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III. Quantitative aspects of phosphorus excretion in ruminants

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Abstract — Ruminant phosphorus excretion and metabolism were studied through a database. Faecal endogenous phosphorus is the main pathway of phosphorus excretion and averages 0.85 of total faecal phosphorus. The remaining 0.15 is unabsorbed dietary phosphorus. Faecal endogenous phosphorus is mainly unabsorbed phosphorus, with saliva being the major source, and is correlated to factors influencing saliva secretion (DM intake, physical dietary characteristics and dietary phosphorus content). Another source of faecal endogenous phosphorus is rumen microbial phosphorus that escaped solubilisation during post-rumen digestion. All factors stimulating microbial growth would increase phosphorus uptake by the rumen microbes and consequently the faecal endogenous phosphorus. Understanding the determinants of faecal endogenous phosphorus flow will help to precise the determination of net phosphorus requirements for maintenance. The role of plasma phosphorus in urinary phosphorus loss is discussed.

excretion / phosphorus / quantitative aspect / ruminant

1. INTRODUCTION

Animal husbandry concentration in production areas greatly contributes to water pollution through excessive nutrient dejection output. Because it promotes the development of green algae in lakes and rivers, phosphorus is a major freshwater pollutant. In the 1990s, numerous studies were carried out on monogastric phosphorus metabolism. In Europe, ruminant livestock produces as much as 0.70 of total phosphorus dejection from animal husbandry [1]. Ruminants excrete phosphorus mainly in the faeces, with the faecal loss being constituted of unabsorbed dietary phosphorus and endogenous phosphorus (P from saliva, intestinal cells, and digestive secretions). Relatively high urinary phosphorus excretion is, however, observed in ruminants under specific nutritional conditions. Since urinary phosphorus is highly labile, its polluting potential is higher than that of faecal phosphorus.

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The aim of the present paper was to improve the understanding and quantification of phosphorus excretion for a better adjustment of phosphorus supply to phosphorus requirements through the analysis of a database described previously [2].

2. MATERIALS AND METHODS

The constitution of the database, the statistical analysis of the data and the abbreviations used in the text were described in a previous article [2]. The term RES[Y(X)] is used when a variable Y that is dependent on a variable X is analysed after eliminating the variation effect of X.

Urinary phosphorus loss (PURI) and endogenous faecal phosphorus loss (PFEC\textsubscript{ENDO}) are the two sources of endogenous phosphorus excretion in the ruminant. Total faecal phosphorus (PFEC\textsubscript{TOT}) also contains unabsorbed dietary phosphorus (PFEC\textsubscript{EXO}). The aim of this paper was to analyse the PURI and PFEC\textsubscript{ENDO} flows in relationship to the diet and other factors involved in phosphorus metabolism, notably plasma phosphorus concentration (PPLASM).

For each of the following relationships, the number of observations or treatments (TRT), the number of papers taken into account (EXP), the total number of animals involved (ANIM), the model root mean square error (RMSE), the adjusted square of the correlation coefficient ($r^2$) and the significance probability level ($P$) are indicated. The regression coefficients are followed with their standard error in brackets. Table I describes the publications used in each model of the present paper using the codification of the variable PUB detailed in the “References – Appendix” of [2].

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3. RESULTS

3.1. Endogenous faecal phosphorus

3.1.1. Endogenous faecal phosphorus and phosphorus components of metabolism

Endogenous faecal phosphorus flow is a variable part of total faecal phosphorus flow. Their variations are highly correlated as shown by the following model adjusted on DM intake:

$$\frac{PFECTOT}{DMI} = -0.66 (\pm 0.13) + 2.26 (\pm 0.08) \times \frac{PFECENDO}{DMI}$$

(TRT = 356, EXP = 53, ANIM = 1266, RMSE = 2.26, \(r^2 = 0.66, P < 0.01\), model 1).

When considering only the database trials in the current range of dietary phosphorus (from 2.5 g to 5.0 g ingested phosphorus per kg DM), this model becomes:

$$\frac{PFECTOT}{DMI} = 0.90 (\pm 0.14) + 1.03 (\pm 0.08) \times \frac{PFECENDO}{DMI}$$

(TRT = 113, EXP = 31, ANIM = 433, RMSE = 0.99, \(r^2 = 0.55, P < 0.01\), model 2).

Endogenous faecal phosphorus consists of unabsorbed phosphorus from digestive secretions, principally saliva. When normalised with respect to DM intake, endogenous faecal phosphorus is effectively highly correlated with salivary phosphorus flow:

$$\frac{PFECENDO}{DMI} = 0.31 (\pm 0.02) \times PSAL/DMI$$

(TRT = 34, EXP = 5, ANIM = 139, RMSE = 2.06, \(r^2 = 0.79, P < 0.01\), model 3).

Because this relationship was observed in almost all of the trials considered, we calculated the within-trial model that is a general response:

$$\frac{PFECENDO}{DMI} = 0.33 (\pm 0.02) \times PSAL/DMI$$

(TRT = 34, EXP = 5, ANIM = 139, RMSE = 2.01, \(r^2 = 0.85, P < 0.01\), model 4).

According to these highly precise models (RMSE = 2.01 and \(r^2 = 0.85\)), when a ruminant produces 1 g of salivary phosphorus, it loses 0.33 g of endogenous phosphorus in the faeces. Since the constant term is not different from 0, these models also revealed that in the absence of saliva production, there is no endogenous faecal loss. This was surprising since it is generally accepted that endogenous faecal phosphorus is also provided by other digestive secretions [3]. According to model 4, 0.85 of the variation of endogenous faecal phosphorus flow are due to variations of salivary phosphorus. Then, we can infer that dietary components influence the faecal endogenous phosphorus loss via their action on salivary phosphorus flow.

3.1.2. Endogenous faecal phosphorus, salivary phosphorus and dietary influences

DM intake impacts salivary phosphorus as shown by the model adjusted on body weight:

$$PSAL/BW = 0.07 (\pm 0.01) + 3.22 \times 10^{-3} (\pm 0.61 \times 10^{-3}) \times \frac{DMI}{BW}$$

(TRT = 48, EXP = 9, ANIM = 181, RMSE = 6.43 \times 10^{-2}, \(r^2 = 0.36, P < 0.01\), model 5).

As suggested by the relationship between salivary phosphorus flow and faecal endogenous phosphorus flow (model 4), DM intake also closely influenced the endogenous faecal phosphorus as shown by the within-trial model calculated in the usual range of dietary phosphorus (2.5–5.0 g of phosphorus per kg DM):

$$\frac{PFECENDO}{BW} = 1.41 \times 10^{-3} (\pm 0.04 \times 10^{-3}) \times \frac{DMI}{BW}$$

(TRT = 341, EXP = 55, ANIM = 1252, RMSE = 3.19 \times 10^{-2}, \(r^2 = 0.78, P < 0.01\), model 6, Fig. 1).

Endogenous faecal phosphorus also increased linearly with ingested phosphorus when related to body weight or to DM intake:

$$\frac{PFECENDO}{BW} = 1.17 \times 10^{-2} (\pm 0.08 \times 10^{-2}) + 0.250(\pm 0.01) \times \frac{PING}{BW}$$

(TRT = 377, EXP = 56, ANIM = 1317, RMSE = 2.19 \times 10^{-2}, \(r^2 = 0.68, P < 0.01\), model 7)

$$\frac{PFECENDO}{DMI} = 0.70 (\pm 0.04) + 0.20 (\pm 0.01) \times \frac{PING}{DMI}$$
According to these models, the minimum theoretical endogenous faecal phosphorus loss was 0.0117 g·kg⁻¹ of body weight or 0.70 g·kg⁻¹ DM intake when no phosphorus was ingested representing 8.19 g·day⁻¹ for a 700 kg cow as for a cow fed 11.7 kg of DM, and 16.1 g·day⁻¹ for a cow ingesting 23 kg of DM expected to produce 35 kg of milk.

The forage dietary content is an indicator of the dietary physical form that significantly affects saliva phosphorus flow as shown by the following between-trial model:

\[
\text{PSAL/BW} = 8.58 \times 10^{-2} (\pm 1.25 \times 10^{-2}) + 6.98 \times 10^{-4} (\pm 1.67 \times 10^{-4}) \times \%\text{FOR} \\
(\text{TRT} = 32, \text{EXP} = 8, \text{ANIM} = 126, \text{RMSE} = 0.06, r^2 = 0.34, P < 0.01, \text{model 9}).
\]

The variation of salivary phosphorus was also positively influenced by the ingested phosphorus as shown on the residual variation of the previous model 9 for a constant dietary forage content (RMSE = 0.03 and \( r^2 = 0.13 \)).

The total forage content of the diet also increased the faecal endogenous phosphorus flow:

\[
\text{PFEC}_{\text{ENDO}} = 1.11 (\pm 0.10) + 1.65 \times 10^{-2} (\pm 0.17 \times 10^{-2}) \times \%\text{FOR} \\
(\text{TRT} = 342, \text{EXP} = 59, \text{ANIM} = 1190, \text{RMSE} = 2.43, r^2 = 0.20, P < 0.01, \text{model 10}).
\]

Dietary crude fibre content significantly affected the faecal endogenous phosphorus flow, however, even though crude fibre was well correlated with forage content, the model was of lower precision:

\[
\text{PFEC}_{\text{ENDO}} = 0.95 (\pm 0.25) + 4.66 \times 10^{-3} (\pm 1.13 \times 10^{-3}) \times \text{CF} \\
(\text{TRT} = 276, \text{EXP} = 39, \text{ANIM} = 995, \text{RMSE} = 2.71, r^2 = 0.05, P < 0.01, \text{model 11}).
\]

The precision was higher for the relationship between crude fibre content and the

![Figure 1](https://example.com/figure1.png)  

**Figure 1.** Intra-trial relationship between faecal endogenous phosphorus (PFEC\(_{\text{ENDO}}\) g·day\(^{-1}\)) and DM intake (DMI, g·day\(^{-1}\)) normalised according to body weight (BW, kg). The intra-trial model appears as a solid line.
part of faecal endogenous phosphorus over total faecal phosphorus:

\[
\frac{PFEC_{ENDO}}{PFEC_{TOT}} = 0.44 (±0.03) + 1.53 \times 10^{-3} (±0.30 \times 10^{-3}) \times CF - 1.97 \times 10^{-6} (±0.73 \times 10^{-6}) \times CF^2
\]

(TRT = 273, EXP = 38, ANIM = 986, RMSE = 0.14, \(r^2 = 0.20\), \(P < 0.01\), model 12, Fig. 2).

However, since no trial reported the influence of dietary crude fibre content, we also calculated the inter-trial model.

\[
\text{MEAN}[\frac{PFEC_{ENDO}}{PFEC_{TOT}}] = 0.42 (±0.02) + 1.88 \times 10^{-3} (±0.75 \times 10^{-3}) \times \text{MEAN}[CF] - 3.0 \times 10^{-6} (±0.02 \times 10^{-6}) \times \text{MEAN}[CF]^2
\]

(TRT = 273, EXP = 38, ANIM = 986, RMSE = 0.11, \(r^2 = 0.34\), \(P < 0.01\), model 13).

No significant relationship was observed between phytate phosphorus content and neither faecal endogenous phosphorus nor the ratio of faecal endogenous phosphorus to total faecal phosphorus.

### 3.1.3. Prediction of faecal endogenous phosphorus

The database used in the present paper can help by the number of treatments, experiments and animals involved in standardising a relationship of good precision. For this reason, it can be an interesting tool for the prediction of faecal endogenous phosphorus. Since DM intake and dietary crude fibre content are parameters easy to know, we investigated both in order to predict the amount of endogenous faecal phosphorus:

\[
PFEC_{ENDO} = 0.96 (±0.15) + 0.76 (±0.03) \times DMI - 1.67 \times 10^{-3} (±0.70 \times 10^{-3}) \times CF
\]

(TRT = 268, EXP = 36, ANIM = 972, RMSE = 1.51, \(r^2 = 0.71\), \(P < 0.01\), model 14).

According to this model, a dairy cow ingesting 20 kg of DM containing 400 g of crude fibre per kg of DM would excrete 16.01 g·day⁻¹ of faecal endogenous phosphorus. This model also predicts 1.57 g·day⁻¹ of faecal endogenous phosphorus in an
adult sheep ingesting 1.5 kg of DM containing 300 g of CF per kg of DM.

Since in apparent phosphorus digestibility trials, the ingested, faecal and urinary phosphorus values are available data whereas endogenous faecal phosphorus flow measured by radioisotopic dilution is more difficult to obtain, we investigated all three parameters combined to predict the amount of endogenous faecal phosphorus. The following model was calculated from the 470 adult sheep involved in the database:

\[
PFE_{ENDO} = 0.55 (\pm 0.03) + 0.10 (\pm 0.05) \times PING + 0.21 (\pm 0.05) \times PFE_{TOT} - 0.19 (\pm 0.07) \times PURI
\]

(\(TRT = 169, EXP = 16, ANIM = 470, RMSE = 0.50, r^2 = 0.84, P < 0.01, model 15\)).

This model of good precision (RMSE = 0.50, \(r^2 = 0.84\)) predicts 1.6 g·day\(^{-1}\) of faecal endogenous phosphorus for 3.12 g·day\(^{-1}\) of phosphorus intake and 2.63 g·day\(^{-1}\) and 0.0035 g·day\(^{-1}\) phosphorus loss by the faecal and urinary routes, respectively, as measured on 65 kg sheep (Bravo, unpublished data).

### 3.2. Urinary phosphorus

Urinary phosphorus is an alternative way of excretion of phosphorus in the ruminant. Even if it is usually a low flow, it is still interesting to better know its determinism. In the database, 0.47 of the variations in urinary phosphorus were statistically attributable to variations in plasma phosphorus concentration:

\[
PURI/BW = -6.64 \times 10^{-3} (\pm 1.51 \times 10^{-3}) \times PPLASM + 4.58 \times 10^{-3} (\pm 0.56 \times 10^{-3}) \times PPLASM^2
\]

(\(TRT = 254, EXP = 33, ANIM = 791, RMSE = 2.58 \times 10^{-2}, r^2 = 0.47, P < 0.01, model 16, Fig. 3\)).

---

**Figure 3.** Relationship between urinary phosphorus (PURI, g·day\(^{-1}\)) normalised according to the body weight (BW) and plasma phosphorus concentration (PPLASM, mmol·L\(^{-1}\)). The points from the same trial are connected by a solid line.
This model shows that all the factors increasing plasma phosphorus content will entail urinary excretion of phosphorus. For instance, model 17 presents a significant relationship explaining that 0.53 of the variation of urinary phosphorus are also attributed to ingested phosphorus variation:

\[
\text{PURI/DMI} = 0.10 ( \pm 0.03 ) \times \text{PING/DMI} - 6.58 \times 10^{-5} ( \pm 0.44 \times 10^{-5} ) \times \text{PING/DMI}^2
\]

(TRT = 374, EXP = 59, ANIM = 1274, RMSE = 4.75, \( r^2 = 0.53 \), \( P < 0.01 \), model 17, Fig. 4).

According to this model, when phosphorus ingestion increases from 2 g·kg\(^{-1}\) of DM intake to 6 g·kg\(^{-1}\) of DM intake, urinary phosphorus excretion increased from 0.29 g·day\(^{-1}\) to 0.89 g·day\(^{-1}\). Whereas only 0.17 of the variation of ingested phosphorus influenced plasma phosphorus, 0.53 of the variation of ingested phosphorus influenced urinary phosphorus. Urinary phosphorus excretion is influenced by ingested phosphorus as shown by model 19, determined on only the trials involving variations in phosphorus ingestion:

\[
\text{PPLASM} = 1.39 ( \pm 0.11 ) + 0.31 ( \pm 0.05 ) \times \text{PING/DMI} - 1.83 \times 10^{-2} ( \pm 0.41 \times 10^{-2} ) \times \text{PING/DMI}^2
\]

(TRT = 315, EXP = 52, ANIM = 1646, RMSE = 0.67, \( r^2 = 0.17 \), \( P < 0.01 \), model 18, Fig. 5).

According to this model, with a diet supplying 3.5 g of phosphorus per kg of DM intake, plasma phosphorus concentration would be of 2.5 mmol·L\(^{-1}\). However, only 0.17 of the variation of plasma phosphorus are due to ingested phosphorus differences.

Besides the dietary phosphorus content or ingested phosphorus flows, other dietary components influence plasma phosphorus content and are then susceptible to influence...
urinary phosphorus excretion. This is the case for the dietary forage (model 20) or crude fibre contents (model 20):

\[ P_{\text{PLASM}} = 2.45 \pm 0.06 - 7.24 \times 10^{-3} \pm (0.99 \times 10^{-3}) \times \% \text{FOR} \]

(TRT = 332, EXP = 44, ANIM = 2077, RMSE = 0.70, \( r^2 = 0.14, P < 0.01 \), model 19)

\[ P_{\text{PLASM}} = 2.91 \pm 0.13 - 3.84 \times 10^{-3} \pm (0.51 \times 10^{-3}) \times \text{CF} \]

(TRT = 280, EXP = 33, ANIM = 1376, RMSE = 1.36, \( r^2 = 0.16, P < 0.01 \), model 20).

These models show that high plasma phosphorus contents were observed in certain animal groups receiving low dietary crude fibre, such as the milk-fed ruminants and the sheep fed with high dietary concentrate, including dried poultry manure. This model can be criticised because it included both of these particular treatments. For this reason, we removed them, and the model was still significant showing the low statistical weight of these treatments in the negative relationship between fibre content and plasma phosphorus. Since only one experiment included variations in the dietary crude fibre content [36], we also calculated the inter-trial relationship:

\[ \text{MEAN}[P_{\text{PLASM}}] = 3.03 \pm 0.08 - 4.49 \times 10^{-3} \pm (0.42 \times 10^{-3}) \times \text{MEAN}[\text{CF}] \]

(TRT = 280, EXP = 33, ANIM = 1376, RMSE = 0.89, \( r^2 = 0.33, P < 0.01 \), model 21, Fig. 6).

Since crude fibre content influences notably plasma phosphorus, we expected a significant relationship with urinary phosphorus:

\[ \text{PURI/BW} = 1.13 \pm 0.06 \times 1/\text{CF} \]

(TRT = 250, EXP = 36, ANIM = 897, RMSE = 2.20 \times 10^{-2}, \( r^2 = 0.56, P < 0.01 \), model 22, Fig. 7).
Figure 6. Inter-trial relationship between the plasma phosphorus concentration (PPLASM, mmol·L$^{-1}$) and the dietary crude-fibre content (CF, g·kg$^{-1}$ DM). The inter-trial model appears as a solid line.

Figure 7. Relationship between urinary phosphorus (PURI, g·day$^{-1}$) and dietary crude-fibre content (CF, g·kg$^{-1}$ DM). The model appears as a solid line.
According to this model, 0.56 of the urinary phosphorus variations are explained by differences in dietary crude fiber content and urinary excretion of phosphorus is low and almost constant when the diet contains more than 100 g·kg⁻¹ of crude fibre.

Beside the forage and crude fibre contents of the diet, the cereal and cereal by-products and phytate phosphorus content were positively but lowly correlated with plasma phosphorus (respectively, \( r^2 = 0.09 \) and \( r^2 = 0.13 \)).

4. DISCUSSION

4.1. Urinary phosphorus excretion

While phosphorus excretion is mainly urinary in pigs, and urinary and faecal in humans, urinary phosphorus is the alternate phosphorus excretion pathway in the ruminant; it is quantitatively negligible [4, 5] but not always [6]. In growing calves urinary phosphorus could represent as much as 0.88 of the total phosphorus loss [7].

Because urinary excretion of phosphorus persists after correction of metabolic acidosis [8] it is not a way for the organism to correct metabolic acidosis as previously thought [6]. Urinary phosphorus excretion might be genetically predetermined [9] or occur in animals with low phosphorus requirements [10]. Nevertheless, despite its high variability, urinary phosphorus excretion contributes to phosphorus homeostasis of ruminants [11] and can be stated as a fixed phosphorus loss [12]. By decreasing the glomerular filtration rate, the organism can adjust the plasma phosphorus [13] and excrete extra phosphorus when plasma increases above a plasma phosphorus

![Figure 8. Relationship between the ratio of faecal endogenous phosphorus to total faecal phosphorus (PFEC\textsubscript{ENDO} / PFEC\textsubscript{TOT}) and urinary phosphorus (PURI, g·kg\textsuperscript{-1}) normalised according to body weight (BW, kg).](image-url)
concentration threshold close to 2 mmol·L⁻¹ (1.6 mmol·L⁻¹ [14], 2 mmol·L⁻¹ [15], 2.6 mmol·L⁻¹ [16] and 2–3 mmol·L⁻¹ [17] as in pigs (MacIntosh and Scott, 1975 cited by [18] as illustrated in model 16 and Figure 3. We found urinary phosphorus flow non significant when dietary supply was low but significant above a step of requirements [7, 19], and correlated ($r^2 = 0.53$) with ingested phosphorus (model 17, Fig. 4). The meta-analysis of our data base pointed out the dietary crude fibre content as the secondary factor to reduce urinary phosphorus (model 22, Fig. 7) as reported by [6]. The low correlation of dietary factors with plasma phosphorus content is the consequence of homeostasis regulation [20]. However, urine is a secondary excretion route for phosphorus in the current range of dietary phosphorus supply. Figure 8 provides a schematic representation of the partitioning of phosphorus excretion between urine and faeces.

4.2. Faeces, the main phosphorus excretion route

Faeces represent the main pathway of phosphorus excretion in ruminants. Faecal phosphorus also contains unabsorbed dietary phosphorus, the exogenous constituent of faecal phosphorus. This ratio of endogenous faecal phosphorus to total faecal phosphorus is highly variable from 0.12 to 0.95 in our database. The lowest value (0.12) was obtained in a growing calf fed a milk-based diet [7] and in adult sheep fed a diet rich in wheat bran [21]. The highest value (0.95) was obtained with high forage content diets as in the study of [22] for the determination of phosphorus availability in alfalfa hay.

4.2.1. Saliva: the main origin of faecal phosphorus excretion

In a cow, the daily ingestion induces up to 250 L of liquid digesta flow through the duodenum versus 15 L in monogastric animals of similar size [23]. In ruminants, saliva secretion controls the homeostasis of the rumen ecosystem by supplying buffers [24] and one of the primary roles of saliva is to transfer phosphorus from the plasma to the digestive tract [25]. This transfer explains why plasma phosphorus concentration rises when the parotid vein is ligated [26] or when chewing is limited [7]. Saliva production was investigated by [27]. Few studies report on salivary phosphorus secretion and the factors mediating its variation [8, 28, 29]. Salivary phosphorus flux or secretion (PSAL) is determined by the daily saliva flux ($F_{SAL}$, in L·day⁻¹), and the saliva phosphorus content ($C_{PSAL}$, in g·L⁻¹), according to the equation $PSAL = C_{PSAL} \times F_{SAL}$. None of these parameters could be recorded in the database. Daily saliva flow ($F_{SAL}$) is influenced mainly by DM intake and by dietary fibre content [30] since both affect chewing intensity [5, 31]. These factors also affect PSAL (DM intake, model 5 and dietary forage content, model 9). Salivary and plasma phosphorus concentrations are highly correlated [25]. They respectively averaged 16.2 mmol·L⁻¹ [32] and 2 mmol·L⁻¹ [16]. Therefore the salivary phosphorus to plasma phosphorus ratio ranges from 6 to 13 in ruminants compared to 4 in humans [23]. The direct correlation of ingested phosphorus and PSAL for a constant dietary forage content (model not shown) would suggest that ingested phosphorus influences salivary phosphorus content ($C_{PSAL}$). In the literature, the determining factors of salivary phosphorus concentration are subject to debate. In fact, salivary phosphorus flux would be a consequence of the saliva flow, saliva phosphorus concentration being inversely proportional to saliva flow [29, 33], but only up to a given threshold value [34]. A relationship between salivary phosphorus flux and dietary phosphorus was investigated in our study. Firstly, when dietary phosphorus content increases, salivary phosphorus flux decreases since the duodenal phosphorus flux remains
unchanged in dairy cows [35]. Secondly, when dietary phosphorus content decreases, the ratio of salivary phosphorus content to plasma phosphorus content (phosphorus concentration capacity of the salivary glands) remains constant in sheep [36]. Both results would indicate the existence of a step value for ingested phosphorus. At a higher ingested phosphorus, the salivary phosphorus flux decreases [35]. For lower amounts of ingested phosphorus, the salivary phosphorus flux does not diminish [36], maintaining a sufficient phosphorus supply to the rumen microbes, as we previously underscored [37] observed that over a phosphorus intake of 100 mg·kg⁻¹ of body weight, the salivary phosphorus flux is blocked at a maximum value. However, a possible level of saturation remains to be demonstrated [15]. Moreover, some authors believe that salivary phosphorus is not under homeostatic control [17].

4.2.2. Faecal endogenous loss

4.2.2.1. The conventional approach to PFEC<sub>ENDO</sub>

It is widely held that endogenous faecal phosphorus is composed of unabsorbed salivary phosphorus. Saliva is the major contributor of endogenous faecal phosphorus (up to 0.80, [3]) since as much as 200 mg·h⁻¹ of phosphorus enters the rumen from the saliva compared to 50 mg in the omasum, 30 mg in the abomasum and 12 mg in the intestines from the other digestive secretions in a 35–40 kg sheep [38]. The difference in saliva secretion explains why pre-ruminants have 13 times less endogenous faecal phosphorus than adult ruminants [39]. The endogenous faecal phosphorus level was influenced by the same factors that modify salivary phosphorus secretion and the extent of phosphorus absorption, particularly the dietary factors such as DM intake (model 6, for PFEC<sub>ENDO</sub> and model 5 for PSAL). In our database, no significant relationship between endogenous faecal phosphorus and dietary forages was observed in contrast with [31] but a significant relationship linked crude fibre content and the ratio of endogenous faecal phosphorus to total faecal phosphorus (models 12 and 13; [26, 28, 29]). The relationship between ingested phosphorus and endogenous faecal phosphorus (models 7 and 8) was observed previously [31, 40, 41]. Ingested phosphorus would affect endogenous faecal phosphorus at two levels, the concentration of salivary phosphorus and the extent of phosphorus absorption. The latter determines the partitioning between absorbed salivary phosphorus (PAB<sub>SAL</sub>) and unabsorbed salivary phosphorus (PFEC<sub>ENDO</sub>).

4.2.2.2. Another approach to PFEC<sub>ENDO</sub>

Conventionally, endogenous faecal phosphorus is separated into two compartments: an obligatory loss imposed by salivary secretion and a homeostatic adjustment portion dependent on the dietary phosphorus [5]. [41] assumed faecal endogenous phosphorus to be constituted by rumen microbial phosphorus that escapes solubilisation by post-rumen digestion. Unfortunately, because no information on faecal DM content (DM<sub>FEC</sub>, in g/day) was included in our database, we could not use Conrad’s equation (1999), cited by [42] to estimate faecal microbial phosphorus (PFEC<sub>BACT</sub> = 3.7 × 10⁻³ × DM<sub>FEC</sub>). Endogenous faecal phosphorus would be separated into homeostatic phosphorus loss (PFEC<sub>HS</sub>) and rumen microbial phosphorus (PFEC<sub>BACT</sub>). All factors stimulating microbial growth (eg., digestible organic matter) would increase phosphorus uptake by rumen microbes and consequently faecal endogenous phosphorus content (PFEC<sub>BACT</sub>; [43]). Moreover, a relationship between the homeostatic portion of endogenous faecal phosphorus and plasma phosphorus would then be expected.

4.2.2.3. Calculation of the net maintenance phosphorus requirement

Better knowledge of the determining factors of endogenous faecal phosphorus
excretion leads to a more precise calculation of the net phosphorus requirement for maintenance. The net phosphorus requirement for maintenance has been estimated based upon body weight [44], although different animals with the same weight ingest different amounts and types of feed. Precision was improved by predicting the net phosphorus requirement for maintenance according to DM intake [45]. As suggested in the alternate endogenous fecal phosphorus partitioning approach described above, additional precision would be possible by predicting the net phosphorus requirement for maintenance according to dietary composition, in particular according to factors influencing salivation and microbial uptake. [46] has already implemented the principle of such an approach by attributing a higher net phosphorus requirement for maintenance for forage-based than for concentrate-based diets. At present, it would be preferable to integrate the dietary characteristics to calculate the net phosphorus requirement for maintenance as suggested by the relatively high correlation coefficient of our model 13. As expressed recently by [45], more extensive experimental data would be useful to allow one to take these additional elements into account.

5. CONCLUSION

In ruminants, phosphorus homeostasis is assured at three levels: (i) primarily in the salivary glands by ruminant phosphorus excretion into the saliva, (ii) in the intestinal sites of phosphorus absorption by variable rates of absorption, and (iii) in the kidney by the alternative route of phosphorus excretion. Bone phosphorus also plays a role in phosphorus homeostasis but it was not studied in this paper.

The present review points out the dietary situations influencing phosphorus homeostasis. The modality and extent of endogenous phosphorus excretion in the faeces vary according to the dietary characteristics (such as fibre content). Knowing the endogenous faecal phosphorus value is important for the calculation of the phosphorus maintenance requirement. The quantitative models developed in this study, the putative contribution of rumen microbes to endogenous faecal phosphorus, and trials being carried out in several countries to decrease phosphorus pollution from animal husbandry might be able together to provide a mechanistic model that could improve the precision of the factorial method. Such an improvement would no doubt lead to better control of phosphorus pollution from ruminant livestock.

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