The Role of Vitamins and Feed Enzymes in Combating Metabolic Challenges and Disorders

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Primary Audience: Nutritionists, Researchers, Poultry Producers

SUMMARY

The potential exists for vitamins and feed enzymes to counteract some metabolic disorders and challenges in poultry. Recent research indicates that increased levels of vitamins E and C are able to reduce ascites-related mortality in broiler chickens. Fatty liver and kidney syndrome is a nutritionally induced metabolic disorder that has been eliminated under practical production conditions due to regular supplementation of poultry diets with biotin. There is also experimental evidence for beneficial effects of 25-hydroxycholecalciferol on the incidence and severity of skeletal and performance anomalies in broilers. Another challenge is the presence of high levels of indigestible and partly soluble nonstarch polysaccharides in many cereals and other plant feed ingredients, resulting in various antinutritive effects. The use of feed enzymes, in particular endo-1,3-1,4-β-glucanase and endo-1,4-β-xylanase, to counteract these adverse effects is well implemented by the feed industry. A typical example of metabolic challenge is the reduced availability of phytate P in plant feed ingredients and the potential for phytate to act as an antinutritional component. Microbial phytase provides a practical solution for improving P availability in plant ingredients. In addition, recent studies suggest phytate can stimulate endogenous losses, and phytase can help prevent those losses.

Key words: metabolic disorder, vitamin, metabolic challenge, microbial phytase, nonstarch polysaccharide-degrading enzyme


DESCRIPTION OF PROBLEM

The major objective of modern poultry nutrition is to fully utilize the genetic potential of birds by providing complete diets covering all known nutrient requirements according to the species, age, production category, and performance level. However, intensive production methods have resulted in a broad range of metabolic disorders, such as gizzard erosion, urolithiasis, electrolyte imbalance, fatty liver and kidney syndrome (FLKS), fatty liver hemorrhagic syndrome, pulmonary hypertension syndrome (ascites), sudden death syndrome, and various skeletal disorders, e.g., tibial dyschondroplasia. Furthermore, there are some obvious metabolic challenges that require the attention of poultry nutritionists when formulating practical diets. One typical example is the presence of high levels of indigestible and partly soluble nonstarch polysaccharides (NSP) in many cereals and other plant feed ingredients that result in various antinutritive effects, mainly in young poultry. Another example is the reduced availability of P from plant feed ingredients for poul-
try and other monogastric farm animals, because its greater part is present in the nonavailable phytate form, which cannot be utilized due to the lack of the relevant enzyme in their gastrointestinal tract.

The objective of this paper is to review possible use of vitamins and feed enzymes to counteract some metabolic disorders and challenges.

**ASCITES AND VITAMINS E AND C**

Mortality in broiler chickens associated with fluid accumulation in the abdominal cavity (ascites) is the ultimate consequence of an excessively high blood pressure in the pulmonary circulation known as pulmonary hypertension syndrome (PHS). The symptoms are a generalized edema, hydropericarditis, ascites, hypertrophy and dilatation of the heart, and, in particular, hypertrophy of the right ventricle [1]. Ascites is a metabolic disorder that is no longer restricted to high altitudes [2]. This syndrome is caused by an imbalance between O2 supply and its requirement to sustain the fast growth rate and high feed efficiency [1, 3]. It clearly has a multifactorial etiology, with several nutritional factors such as feed structure and diet composition showing a major effect on the incidence.

Potential involvement of free radicals and antioxidant mechanisms in the pathogenesis of PHS was hypothesized by Bottje and Wideman [4]. Enkvetchakul et al. [5] found that lower pulmonary and hepatic α-tocopherol and glutathione levels were found in broilers exhibiting PHS experimentally induced by low ventilation, indicating that the antioxidant status of these birds was compromised. Bottje et al. [6] found that attenuation of PHS mortality induced by low ventilation conditions was achieved by implanting birds with a vitamin E pellet that released a total of 15 mg of α-tocopherol from 0 to 21 d of age. In contrast, in another study, Bottje et al. [7] found that dietary supplementation with α-tocopherol acetate up to 87 mg/kg was ineffective in lowering PHS mortality induced by cool temperature in combination with low ventilation conditions. Later, Roch et al. [8] demonstrated that an increased dietary vitamin E level (250 ppm), in combination with an organic Se source, was able to significantly reduce the mortality associated with PHS in cold stressed broiler chickens, suggesting that a level of vitamin E higher than 87 mg/kg may be necessary to decrease the incidence of PHS.

Supplemental vitamin C at 150 to 450 ppm has also been shown to reduce the incidence of ascites in broilers caused by feeding extremely high dietary levels of NaCl [9]. In other studies [10, 11], dietary vitamin C at 500 ppm reduced PHS mortality induced by cool environmental temperatures and feeding of a thyroid hormone. Across several model systems, there is increasing evidence that higher dietary levels of vitamins E and C are able to reduce ascites-related mortality in broiler chickens. However, due to a multifactorial etiology of this metabolic disorder, further research is needed to confirm this beneficial effect under more practical conditions.

**FLKS AND BIOTIN**

The history of FLKS in broiler chickens provides an excellent example of the importance of optimum vitamin supplementation of commercial poultry diets. About 40 yr ago, FLKS was described in young chickens in several countries (Denmark, United Kingdom, Australia, Canada) and was characterized by pale, enlarged fatty livers with or without hemorrhages, hydropericardium, and pale, swollen kidneys [12, 13]. It became apparent from practical experience that FLKS occurred mainly in countries where wheat is used as the predominant carbohydrate source in commercial poultry diets. Outbreaks of FLKS with an increased mortality in broiler flocks to 10 to 20% were reported [14, 15]. Further research demonstrated that FLKS is a typical metabolic disorder of young chickens fed biotin-deficient diets, being mainly related to the reduced bioavailability of this vitamin in wheat [16, 17]. The syndrome is characterized by an accumulation of lipids in the liver, kidney, and other organs [12] and a severe hypoglycemia, which seems to be the cause of death [18]. A major cause of the hypoglycemia is failure of hepatic gluconeogenesis, caused by very low activity of pyruvate carboxylase, a biotin-dependent enzyme. It has been demonstrated that its incidence can be reduced by increasing the level of dietary fat [19] or protein [20] and eliminated by supplementing the diet with biotin [15, 16, 17]. Whitehead et al. [17] found that the minimum levels of supplementary dietary biotin required to prevent mor-
tality varied from 50 to 150 μg/kg, depending on the diet. Payne et al. [16] also suggested that dietary biotin levels in breeder diets may affect the incidence of FLKS in their progeny. Whitehead and Blair [20] confirmed that supplementing the breeder diet with biotin reduced FLKS mortality among progeny during the first month after hatching, but not in later stages.

It can be concluded that FLKS is a nutritionally induced metabolic disorder, caused mainly by biotin deficiency. Due to regular supplementation of commercial poultry diets with this important vitamin, this disorder is not as common under practical production conditions.

**SKELETAL DISORDERS AND VITAMIN D₃ METABOLITES**

Classical skeletal disorders, such as rickets in young birds or cage layer fatigue in laying hens, caused by a reduced or imbalanced supply of Ca, P, and vitamin D₃ have become less critical under practical production conditions. On the other hand, some other disorders related to increasing stress factors and continuously growing performance of modern genotypes are a continuing concern for the poultry industry worldwide, being responsible for production losses as well as for poor welfare. Tibial dyschondroplasia (TD) is a widespread abnormality found in rapidly growing meat-type poultry [21, 22]. It is characterized by the persistence of cartilage below the growth plate. The TD lesion arises from a failure of growth plate chondrocyte differentiation, which results in an accumulation of prehypertrophic cells [23]. The cause of TD is believed to be a genotype and nutrition interaction that can be influenced by other factors such as heat stress or mycotoxins.

Experimental studies have shown that addition of the active vitamin D₃ metabolite 1,25-dihydroxycholecalciferol to the diet of young, growing broiler chickens can result in a dramatic decrease of the incidence of TD in a dose-dependent manner [24, 25, 26], with complete protection being afforded by a dietary supplement of 5 μg/kg of diet [26]. However, 1,25-dihydroxycholecalciferol is a potent calcitropic hormone involved in Ca and phosphate homeostasis. It is a very expensive compound with a limited safety margin, used for many years as a drug for various human maladies, thus unavailable as a feed additive. Some years ago, 25-hydroxycholecalciferol (25-HCC), the primary plasma metabolite of vitamin D₃ produced by hydroxylation in the liver, was developed and launched as a feed additive for poultry nutrition. It has been suggested that 25-HCC could be used as an alternative to dietary supplementation with vitamin D₃, resulting in improvements in weight gain and feed conversion efficiency when used at 50 to 70 μg/kg of diet [27, 28]. More recent research has demonstrated that the use of dietary 25-HCC can reduce the incidence and severity of TD in broilers [23, 29, 30, 31]. Rennie and Whitehead [23] observed that replacing 75 μg of cholecalciferol/kg of diet with the same concentration of 25-HCC significantly reduced the incidence of TD from 65 to 10%. In another experiment, Rennie and Whitehead [23] noted that the incidence of TD in the control group was lower, but dietary addition of 25-HCC up to 250 μg/kg of diet showed a linear beneficial effect of the incidence of TD. These observations were corroborated by the findings of Fritts and Waldroup [29] that the incidence and severity of TD was significantly lower in birds fed 25-HCC when compared with the same levels of cholecalciferol. In contrast, Bar et al. [32] did not find any difference between conventional vitamin D₃ source and 25-HCC concerning the severity or frequency of TD. Ledwaba and Roberson [30] reported that dietary 25-HCC decreased the incidence of TD similarly at 40 and 70 μg/kg when a TD-inducing diet was fed. Its effectiveness in young broilers was also higher when the dietary Ca level was below 0.85%. Parkinson and Cranberg [31] demonstrated again that 25-HCC...
added at 69 μg/kg to the conventional broiler diet containing vitamin D₃ (75 μg/kg) can reduce the incidence of TD in young chickens.

It can be concluded that dietary administration of 25-HCC can effectively reduce the incidence and severity of TD in broiler chickens, but additional research efforts are required to evaluate other interacting dietary or genetic factors. Additionally, BW and feed conversion improvements can be elicited by 25-HCC when TD does not appear to be present.

USE OF FEED ENZYMES TO COUNTERACT ADVERSE EFFECTS OF NONSTARCH POLYSACCHARIDES

In poultry nutrition, it is accepted that the nutritive value of many cereals, such as barley, triticale, rye, wheat, and various grain legumes, is adversely affected by the presence of nonstarch polysaccharides (NSP), e.g., mixed linked β-D-glucans or arabinoxylans, in their endosperm cell walls. An important characteristic of these NSP is their partial solubility in water, resulting in the formation of viscous gel solutions. This results in a dramatic increase in the viscosity of intestinal digesta, particularly in young poultry, which may impair the action of digestive enzymes, decrease the rate of passage, and interfere with the absorption of nutrients. In addition, there is also a hypothesis that the presence of indigestible plant cell wall fraction in the digestive tract may reduce the access of digestive enzymes to cell contents. As a consequence, digestibility of nutrients and utilization of dietary energy may be markedly reduced. These antinutritive effects of NSP in poultry are well described in the literature [33, 34, 35, 36]. In the case of cereals, a strong negative correlation between the content of NSP and the metabolizability of dietary energy was clearly demonstrated by Choct and Annison [33] (see Figure 1). More information about the average levels of NSP present in cereals, as well as in some relevant legume grains, is presented in Table 1 [37].

To enhance the nutritive value of various low-energy cereals, specific enzyme products have been developed that are able to partly degrade the relevant NSP. Endo-1,3(4)-β-glucanase (EC/IUB 3.2.1.6), endo-1,4-β-xylanase (EC/IUB 3.2.1.8), or both appear to be the crucial enzymes responsible for beneficial effects. At present, a wide spectrum of NSP-degrading enzyme products are routinely used by the feed industry in Europe and Canada to enhance the feeding value of wheat- and barley-based diets. More than 55 products have been approved in the European Union, which can be divided into 4 groups:

- Enzyme complexes derived from single strains (e.g., Trichoderma longibrachiatum, Trichoderma viride, Aspergillus niger, Humicola insolens).
- Enzyme mixtures based on 2 or more fermentation products.
- Monocomponent enzymes derived from genetically modified strains.
- Combinations of an enzyme complex and monocomponent enzyme.

The use of such NSP-degrading enzymes is commonly integrated in practical poultry nutrition. The beneficial effects on performance, as well as the mechanisms of action, have been adequately described in various review papers [38, 39, 40, 41, 42]. When added to relevant poultry diets, NSP-degrading enzymes usually result in numerous beneficial effects, such as increased utilization of nutrients (e.g., fat, protein), improved AME values, increased growth rate, improved feed:gain, decreased viscosity of intestinal digesta, reduced incidence of sticky excreta, and improved litter conditions. Published beneficial effects of enzyme supplementation on AME values of wheat in young broiler

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Soluble NCP</th>
<th>Insoluble NCP</th>
<th>Cellulose</th>
<th>Total NSP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat</td>
<td>25</td>
<td>74</td>
<td>20</td>
<td>119</td>
</tr>
<tr>
<td>Barley (hulled)</td>
<td>56</td>
<td>88</td>
<td>43</td>
<td>186</td>
</tr>
<tr>
<td>Rye</td>
<td>42</td>
<td>94</td>
<td>16</td>
<td>152</td>
</tr>
<tr>
<td>Oats (hulled)</td>
<td>40</td>
<td>110</td>
<td>82</td>
<td>232</td>
</tr>
<tr>
<td>Corn</td>
<td>9</td>
<td>66</td>
<td>22</td>
<td>97</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>63</td>
<td>92</td>
<td>62</td>
<td>217</td>
</tr>
<tr>
<td>Peas</td>
<td>52</td>
<td>76</td>
<td>53</td>
<td>181</td>
</tr>
<tr>
<td>White lupins</td>
<td>134</td>
<td>139</td>
<td>131</td>
<td>404</td>
</tr>
</tbody>
</table>

1NCP = noncellulosic polysaccharides.
Table 2. Effects of enzyme supplementation on AME values of wheat

<table>
<thead>
<tr>
<th>Cereal characteristics</th>
<th>AME value (MJ/kg of DM)</th>
<th>Improvement (%)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>− Enzyme</td>
<td>+ Enzyme</td>
<td></td>
</tr>
<tr>
<td>Low AME wheat</td>
<td>12.02</td>
<td>14.94*</td>
<td>+24.3</td>
</tr>
<tr>
<td>Normal AME wheat</td>
<td>14.52</td>
<td>14.83</td>
<td>+2.1</td>
</tr>
<tr>
<td>13 wheat cultivars</td>
<td>13.23</td>
<td>14.36**</td>
<td>+8.6 (+4.5 − 12.4)</td>
</tr>
<tr>
<td>Wheat (cultivar Ibis)</td>
<td>14.76</td>
<td>14.94</td>
<td>+1.2</td>
</tr>
<tr>
<td>Wheat (cultivar Alidos)</td>
<td>13.98</td>
<td>14.65*</td>
<td>+4.8</td>
</tr>
<tr>
<td>Wheat</td>
<td>12.35</td>
<td>13.91</td>
<td>+12.6</td>
</tr>
<tr>
<td>Wheat (cultivar Alba)</td>
<td>13.78</td>
<td>14.37*</td>
<td>+4.3</td>
</tr>
<tr>
<td>Wheat (cultivar Sirvinta)</td>
<td>13.29</td>
<td>14.24*</td>
<td>+7.1</td>
</tr>
</tbody>
</table>

1Bioassays conducted with young broiler chickens.  
*P < 0.05; **P < 0.01.

Grain legumes are usually used in poultry diets as important protein sources. However, when some legumes such as lupins are included in poultry diets as the sole protein source, bird performance suffers due to high levels of NSP and the oligosaccharides present [42]. Legume NSP are much more complex in structure than those present in cereals, and, therefore, the use of “classical” NSP-degrading enzyme products tends to provide limited and inconsistent responses. In contrast to cereals, legumes contain a mixture of colloidal polysaccharides called pectic substances (galactouronans, galactans, and arabinans) and neutral polysaccharides such as xyloglucans and galactomannans [42]. Furthermore, their concentration is much higher than in the case of cereals (see Table 1). In addition, they also contain a high level of nondigestible oligosaccharides (about 9%), such as verbascose and stachyose, but almost no starch. In recent years, some beneficial effects of NSP-degrading enzymes on the nutritive value of peas and particularly lupins for broiler chickens have been reported in the literature [48, 49, 50, 51, 52, 53, 54]. However, it is obvious that total depolymerization of such complex NSP like those occurring in legume seeds requires extremely complex enzyme activities, thus presenting challenges to develop enzyme products that are effective and economical.

Soybean meal is traditionally the major vegetable protein source in poultry diets. Like other grain legumes, the carbohydrate fraction of soybean meal includes only traces of starch, relatively high amounts of galactooligosaccharides, and some free sucrose. The substantial portion of NSP is in the cell walls [55]. Thus, due to its carbohydrate composition, AMEn values of soybean meal for poultry are relatively low (<2,400 kcal/kg). The total content of NSP in soybean meal may vary from 18 to 22%, but water soluble NSP represent only a smaller part and the remainder is insoluble [37, 55, 56]. Raw soybeans contain various antinutritive factors, e.g., trypsin inhibitors, lectins, saponins, that are usually destroyed during the processing of soybean meal. Potential improvements in the feeding value of soybean meal for poultry have attracted extensive research efforts. Several concepts involving relevant enzymes have been reported recently [40, 57]. These experimental approaches include the use of α-galactosidase, β-galactanase or a pectinase complex, β-mannanase, combination of xylanase and cellulase, or various proteases. Despite some beneficial, but still limited effects, a real breakthrough has not been achieved. More research is required to identify enzymes that substantially enhance the nutritive value of this important feed ingredient.

USE OF PHYTASE TO COUNTER ADVERSE EFFECTS OF PHYTATE

In feedstuffs of plant origin, the greater proportion of the total P (60 to 80%) is present in the phytate form, which is practically unavailable to nonruminant animals, especially poultry. The typical total and phytate P levels in some feed ingredients in poultry diets are summarized in...
Table 3. Total and phytate P content in some feed ingredients [58]

<table>
<thead>
<tr>
<th>Feed ingredient</th>
<th>Total P (%)</th>
<th>Phytate P (%)</th>
<th>Phytate P (% of total P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat</td>
<td>0.33</td>
<td>0.22</td>
<td>67</td>
</tr>
<tr>
<td>Barley</td>
<td>0.37</td>
<td>0.22</td>
<td>60</td>
</tr>
<tr>
<td>Rye</td>
<td>0.36</td>
<td>0.22</td>
<td>61</td>
</tr>
<tr>
<td>Maize (corn)</td>
<td>0.28</td>
<td>0.19</td>
<td>68</td>
</tr>
<tr>
<td>Peas</td>
<td>0.38</td>
<td>0.17</td>
<td>46</td>
</tr>
<tr>
<td>Rapeseed meal</td>
<td>1.12</td>
<td>0.40</td>
<td>36</td>
</tr>
<tr>
<td>Soybean meal (44% CP)</td>
<td>0.66</td>
<td>0.35</td>
<td>53</td>
</tr>
<tr>
<td>Soybean meal (48% CP)</td>
<td>0.61</td>
<td>0.32</td>
<td>52</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>1.16</td>
<td>0.97</td>
<td>84</td>
</tr>
<tr>
<td>Rice bran</td>
<td>1.71</td>
<td>1.10</td>
<td>64</td>
</tr>
</tbody>
</table>

Table 3 [58]. The limited availability of P from plant feed ingredients represents a well known challenge to poultry nutritionists. A classical solution to this problem is the addition of various inorganic phosphates, e.g., dicalcium phosphate, to compound feeds. Phytic acid (PA; chemically myoinositol 1,2,3,4,5,6-hexakis dihydrogen phosphate) represents a storage form of P in plants. The molecule of phytic acid has a relatively high P content (28.2%), and its 6 phosphoric acid residues have various affinities to several cations. Minerals and trace elements, such as Ca, Mg, Fe, and Zn, are frequently bound with phytic acid, and these salts are described as phytates.

The phytate-bound P must be hydrolyzed before it is utilized by poultry. Phytase is a specific kind of phosphatase that catalyzes the stepwise removal of inorganic orthophosphate from phytate. However, only negligible endogenous phytase exists in intestinal mucosa [59]. In contrast to animals, phytases are known to occur widely in microorganisms and plants, either as 3-phytase (EC/IUB 3.1.3.8), which splits the phosphate groups at the C$_3$ atom first, or as 6-phytase (EC/IUB 3.1.3.26), which acts first at the C$_6$ atom and is the only phytase activity present also in plants. However, the practical relevance of such native phytase activity is limited, because the pelleting of feeds utilizes temperatures that can completely destroy the phytase.

Progress in genetic engineering of microbial phytases has become available for practical applications at costs increasingly more favorable than the cost of inorganic phosphate. Some products contain 3-phytase derived from various Aspergillus spp., which have been expressed in genetically modified Aspergillus or Trichoderma production strains. Another product contains 6-phytase derived from Peniophora lycii, which is manufactured by a genetically modified strain of Aspergillus oryzae.

The efficacy of supplemental phytases in broiler chickens, laying hens, and turkeys is well documented in the literature [60, 61, 62, 63, 64, 65, 66, 67, 68, 69]. Main effects of phytase addition may be summarized as follows:

- Improved availability and utilization of phytate P, which can be well demonstrated by improved ash percentage in bones.
- Improved availability of Ca, Zn, and amino acids.
- Enhancement of performance parameters when compared with respective controls receiving low-P diets.
- Reduction of P excretion and subsequently decreased environmental pollution.

New bacterial phytases derived from Escherichia coli and expressed in various yeasts have been developed, and their efficacy in poultry has been described [70, 71, 72, 73, 74, 75, 76]. Substrate specificity is an essential and an important characteristic concerning further innovation, to increase in vivo efficacy of new phytases. The achievement of the complete release of phytate P becomes of greater importance as the relative price of phytase to phosphate becomes more desirable and environmental concerns become more acute.

Research efforts in recent years have focused on the isolation and development of new, heatstable microbial phytases from other microbial sources. Improvements in pelleting stability of phytase preparations are achieved by specific formulation [77, 78, 79] or by modifying the phytase molecule [80]. In commercial application of phytases in pelleted feeds, the heat stability plays a pivotal role in the ultimate successful use of that phytase.

Using A. niger, Ravindran et al. [81] noted that the improvement in Thr digestibility was often highest among the amino acids tested in several ingredients, a phenomena observed elsewhere [82, 83, 84]. Rutherfurd et al. [85] also found that P. lycii phytase increased the ileal
digestibility of amino acids in mixed diets and across several ingredients [86], and Thr digestibility was usually improved the most.

Phytic acid has been theorized to stimulate endogenous protein losses [81, 84], in part, because intestinal mucin contains a relatively high concentration of Thr for both poultry and swine [87, 88]. In pigs, for example, Thr comprises 28 to 35% of the protein portion of mucin [89]. Produced by goblet cells throughout the small intestine [90], this glycoprotein functions as a “living” protective barrier between intestinal contents and the absorption system, and its disruption could marginalize the uptake of nutrients [91]. Increased losses of endogenous protein in digesta and feces due to antinutritional factors can be interpreted as a reduction in apparent digestibility of feedstuffs and amino acids and especially Thr, according to Montagne et al. [89].

Cowieson et al. [92, 93] showed PA to increase endogenous losses of proteins and carbohydrates in precision-dosed broilers. The ingestion of 1 g of PA significantly increased the excretion of DM (+28%), N (+21%), nonprotein N (+19%), and sialic acid (+185%) compared with birds dosed with glucose. Sialic acid is a component of mucin and serves as a marker for mucin. About 82% of the PA was found in the feces in control birds, but this was decreased (P < 0.001) to 47% with phytase [93]. Amino acid excretion with PA was increased (P < 0.05) from 11 to 60%. Over the 48-h collection period, approximately 90 mg of Lys was excreted when PA was dosed by itself, but with the inclusion of phytase, Lys excretion was reduced to 21 mg [93]. The excretion of essential amino acids with PA was lower (P < 0.05) when phytase was present, consistent with the concept that PA was binding to Lys and preventing it from being absorbed.

Phytase potentially could increase amino acid digestibility and ME, not only by eliminating potential binding to these components through its ability to degrade phytate, but also by reducing endogenous losses [94, 95]. In this manner, phytase could play a significant role in the metabolic challenges imposed by ingredients as innocuous as corn.

CONCLUSIONS AND APPLICATIONS

1. Pulmonary hypertension syndrome (ascites) has a multifactorial etiology, apparently involving free radicals and antioxidant mechanisms in the pathogenesis. There is experimental evidence that higher dietary levels of vitamins E and C are able to reduce ascites-related mortality in broiler chickens.

2. Fatty liver and kidney syndrome is a nutritionally induced metabolic disorder, caused mainly by biotin deficiency. Due to regular supplementation of commercial poultry diets with this vitamin, this syndrome has been eliminated under practical production conditions.

3. Tibial dyschondroplasia is a widespread abnormality occurring in rapidly growing meat-type poultry. Dietary administration of 25-hydroxycholecalciferol may offer an effective measure to reduce the incidence and severity of this skeletal disorder in broiler chickens.

4. Nutritive value of many cereals and grain legumes is adversely affected by the presence of NSP. The antinutritive effects of NSP can be counteracted and partly eliminated by supplementation of poultry diets with relevant NSP-degrading enzyme products.

5. Reduced availability of P in plant feed ingredients for poultry represents another challenge for nutritionists when formulating practical diets.

6. Metabolic challenges could encompass ingredients such as corn through the effects of phytate on endogenous losses, and phytase could play a meaningful role in this venue.

REFERENCES AND NOTES


