The impact of phytate as an anti-nutrient in poultry diets

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The role of phytates in reducing the availability of phosphorus (P) within plant-based feed ingredients is widely recognised, with the use of phytase feed enzymes to release phytate-P and reduce feed costs almost ubiquitous. Yet the significant negative impact of phytate as an antinutrient in poultry diets is much less well understood, as are the substantial performance and financial gains that come from phytate elimination through use of higher doses of an appropriate phytase.

Phytate itself, also sometimes referred to as phytin or phytic acid, is the major storage form of P in seeds, and is part of a complex that also contains potassium, magnesium and calcium. Originally recognised as a key source of P during seed germination, the presence of phytate is now also known to play an important role in reducing oxidative stress during the germination process, preventing plant embryo death.

Phytate challenge

In animal nutrition, however, phytate has long posed a challenge. Initially viewed as an extremely valuable potential source of P that could be made available through targeted use of phytase feed enzymes, the focus in recent years has shifted to the antinutrient role phytate plays within the digestive tract.

This anti-nutrient effect is substantial, and clearly demonstrated by the significant reductions in performance seen in Fig. 1, where normal (high phytate) and genetically modified (low phytate) cereals were compared using 0 to 21-day-old broilers.

The phytate content in the unmodified grains increased feed conversion ratio (FCR) by 4.6% in the corn-based diets, and reduced bodyweight gain (BWG) in the barley-based diets by 5.0%.

There are several modes of action by which phytate has this negative effect, though all act to reduce digestibility and utilisation of important nutrients supplied in the diet. Key amongst these is the ability of the phytate molecule to bind with both proteins



Fig. 1. Bodyweight gain (BWG) and mortality-corrected feed conversion ratio (FCR) in 0 to 21-day-old broilers fed cereals with normal or low phytate contents.

and other minerals present in the intestinal tract, resulting not only in reduced availability, but also detrimental endogenous responses by the bird.

Mineral binding

Much of this binding effect can be attributed to the negative charge of the phytate molecule itself when in solution, even at the low pH found in the stomach and gizzard.

Further, this negative charge increases as the pH rises, thereby increasing phytate's ability to react with positively charged cations (primarily divalent cations such as calcium, zinc and copper) as it passes through the digestive tract from the acid stomach to the more neutral pH of the lower intestine.

The net result of these interactions is the formation of stable salts which then precipitate out of solution.

Even though phytate has a higher affinity for cations such as copper and zinc, it is the molecule's affinity for calcium that is of greatest concern in poultry nutrition. For example, it has been shown that the dietary calcium requirement for broilers increases from 0.60% to 0.95% when the phytate-P level in the diet increases from 0 to 0.25%, the latter being approximately equivalent to the phytate concentration found in a standard corn-soyabean diet.

The impact on other minerals can be seen when assessing the impact of progressive phytate reduction through application of increasing doses of phytase feed enzyme.

The results in Fig. 2, for example, show how yolk selenium content rises when the same diet is supplemented with a commercial phytase (Quantum Blue) at 700, 1400 and 2100 FTU/kg feed (the standard dose is 300 FTU/kg feed).

Protein impact

The reduction in protein digestibility caused by dietary phytate occurs through a different mode of action, with in vitro studies showing reduced pepsin activation between pH 0.8 and 2.8 in the presence of phytate.

Since the pH of the stomach contents is typically pH 2 to 3, it is likely that the subsequent reduction in pepsin activity results in less protein being initially broken down during the acid phase of the digestive process, leading to a lower overall protein digestibil-*Continued on page 17*



Fig. 2. Impact of phytate elimination on selenium nutrition in layers (Auburn University).



ity. Whilst this reduced pepsin activation may be overcome by higher production of its precursor, pepsinogen, in the stomach, phytate presence also directly reduces protein solubility and subsequent digestibility.

This occurs through feed proteins (positively charged at low pH) binding to phytate (negatively charged), and subsequently coprecipitating.

Finally, the presence of phytate can reduce the activity of the sodium/potassium 'pump', which is crucial to amino acid uptake across the gut wall.

Endogenous losses

The reduction in protein digestibility also has a further negative effect on the bird. The resulting rise in the level of undigested protein reaching the duodenum can increase secretion of the hormones gastrin and cholecystokinin, stimulating additional production of hydrochloric acid (HCI) and pepsinogen in the stomach whilst reducing gastric emptying.

The pH in the gizzard of broilers at seven and 21 days of age has been shown to be significantly lower when feeding a higher phytate diet, for example. The result is a substantial increase in endogenous losses. A greater amount of sodium bicarbonate has to be secreted into the duodenum to neutralise gut content pH, whilst the additional irritant effect on the gut mucosa leads to extra mucus production to maintain protection levels.

Phytate elimination

Perhaps the clearest indication of the impact on performance from the anti-nutrient effects of phytate comes from trials where phytate is effectively eliminated through phytase 'superdosing'.

The results in Fig. 3 are a composite of several trials evaluating bodyweight corrected FCR in broilers fed either a positive control (PC; full mineral supply), a negative control (NC; reduced mineral levels based on the standard phytase matrix) or the latter supplemented with progressively higher doses of an enhanced E. coli phytase (Quantum Blue).

Not all phytases are sufficiently active at the pH found in the stomach – or able to continue degrading phytate towards elimination as concentrations fall – to be effective for such superdosing.

However, the results in Fig. 3 clearly show



Fig. 3. Composite of broiler trials (0 to 35- or 42-days old) showing improvement in bodyweight corrected feed conversion ratio (FCR) from phytate elimination with higher levels of added Quantum Blue (QB) phytase.

the efficacy of Quantum Blue phytase in producing performance benefits beyond simple mineral supply (the standard dose equals the positive control performance) through phytate elimination.

Financial gains

The net effect is a four point improvement in FCR beyond that achieved by the positive control or the standard phytase dose, equivalent to a cost saving of \leq 4-6/tonne of feed. It is a gain that has far reaching implications, with estimates suggesting the anti-nutritional effects of phytate could be costing the global monogastric feed industry over \leq 2 billion per year.

As such, there is little doubt that as awareness of the negative impact of phytate on bird performance and profitability increases, so too will the use of high doses of phytase to achieve phytate elimination.

The arrival of commercial phytases like Quantum Blue specifically developed to maximise phytate destruction will also help to ensure end-users are able to achieve consistent results, a factor likely to be critical to the widespread uptake of superdosing and to reclaiming the revenue currently lost to the anti-nutrient phytate.