# A review of PHYTASE in POULTRY DIETS

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Originally phytase was developed for use in the Netherlands to reduce phosphorus (P) pollution from intensive agriculture. The increase in raw material prices and growing concern for the environmental impact of meat production are the most often cited factors for the increase of enzyme use in poultry and swine production in the last 10 years. First commercialised in 1991 (Selle, 2007), phytase is now present in over 60% of monogastric feed worldwide (Graham, 2010) and even in a higher percentage in poultry diets.

Since first commercial utilisation, phytase has mainly been considered to be a tool to increase P availability/digestibility from vegetable sources, and so reduce the inclusion of higher cost P sources such as organic phosphates and animal by-products. Here, phytase releases the P bound in the phytate molecule (myo-inositol with six bound phosphate units, the main source of P in plant materials), increasing the availability/digestibility of this mineral to the animal (Onyango, 2005). Thus, increasing the inclusion rate of phytase would be expected to release additional P from the indigestible feed phytate and consequently allow an even greater substitution of higher cost P sources (Slominski, 2010; Ruiz, 2010).

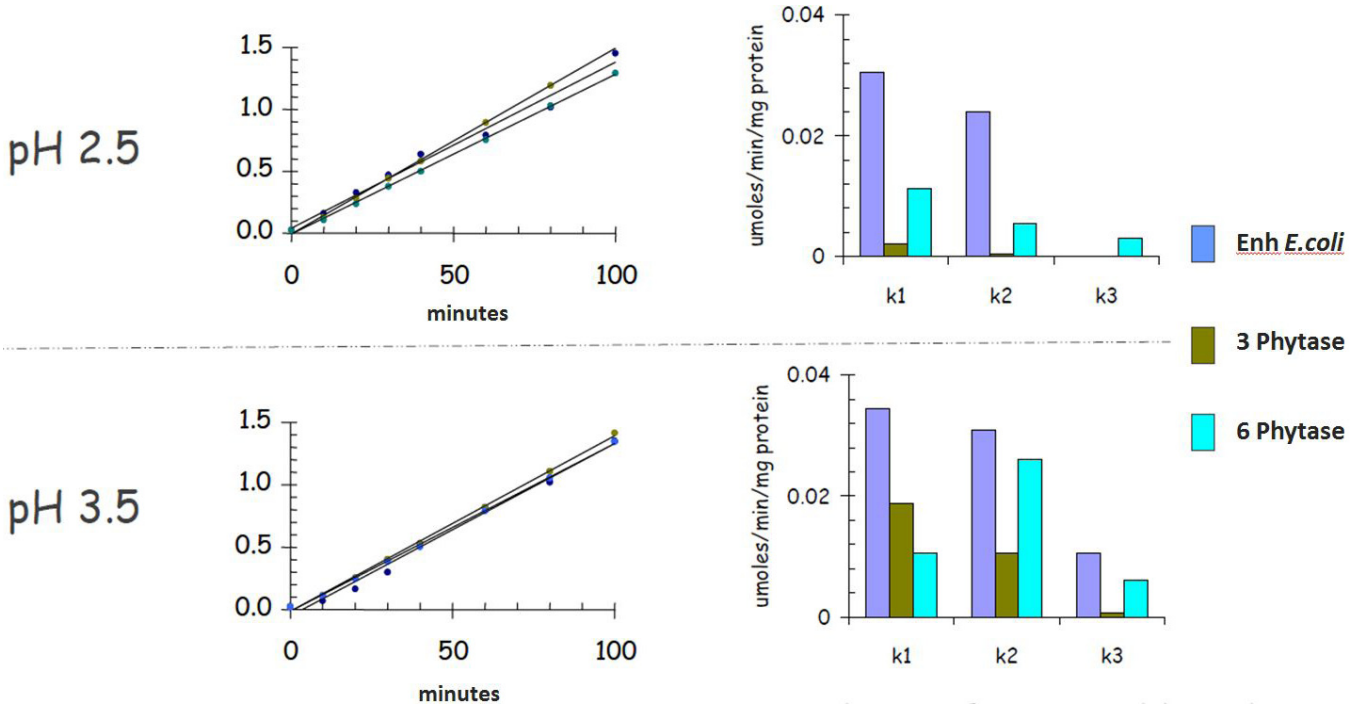
With different suppliers competing for a share of the phytase market, it is clear that product innovation and technical excellence is one successful strategy. Phytase products available on the market have thus changed substantially since the concept was first used commercially in the nineties. A clear evolution of products can be seen; there has been a move away from fungal origin products towards bacterial products and then subsequently to further modified bacterial products. These new products can deliver higher levels of P release per unit of activity, and are also capable of destroying a higher percentage of the phytate in the diet. These products are thus marketed with higher diet matrixes than were used traditionally, which poses the question whether there is always sufficient phytate available in the diet to allow the expected P-release from the given product and dose.

Measuring phytate in materials and diets has traditionally been quite difficult and thus expensive, but different analytical techniques have been developed. Recently it has been shown that it is possible to develop a NIR calibration for phytate (Flanagan, 2012). This technique is now routinely used to predict phytate levels in diets. It has been shown that there is substantial variability in phytate levels within and between materials (Santos & Bedford, 2012). Understanding actual phytate-P levels and variability in diets allows for maximal use of phytase in a safe manner. After all, even the best phytase in the world cannot release minerals that are not there in the first place! But, release of minerals is not the only thing phytase does, nor maybe the most important one, even though it is what the feed industry has concentrated on for a long time. When phytases act on the phytate molecule, releasing P, they also increase the solubility of the phytate while reducing its anti-nutritional effect.

Phytate is known to be an anti-nutrient, affecting an increase in mucus production and the endogenous loss of amino acids (Cowieson,

**Figure 1: Phytic acid precipitation at pH value above 4.0 (2mmol Phytic Acid + 30mmol Ca) – University of Maryland, unpublished-2006**

**Figure 2: Activity of three different enzymes measured by P release and affinity with IP6 (k1), IP5 (k2) and IP4 (k3). Prata, 2007**




2004), altering patterns of sodium (Na) secretion into the gut (Ravidran, 2008), and influencing the absorption of minerals such as calcium (Ca; Plumstead, 2008) and zinc (Schlegel, 2009). Part of the anti-nutritional effect of phytate is related to its link with minerals such as Ca at pH values higher than 4.0, reducing the availability of those minerals (and phytate-P) to absorption in the small intestine (Figure 1). Also, the link between phytate and proteins (usually between phytate and basic amino acids such as lysine when gut pH is less than the isoeletric point of proteins; Selle, 2007) can reduce protein digestibility. This results in increased amounts of intact protein in the small intestine, to which the animal reacts by increasing hydrochloric acid (HCl) and pepsin production. The presence of phytate in the diet has been shown to increase pancreatic juice and mucus production, which can be reduced back to normal levels by phytase addition. The increased mucus production may be a direct effect from the phytate (an irritant effect) or an indirect effect in response to the increased pepsin and HCl production. All this translates into an increased endogenous flow of amino acids and minerals to the gut and reduced Na reabsorption, also impairing membrane transporters (Liu, 2008).

To reduce the anti-nutritional effects of phytate, it is important to degrade the phytate molecules whilst they are soluble, which is in the lower pH part of the digestive tract. As the main anti-nutritional effect of phytate occurs when the molecule has six (IP6) or five (IP5) bound P units (Luttrell, 1993), the release of P is not necessarily completely correlated with the reduction of the anti-nutritional effects of phytate. Prata (2007) observed that three different phytases included at the same activity had different abilities to bind to and release P from phytates with six, five or four (IP4) bound P units (km). The phytase with higher affinity to phytate with six or five bound P units had a higher ability to reduce the anti-nutritional effects of the phytate (lower km), even when releasing the same amount of P (Figure 2).

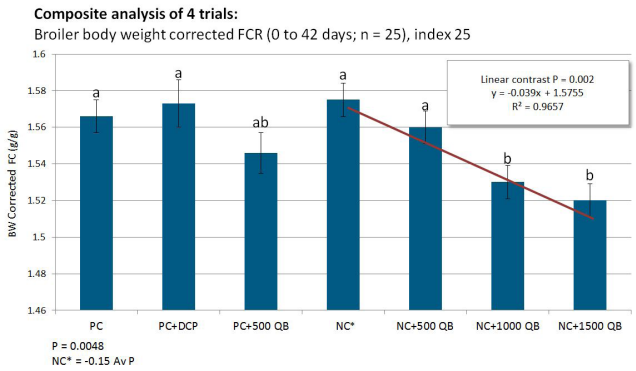
Considering this, to use a phytase focusing primarily on the reduction of the anti-nutritional effect of phytate, it is necessary to go beyond the release of P obtained by the enzyme. Initially a higher dose of phytase would have been considered to give an equivalent decrease in the need for supplemental inorganic P in the diet, looking for a further reduction in the cost of the diet while maintaining animal performance. However, an alternative approach is to look to eliminate the anti-nutritional effect of the phytate through higher enzyme doses, thereby increasing nutrient absorption and animal performance. Additionally, the possible absorption of the myo-inositol molecule and its utilisation as a component of animal metabolism may also give an increase in performance. As an example, Karadas (2010) provided high doses of phytase (12,500 FTU/kg) for broilers in a diet with a reasonable available P level (0.25%), and observed improved performance. Pirgozlev and co-workers (2009) observed not only higher performance but also higher content of hepatic carotenoids in poultry fed with higher doses of phytase.

Several trials with higher doses of phytase using diets with normal levels of P have already shown better poultry performance (Cowieson, 2006; Pirgozliev, 2007; Pirgozliev, 2008), but this improvement in performance was always correlated to an increase in P digestibility even if the diet did not have lower P levels. Interestingly, in all these trials the increase in performance with higher doses of phytase was correlated to an improvement in the feed conversion ratio (FCR). This is not common in trials where the dietary P levels are reduced,

where the main effect of phytase inclusion over the control is the recovery of feed consumption and body weight gain, but with no effect on feed conversion, as described in a holo-analysis published by Rosen (2004).

The concept of superdosing phytase, with the distinct aim of breaking down phytate and improving performance, has already been successfully implemented in piglet diets in the UK (Toplis *et al.*, 2010). Poultry trials have also been successful, and commercial implementation has followed in those markets where the most appropriate product for the concept is already available. Trials with this product in broilers have shown consistent improvements in FCR as the dose rate of phytase added to a negative (NC) diet increases from 500-1500 FTU/kg, as shown in Figure 3.

**Figure 3: Improved performance beyond 500 FTU with enhanced phytase**




The concept of using phytase at high doses to reduce the anti-nutritional effect of phytate means that there is an opportunity to redefine nutritional knowledge, since animal requirements to date have been determined in the presence of phytate and thus the anti-nutritional effects resulting from its presence. When using a high dose of phytase to reduce or eliminate the anti-nutritional effects of phytate, special attention needs to be paid to the choice of enzyme. Ideally this product needs to be active at low pH and have higher affinity for IP6 and IP5, the phytate molecules with higher anti nutritional effects. The overall diet formulation also needs to be considered as the absence of phytate will change other nutrient requirements, mainly minerals. Commercially it is suggested to include an enhanced *E. coli* phytase at 500 FTU/kg while taking into consideration its matrix values for minerals (P, Ca and Na) and then to add additional phytase to the diet to destroy phytate without making any further formulation changes. Using this approach, the matrix from the first 500 FTU/kg would already make an adjustment in the mineral content of the diet, helping reduce diet costs and ensuring a balanced supply of digestible minerals to the bird, while the extra enzyme will mainly destroy dietary phytate and boost poultry performance.

It can be concluded that the use of phytase in poultry diets has moved on considerably over the last few years, due to a better understanding of the negative effects of phytate and how best to develop and use phytase products.

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