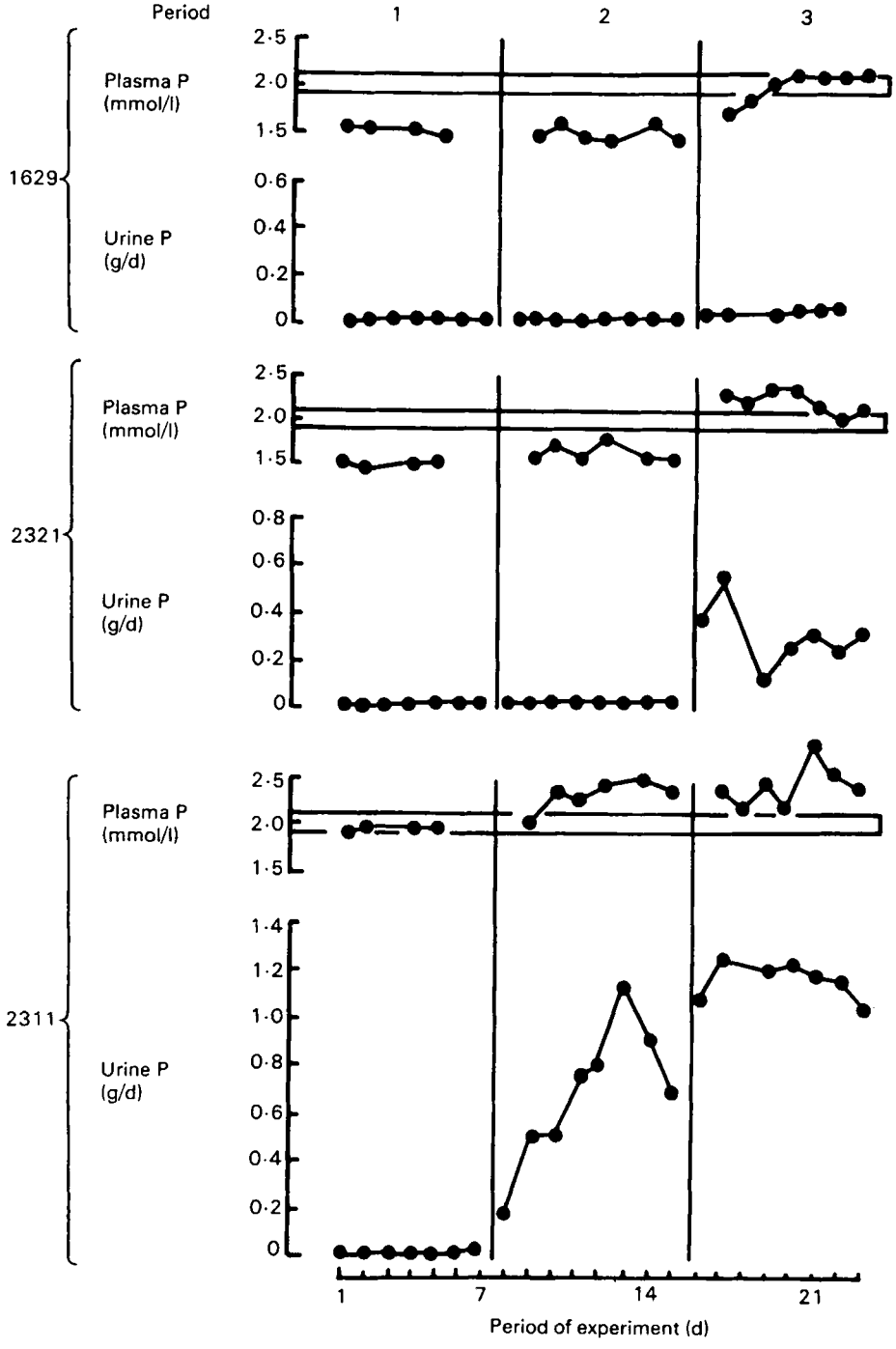

Fig. 5. The relationship between the concentration of inorganic P in the plasma and urine P excretion in sheep fed on a pelleted grass diet. The horizontal bar illustrates the renal threshold for P excretion. The basal diet supplied 2·05 g P/d and in periods 2 and 3 this was supplemented by giving either 3 or 6 g P/d as Na_2HPO_4 into the rumen.

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To see how frequently such individuals occur we sampled 100 ewes (fifty-nine lactating, forty-one non-lactating) grazing a predominantly ryegrass sward. In eighty-six ewes the mean concentration of inorganic P in the plasma was 1·67±0·30 mmol/l, i.e. a value below the renal threshold seen earlier and consistent with this the concentration of P in their urine was low (18·6±14·3 mg/l). In the other fourteen sheep, however, plasma inorganic P concentration averaged 2·54±0·21 mmol/l and the concentration of P in their urine was higher with some individuals giving values in excess of 200 mg/l.

Manston & Vagg (1970) have reported similar results from cattle. They collected urine samples from 137 cows both under grazing and indoor conditions and found that about 90% regularly excreted very little P in their urine despite the fact that indoors, dietary P intake was greatly in excess of requirement. Symonds & Manston (1974) subsequently observed that little P was excreted in the urine of cattle (diet not defined) until the concentration of inorganic P in the plasma was raised to over 2·25 mmol/l, a value well above the mean concentration of 1·75±0·29 mmol/l seen in 2400 dairy cows surveyed by Payne *et al.* (1970).

At this point it is perhaps pertinent to consider what advantage a ruminant may derive by maintaining the renal threshold for P excretion at such a high level compared to that seen in monogastric species. Poor quality roughage diets, apart from their low digestibility, also tend to contain little P, therefore the ratio between the amount of P required for saliva production and dietary intake is wide. In a 40 kg sheep only about 200 mg inorganic P is present in the circulation at any time whereas the saliva may contain in excess of 5–6 g/d (Fig. 2). With episodic feeding and a site of absorption far removed from the site of secretion, these two processes will tend to be out of phase and marked diurnal variation in the concentration of inorganic P in the plasma may occur. If the renal threshold for P excretion in ruminants were as low as in monogastric species, then at times when the concentration of inorganic P in the plasma rises in response to reabsorption, P would be excreted in the urine. This P would not, however, in any real sense be surplus to requirement and its loss would have to be met from a diet low in available P. In such conditions there is clearly an advantage to the ruminant in maintaining the renal threshold for P excretion at a high level, thereby allowing wider diurnal variation in plasma inorganic P concentration before any renal loss occurs, thus ensuring that P for saliva production continues to be available despite a low dietary P intake.

Such an argument may also have a bearing on the situation seen when concentrate diets are fed in that whereas sheep and cattle fed on roughage diets usually excrete little P in their urine, those fed on concentrate diets usually excrete large amounts (Hyldgaard-Jensen & Simesen, 1966; Hyldgaard-Jensen *et al.* 1966; Scott, 1972). Although attempts have been made to account for the phosphaturia seen when concentrate diets are fed, there is at present no complete explanation. Reed *et al.* (1965) observed a progressive fall in blood and urine pH in cattle fed on increasing amounts of a concentrate diet and they suggested that the phosphaturia might be directly related to this shift in acid–base status, P being excreted in the

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urine to act as a vehicle for acid excretion. However, in other studies, correction of the acid–base disturbance by infusion of $NaHCO_3$ into the rumen failed to produce a reduction in urinary phosphorus excretion (Scott, 1972) which suggests that something other than differences in acid–base status must be responsible for the phosphaturia seen when concentrate diets are fed.

Concentrate diets, especially those which include fish meal, contain much larger quantities of P than roughage diets, to the point where intake may equal or exceed the amount secreted in the saliva. Under these conditions the need to control P absorption is clearly less critical and as a result a different level of control may operate. This situation is somewhat similar to that seen in monogastric species which secrete relatively little P in their saliva and thus are not faced with the same reabsorption problem as ruminants. Under these conditions, increasing dietary P intake leads to increased absorption and increased urinary P excretion.

Despite the importance of this subject there have to date been very few studies aimed at defining the mechanism by which P is absorbed in ruminants. Using sheep with Thiry-Vella loops in the jejunum, P absorption was shown to rise when the concentration of P in the solution used to perfuse the loop was increased (Care *et al.* 1980). However, when the intra-luminal concentration of P was raised above 15 mmol/l no further increase in absorption was observed, a result which these authors viewed as evidence for the saturation of a carrier mechanism in an absorption process involving facilitated diffusion.

The administration of large amounts of parathyroid hormone over several days has been shown to reduce faecal phosphorus excretion in cattle (Mayer *et al.* 1968), though whether this was due to reduced secretion or increased absorption is not clear. In sheep parathyroidectomy has been shown to result in a negative balance for both Ca and P and it has been suggested that the effect on P balance was the result of a reduction in the amount of salivary P reabsorbed (McIntosh & Tomas, 1978). 1,25 $(OH)_2D_3$ has also been suggested as a possible regulator of intestinal P absorption in ruminants (Braithwaite, 1978, 1980; Care *et al.* 1980) though whether this was a primary effect or secondary to its effects on Ca absorption and deposition in bone is as yet not clear.

It is possible that some of the variation in renal response to phosphate loading seen by us and others may relate to differences between individuals in the blood levels of some of these hormones or indeed some other hormone more specifically concerned with P homoeostasis. At present, however, we have no additional information available on this subject though clearly there still remains considerable scope for further study.

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