apparent availability of the dietary Mg. This result was confirmed in the intact sheep by Care & Ross (1963) using deoxycorticosterone acetate instead of aldosterone.

Since in most circumstances the diet provides excessive amounts of Mg, it seems likely that the endocrine factors function to reduce the availability of dietary Mg rather than to increase it. Notwithstanding the recent acceleration in research devoted to Mg homoeostasis, as a result of the introduction of accurate and sensitive methods for the determination of Mg in biological material, much still remains to be known of these factors which regulate its absorption from the digestive tract.

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The availability of the calcium and phosphorus of plant materials for animals

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The availability of calcium and phosphorus for ruminants has been reviewed recently by Hill (1962) and it is proposed, therefore, to discuss only non-ruminants in this paper, and to concentrate largely on the problem of the availability of phytate

P and on the effect of phytate on the availability of Ca. Those who are not working in the field of mineral nutrition might be forgiven for thinking that these problems had been solved long ago, but there is in fact a great deal of confusion still surrounding the role of phytate in nutrition.

The phytate story is inextricably mixed with that of rickets and with the early history of vitamin D, and it began with the demonstration by Mellanby (1920) that cereals, especially oats, are rachitogenic for puppies. It was not until 1934 that Bruce & Callow (1934), working on low-P rickets in rats, provided the key to this mystery. They showed that P from cereal sources was less effective than inorganic phosphate in healing rickets and suggested that P combined as phytate was poorly absorbed from the digestive tract. They also recognized that phytic acid forms an insoluble Ca salt and that it would be liable therefore to interfere with the absorption of Ca. (The fact that something like two-thirds of the total P in cereal grains is present as salts of phytic acid, the hexaphosphoric acid ester of inositol, had been known for many years.)

It is clear from the conclusions which Bruce & Callow (1934) drew from their experiments that they had a clear understanding of the nature of the basis of the rachitogenic effect of cereal diets deficient in vitamin D: (1) on diets high in Ca rickets is due to a P deficiency caused by the low availability of the P in cereals, (2) on diets low in Ca rickets is due to a Ca deficiency caused by the impairment of Ca absorption by the phytate present.

When these basic facts had been confirmed research was largely directed to a study of (a) the conditions under which the breakdown of phytate occurs, i.e. factors affecting the 'availability' of phytate P, including the role of vitamin D and (b) the influence of phytate on the Ca and P requirements of different animals.

Current interest in phytate in animal nutrition is more in relation to the availability of its P than to its ability to immobilize Ca. The reason for this is simple—P is expensive, Ca is cheap. However, in concentrating on the former problem there is a danger that effects associated with the Ca-binding properties of phytate may be overlooked when they occur in feeding experiments.

One of the first demonstrations that breakdown of phytate can occur in the intestines was given by Lowe & Steenbock (1936*a*) in a balance experiment with adult rats, using a basal diet of maize (76 parts) and wheat gluten (20 parts) containing 0·058% Ca and 0·28% P (largely phytate P). When this diet was supplemented with approx. 9% native wheat phytate to provide an additional 397 mg P per rat per period, only an extra 148 mg organic P appeared in the faeces, whereas urinary P increased by 67 mg, inorganic faecal P by 152 mg and the P balance by 30 mg. In the next trial 3% calcium carbonate was added to the phytate-supplemented basal diet, and when the balance values for these periods were compared with those for the periods when the basal diet alone was given it was clear that phytate hydrolysis had been drastically reduced, and urinary excretion of P was hardly detectable (Table 1). Additions of 3% magnesium, strontium or barium carbonate had the same effect as that of 3% calcium carbonate in reducing the breakdown of phytate.

Table 1. *Phosphorus intake and excretion (mg per rat per 4-day period) of rats on a maize–gluten diet (means of eight values) (Lowe & Steenbock, 1936a)*

Diet	Intake	Urine	Faeces			Total output	Balance
			Inorganic	Organic	Total		
Basal	96	45	21	17	38	83	13
Basal + phytate	493	112	173	165	338	450	43
Basal + phytate + 1·2% Ca	486	Trace	56	394	450	450	36

When the maize of the basal diet was replaced by oats or wheat, with or without Ca supplementation, the results were essentially the same.

Mellanby (1950) showed that, in some experiments, as much as 80% of the phytate P given to puppies disappeared from the gut when vitamin D was present. The percentage of dietary phytate hydrolysed was very much less in vitamin D-deficient animals than in animals given the vitamin.

Common (1940), working with pullets, showed that the recovery of ingested phytate in the droppings was considerably greater when a particular diet was supplemented with calcium carbonate than when a calcium phosphate supplement provided the same amount of Ca. In the former situation 77–84% was recovered and in the latter 60–68%. When whole wheat alone was given only 34% of the phytate appeared in the droppings, but when a calcareous grit was given in addition to the wheat 54% of the phytate P was recovered.

A similar observation was made by Moore & Tyler (1955*a*,*b*) in experiments with pigs fed on a cereal-based diet supplemented with either calcium carbonate or calcium phosphate and killed 4 h after feeding (Table 2). The mean content of phytate P in the different sections of the large intestine was approx. four times as great when the dietary Ca was in the form of carbonate as when it was supplied as phosphate. From a practical standpoint, therefore, it was the diet less in need of additional P in which the greater breakdown of phytate occurred.

H. Nott (1964, private communication) in an investigation with laying hens has obtained virtually 100% recovery of dietary phytate in the droppings when the diet contained 3·5% or 4·5% Ca, but with 2·5% and 1·5% Ca the recovery fell markedly to 85% and 55% respectively.

Table 2. *Mean percentage of phytate phosphorus in the dry matter of the food and of certain sections of the gut contents of pigs fed on a cereal-based diet supplemented with calcium carbonate (to provide a total of 1·67% Ca and 0·78% P) or calcium phosphate (to provide 1·06% Ca and 1·28% P) (Moore & Tyler, 1955a,b)*

	With carbonate supplement	With phosphate supplement
Food	0·46	0·48
Last quarter of small intestines	0·91	0·75
Caecum	0·54	0·15
Colon	0·41	0·14
Rectum	0·65	0·10

It is clear from these experiments that phytate P is by no means completely

https://doi.org/10.1079/PNS19650017 Published online by Cambridge University Press

undigested by the rat, dog, pig and hen under appropriate conditions, and it appears that the extent of phytate breakdown in the gut is reduced as the level of dietary Ca increases and that at very high levels phytate destruction may be completely prevented. Species may vary in the level of Ca in the diet at which a particular degree of inhibition of phytate hydrolysis is shown for a standard diet, but it has not been demonstrated that there are any fundamental differences between species in the way phytate behaves in the gut.

The questions that must now be asked are 'How is phytate broken down in the gut and to what extent is the liberated phosphate available to the animal?' Before discussing these questions, however, the possibility that the disappearance of phytate from the gut may be the result of direct absorption must be considered. Few workers took this possibility seriously until Shohl (1939) stated that 'Phytin is readily soluble in gastric juice and is absorbed without decomposition. It must be split before it can be utilised. Part is re-excreted unchanged in the faeces'. This view has been quoted by Singsen (1948), Ashton, Evans & Williams (1960) and Temperton & Cassidy (1964), but experimental evidence in support of this theory is lacking. Mellanby (1950) was unable to detect any phytate in the urine, blood or other tissues of dogs, and he remarked that soluble phytates (it is impossible to conceive that insoluble salts of phytic acid could be absorbed) must be very toxic because of their ability to precipitate Ca from the plasma. The workers who since Shohl have entertained the idea of a direct absorption of unchanged phytic acid have all worked with chicks and they have linked this suggestion with the presence of phytic acid in the red cells (Rapoport, 1940) and of a phytase in the plasma and red cells of birds (Rapoport, Leva & Guest, 1941). The suggestion implicit in this line of thought is that absorbed dietary phytate is used by the chick in the elaboration of its erythrocytes. It is now known that this is certainly not so, for it has been shown by the use of inorganic ^{32}P that the phytic acid in the red cells is synthesized at the time when the red cells differentiate and that it persists throughout the life of the cells without appreciable renewal (Oshima & Taylor, 1963; Oshima, Taylor & Williams, 1964). There was no indication of any fall in the specific activity of the phytic acid P of the blood cells for at least 13 days after the peak was reached so it is unlikely that the phytase present is active in functional red cells (Oshima & Taylor, 1963). The role of the plasma phytase, and possibly that in the erythrocytes also, would appear to be the hydrolysis of the phytic acid of the blood cells at the end of their useful life.

Associated with the idea that phytic acid is absorbed unchanged has grown up a concept that the P of phytate is available for growth but not for bone calcification, because on certain phytate-containing diets chicks grow as fast but do not show such high bone-ash percentages as on control diets containing added inorganic phosphate (Singsen, 1948). Waldroup, Ammerman & Harms (1964), on the other hand, concluded from their chick experiments that 'Calcium phytate phosphorus was relatively unavailable for growth purposes but somewhat more available for bone calcification'. If it is agreed that the P of phytate is in all probability absorbed as phosphate ions, the concept of a greater or lesser 'availability' for a particular physiological function becomes quite unacceptable.

Presumably the fraction of the dietary phytate that is broken down in the gut is hydrolysed by one or more phytases and there are three possible sources for these enzymes, the food, the digestive secretions and the bacteria of the digestive tract. There are powerful phytases present in wheat and rye, particularly the latter, barley contains an enzyme considerably less powerful, and oats and maize possess very little enzyme activity (McCance & Widdowson, 1944). The optimum pH for cereal phytase is about 5·1 and it shows some activity down to pH 3·0 (Hill & Tyler, 1954), so that considerable breakdown of dietary phytate in the crop of chickens and in the stomachs of non-ruminant animals, before the gastric secretion reduces the pH of the ingesta too low for enzyme activity, is theoretically possible when an active cereal phytase is present. Moore & Tyler (1955*a*,*b*) have in fact demonstrated that substantial hydrolysis of phytate from a mixed cereal diet occurs in the stomach of the pig. McCance & Widdowson (1944) have suggested that the lack of an active phytase in oats may explain why this cereal is more rachitogenic than wheat in low-Ca diets.

The activity of wheat phytase is completely destroyed at pH values of 2·5 and below (Hill & Tyler, 1954), so that it is unlikely that any appreciable amount of dietary phytase is able to survive the acid conditions of the stomach to resume its activity in the intestines.

Phytases from all sources act only on soluble phytate and there can be little doubt that the high recovery in the faeces of phytate present in diets containing large amounts of Ca is due to the low solubility of the phytate throughout the digestive tract. The effect noted above of diets high in phosphate promoting the breakdown of phytate may be brought about by increasing the solubility of the phytate by removing, as phosphate, Ca which would otherwise combine with and precipitate phytate, i.e. there may be competition between phosphate and phytate for Ca, a competition in which phosphate is successful only when it is present in large excess.

Increasing the level of Ca in diets containing phytate decreases the availability of zinc for pigs (Tucker & Salmon, 1955) and chicks (O'Dell, Yohe & Savage, 1964), and a possible explanation for this effect is that the breakdown of phytate by phytase is reduced at the higher levels of Ca, so that the amount of Zn precipitated as zinc phytate is increased and less, therefore, is available for absorption.

The relationship between the availability of phytate P and the solubility of the phytate is well illustrated by an experiment carried out by Waldroup *et al.* (1964) in which it was shown that the availability for chicks of the phosphate of free phytic acid and sodium phytate, both of which are highly soluble, was far greater than that of calcium phytate. The most reasonable explanation for these observations is in terms of the varying rates at which the different sources of phytate were likely to have been attacked by phytase.

The significance of plant phytase in the breakdown of dietary phytate may be considerable under favourable conditions but when cereals low in phytase are eaten it seems probable that the major part of phytate hydrolysis occurs in the intestines after plant phytases have ceased to function. The relative importance of intestinal and bacterial phytases in this region of the digestive tract is still unresolved. The very existence of an intestinal phytase was doubted until its presence in rats was

established by Patwardhan (1937) and confirmed by Spitzer & Phillips (1945), who also found the enzyme in the intestines of the chick, pig and cow. Mellanby (1950) was unable to detect appreciable amounts of phytase in the intestinal mucosa of dogs but he reported that the contents of the duodenum contained an active phytase. It has been shown recently that the phytate-splitting enzyme extracted from the duodenum of chicks is probably a non-specific alkaline phosphatase (Maddaiah, Kurnick, Hulett & Reid, 1964).

The requirements of animals, particularly chicks, for vitamin D increase as the proportion of the total P as phytate increases (cf. Gillis, Norris & Heuser, 1949), the requirements being minimal when all the P is inorganic. Another way of expressing the same concept is to say that vitamin D enhances the availability of phytate P (Krieger & Steenbock, 1940) and depresses the rachitogenic properties of phytate on low-Ca diets (Mellanby, 1950). These effects may be due, at least in part, to the influence of the vitamin on intestinal phytase. Steenbock, Krieger, Wiest & Pileggi (1953) showed that vitamin D increases the amount of phytase extractable from the intestines of rats and chicks, and this was confirmed for rats by Pileggi, De Luca & Steenbock (1955) and Roberts & Yudkin (1961). It is equally possible that the effect of vitamin D in stimulating Ca absorption increases the solubility of the phytate by reducing the concentration of Ca in the gut, and, indeed, both mechanisms may operate together.

There is widespread acceptance of the idea that phytate is broken down in the intestines of animals by bacteria as suggested by Lowe & Steenbock (1936*b*), and this is certainly the most obvious explanation of the fall in phytic acid content that Moore & Tyler (1955*b*) observed between the small and large intestines of pigs given a supplement of calcium phosphate. However, the existence of phytate-splitting bacteria in the gut contents of non-ruminants does not appear to have been reliably demonstrated. Mellanby (1950) was unable to detect any such bacteria in the gut contents or faeces of dogs given phytate, although there was an active phytase in bacteria-free filtrates derived from extracts of these materials. These experiments should be repeated for other species. In a recent communication Jenkins (1965) so far from finding a decrease in the phytate content of moist hen droppings over a period of 1 week observed an increase. If bacterial breakdown of phytate does in fact occur it is to be expected that the process would occur most actively in the large intestines, and any inorganic phosphate liberated in this region of the gut would be unlikely to be absorbed to any great extent. It is known, however, that considerable amounts of phytate P are absorbed under favourable conditions (Lowe & Steenbock, 1936*a*; Mellanby, 1950).

Insufficient evidence is available at present for any firm conclusions to be drawn concerning the role of bacteria in the hydrolysis of phytate in the intestines. Experiments with germ-free animals should throw light on this aspect of the phytate problem.

Phytates are normally considered to be important in nutrition in connexion with their ability to induce rickets, but it should be recognized that in certain circumstances phytates may be antirachitic, as Roberts & Yudkin (1961) have pointed out.

Thus, for example, diets high in Ca and relatively low in inorganic phosphate are rendered less rachitogenic by the addition of soluble phytates, which by immobilizing some of the Ca tend to make the phosphate more readily available and, if the Ca level is not too high, may provide additional phosphate from their own hydrolysis. Again, bone calcification is improved when calcium phytate or commercial 'phytin' (a Ca–Mg phytate) is added to a low-Ca diet adequate in vitamin D, and containing moderate amounts of phosphate (Mellanby, 1950). The influence of phytates on calcification depends, therefore, on the cations with which the phytates are associated, as well as on the levels of Ca, inorganic phosphate and vitamin D in the rest of the diet. The phytates extracted from the different cereals vary somewhat in composition and in their ability to precipitate Ca. They contain variable amounts of Ca, Mg, K and Mn (Ashton & Evans, 1962). All native phytates are, however, undersaturated with respect to divalent cations, and their ability to induce rickets on low-Ca diets depends on this fact.

The importance of the phytate content of diets in relation to the Ca and P requirements of animals is considerable. In formulating diets designed to make the most efficient use of the P in the native phytate present in the diet the aim should be to maintain the Ca level as low as possible, consistent with normal bone calcification, and to ensure that there is ample vitamin D in the diet. These principles have been recognized for at least 25 years but their application has led to many difficulties, and the steady stream of papers which continues to appear on this subject indicates that there is still no general agreement as to the extent to which P from plant sources can be relied upon to supply the requirements of different species at various ages. Most of these papers add nothing fundamental to what is already known concerning the availability of phytate P.

Summary and conclusions

(1) Phytates influence calcification in two main ways: by interfering with Ca absorption by forming insoluble calcium phytate and by failing to provide inorganic phosphate equivalent to their content of organic phosphate. In any particular diet they may act in one or both of these ways. Thus, rickets occurring in animals fed on cereal diets low in Ca is due to a deficiency of Ca and with diets high in Ca to a deficiency of P.

(2) The P of phytic acid and phytates may be made available by the action of plant phytases (optimum pH approx. 5) or by phytases present in the intestines (optimum pH approx. 8). There is no evidence that unhydrolysed phytate is absorbed from the gut.

(3) A phytate-splitting enzyme, probably a non-specific alkaline phosphatase, has been extracted from the intestines of several species.

(4) The significance of bacteria in hydrolysing phytate is in doubt, and experiments with germ-free animals are required to establish whether or not they are active in this respect.

(5) Phytases can only act on phytates in solution, and the extent to which phytates are hydrolysed depends largely on their solubility. This in turn depends on

the ions with which they are associated (mainly Ca^{2+}, Mg^{2+}, Mn^{2+}, K^{+} and H^{+} in natural phytates) and on the level of Ca in the diet. Thus, native phytates, free phytic acid, sodium and calcium phytates and commercial 'phytin' (a Ca–Mg phytate) do not necessarily behave in the same way.

(6) Vitamin D probably enhances the utilization of phytate P in two ways, by increasing the production of intestinal phytase and by stimulating Ca absorption, thus rendering the phytate more soluble. The requirements of animals for vitamin D increase as the proportion of the total dietary P as phytate increases.

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Bread and other foods of plant origin as a source of iron

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Iron deficiency is probably the commonest dietary deficiency in the world at the present time, and Fe-deficiency anaemia is an important problem in many countries. This is not altogether surprising as the average dietary intake of Fe in most countries is probably closer to, and more frequently lower than, recommended minimum levels, than is that of any other essential nutrient. Furthermore in times of national or domestic food shortage, some persons are likely to reduce their intake of Fe to a disproportionate degree, for example the elderly (Hobson & Pemberton, 1955), and married