

## VITAMIN AND TRACE MINERAL SUPPLEMENTATION 2010 REVISED GUIDELINES

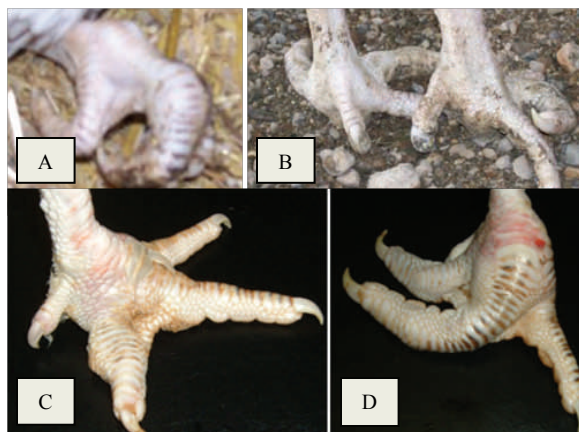
Over the past 10 years (2000-2010), genetic selection has improved the efficiency of feed utilization of Hybrid turkeys by approximately 1% each year. Because of this genetic improvement, it is necessary at this time to re-evaluate the vitamin and trace mineral supplementation guidelines. The guidelines for vitamin and trace mineral supplementation are revised for 2010 for both parent and commercial stock and are available at [www.hybridturkeys.com](http://www.hybridturkeys.com).

There is very little research done on vitamin nutrition of turkeys over the past 10 years. Prior to 2000, there is published nutrition research on turkeys for vitamin A, vitamin D<sub>3</sub>, vitamin E and biotin; but there is very little research on other vitamins. For this reason, the changes in the supplementation guidelines for vitamins are based upon genetic improvements in growth rate and feed efficiency, experience with customers, changes in feed processing techniques and industry data obtained from vitamin suppliers. The changes made to the vitamin and trace mineral guidelines compared to the previous guidelines for parent and commercial stock are presented in Tables 1 and 2, respectively. All the vitamin supplementation levels have been increased except for thiamine, pyridoxine and vitamin B<sub>12</sub>. The majority of the increases in vitamin supplementation are 20% or less compared to the previous guideline levels. Based upon our experience in the field, it was not necessary to change the trace mineral supplementation guidelines from those in the previous guidelines.

To better assist our customers in developing their own vitamin and trace mineral premixes, recommended sources of vitamins and trace minerals are included in the revised guidelines. Vitamin quality can differ by manufacturer and the chemical form of the vitamin. The cheapest vitamin source available may not necessarily be the most economical for inclusion into turkey feed. Trace mineral ingredient selection also requires careful scrutiny because chemical sources differ in bioavailability and may contain undesirable contaminants such as heavy metals, PCB's and dioxins. For those vitamins and trace minerals where multiple ingredient sources are listed in the revised guidelines; either a single or multiple ingredient source of that vitamin or trace mineral can be used to formulate the premix.

The revised vitamin and trace mineral guidelines also contain information on minimum allowable vitamin recovery analyzed post heat- or chemical-processing of complete feed. Many feed manufacturers use excessive heat processing to eliminate bacterial contamination of feed.

Unfortunately, many of these heat processing methods also destroy vitamins and other nutrients. Vitamin deficiencies can occur as a result of excessive heat processing of feed, even though the vitamin supplementation of the feed prior to heat processing was adequate. When problems have occurred in turkeys fed excessive heat processed feed, it is usually due to one or a combination of B-complex vitamins being deficient in feed. This can result in serious feet and footpad problems (*Figures 1 and 2*).



**Figure 1:** Examples of feet deformities associated with B-vitamin deficiencies, most notably, pantothenic acid, riboflavin, niacin and biotin.



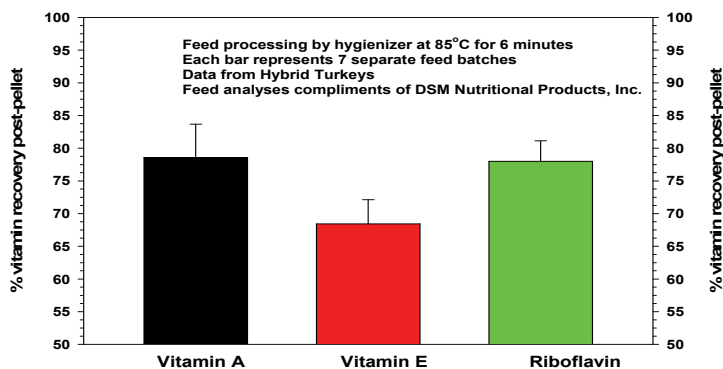
**Figure 2:** Footpad dermatitis associated with biotin deficiency.

Destruction of B-complex vitamins can also result in feathering abnormalities in growing poult (Figure 3).



**Figure 3:** Rough feathering associated with B-vitamin deficiency, notably, pantothenic acid, biotin, pyridoxine, and vitamin B<sub>12</sub>.

Routine determination of vitamin recovery subsequent feed processing is essential to prevent excessive vitamin destruction from affecting turkey production. For such a determination, analyzing feed for all vitamins would be impractical and prohibitively costly. Selecting vitamin A, vitamin E and riboflavin as in Figure 4 to assess post heat-processing recovery can provide information on the need of further vitamin supplementation as a result of excessive heat processing of feed.

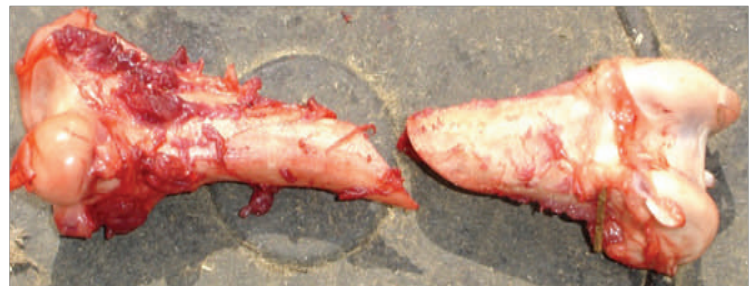


**Figure 4:** Example of vitamin recovery assessment done in heat-processed feed.

Using Figure 4 as an example, riboflavin recovery was 78%. Using the information in the revised vitamin and trace mineral guidelines, the minimum allowable riboflavin recovery is 84%. Adjusting the Hybrid guideline for riboflavin upwards by at least a further 7.7% ( $84 \times 100/78 = 7.7\%$ ) should be considered to prevent excessive vitamin destruction from affecting turkey production. As other B-complex vitamins were not analyzed, they

should be considered to be increased at least similarly to riboflavin. Similar calculations can be done for vitamins A and E, using their recovery data (Figure 4), to determine how much further supplementation above Hybrid guidelines is required for these vitamins. Recovery data on vitamins A and E can be used to estimate whether any increases in vitamins D<sub>3</sub> or K supplementation are required. Overall, the vitamin recovery information provided in the Hybrid vitamin and trace mineral supplementation guidelines are to be used by the nutritionist as an aid to determine if additional vitamin supplementation is required because of excessive heat processing of feed.

Early deficiencies of vitamins or trace minerals in turkeys can cause problems that may only be noticed several weeks later. An example of this is spiral fracture of the femur in heavy toms 17 weeks of age or older (Figure 5).



**Figure 5:** Spiral fracture of the femur in heavy toms >17 weeks of age. Biomechanical stressors placed on the bone resulting in spiral fracture could be caused by much earlier nutritional deficiencies. Suspect nutrients could include niacin, biotin, folic acid, vitamin B<sub>12</sub>, choline, copper, zinc and manganese.

Although the exact cause of this problem is not well understood, it is possible that nutritional deficiencies as early as the first 6 weeks of age could cause abnormal development of the femur. This can result in the bone fracturing at a much later age due to the additional stress that heavier bodyweights place on that bone at that time.

The metabolic functions and deficiency symptoms of vitamins are detailed in Table 3. Because of the lack of data with turkeys, some of the information in Table 3 is extrapolated from other poultry species. Vitamins and trace minerals are essential for the growing poult, and breeding hens and toms. They are also required for the proper development of the embryo in the egg and for the expression of the poult's full genetic potential subsequent hatch.

**Table 1:** Comparison of 2010 Hybrid vitamin trace-mineral supplementation guidelines to the previous guidelines: Parent Stock.

Nutrient	% Difference from Previous Hybrid Guidelines		
	0-42 days	43-210 days	>211days
<i>Vitamin A</i>	20	20	20
<i>Vitamin D<sub>3</sub></i>	0	20	0
<i>Vitamin E</i>	0	20	33
<i>K<sub>3</sub> (Menadione)</i>	0	50	25
<i>Thiamine</i>	0	0	0
<i>Riboflavin</i>	0	20	20
<i>Pantothenic Acid</i>	12	15	20
<i>Niacin</i>	10	6	10
<i>Pyridoxine</i>	0	0	0
<i>Biotin</i>	0	13	0
<i>Folic acid</i>	40	25	80
<i>Vitamin B<sub>12</sub></i>	0	0	0
<i>Choline</i>	0	0	0
<i>Manganese</i>	0	0	0
<i>Iron</i>	0	0	0
<i>Copper</i>	0	0	0
<i>Iodine</i>	0	0	0
<i>Zinc</i>	0	0	0
<i>Selenium</i>	0	0	0

**Table 2:** Comparison of 2010 Hybrid vitamin trace-mineral supplementation guidelines to the previous guidelines:  
Commercial Stock

Nutrient	% Difference from Previous Hybrid Guidelines		
	0-42 days	43-98 days	>99 days
Vitamin A	20	20	14
Vitamin D <sub>3</sub>	0	20	14
Vitamin E	0	20	20
K <sub>3</sub> (Menadione)	0	50	25
Thiamine	0	0	0
Riboflavin	0	20	25
Pantothenic Acid	12	15	13
Niacin	10	6	10
Pyridoxine	0	0	0
Biotin	0	13	6
Folic acid	40	25	0
Vitamin B <sub>12</sub>	0	0	0
Choline	0	0	0
Manganese	0	0	0
Iron	0	0	0
Copper	0	0	0
Iodine	0	0	0
Zinc	0	0	0
Selenium	0	0	0

**Table 3:** Metabolic function and deficiency symptoms of vitamins and trace minerals.

Nutrient	Metabolic Function	Deficiency Symptoms
<i>Vitamin A</i>	<ul style="list-style-type: none"> <li>• Vision</li> <li>• Cell differentiation</li> <li>• Development and integrity of epithelia including respiratory system, digestive tract and reproductive system</li> <li>• Skeletal development</li> <li>• Immune cell function, differentiation and intercellular communication</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of appetite, growth and feed efficiency</li> <li>• Impaired immune function, increased susceptibility to disease</li> <li>• Dry and scaly skin, rough feathering</li> <li>• Night blindness, corneal cloudiness and retinal degeneration</li> <li>• Eye and nasal discharge, creamy pustules in the mouth and esophagus</li> <li>• Excessive urate production</li> <li>• Metaplasia of respiratory epithelia resulting in increased risk of infection</li> <li>• Keratinization of digestive system epithelia, poor nutrient absorption</li> <li>• Cystic pituitary glands</li> <li>• Decreased male reproductive performance including testicular degeneration, reduced numbers and motility of spermatozoa, increased numbers of abnormal shaped spermatozoa</li> <li>• Ovary degeneration resulting in reduced egg production, fertility and hatch</li> </ul>
<i>Vitamin D<sub>3</sub></i>	<ul style="list-style-type: none"> <li>• Calcium and phosphorus absorption and homeostasis</li> </ul>	<ul style="list-style-type: none"> <li>• Decreased growth and appetite</li> <li>• High mortality</li> <li>• Curvature and increased brittleness of bones, enlargement of bone epiphyses</li> <li>• Lameness and muscle weakness, increased TD incidence</li> <li>• Soft and pliant beak of rachitic poults</li> <li>• Increased soft-shelled eggs</li> <li>• Decreased egg numbers, fertility and hatch</li> </ul>
<i>Vitamin E</i>	<ul style="list-style-type: none"> <li>• Fat-soluble biological antioxidant located in the phospholipid fraction of cellular and intracellular membranes</li> <li>• Sparing of selenium as a nutrient</li> <li>• Development of cellular and humoral immune systems and nervous system</li> </ul>	<ul style="list-style-type: none"> <li>• Decreased growth and appetite</li> <li>• Impaired muscle function and weakness involving striated, cardiac and smooth muscle</li> <li>• Gizzard myopathy</li> <li>• Eye irritation and infection</li> <li>• Degeneration of nervous system</li> <li>• Vascular system defects including increased capillary permeability</li> <li>• Testicular degeneration</li> <li>• Ovarian regression and decreased hatchability</li> </ul>
<i>K<sub>3</sub> (Menadione)</i>	<ul style="list-style-type: none"> <li>• Protein function, including plasma proteins that regulate blood coagulation</li> <li>• Post-translational <math>\gamma</math>-carboxylation of peptide-bound glutamate residues of specific plasma and non-plasma proteins</li> </ul>	<ul style="list-style-type: none"> <li>• Hemorrhage in most tissues and organs, bloody feces</li> <li>• General weakness</li> <li>• Rough feathering</li> </ul>
<i>Thiamine</i>	<ul style="list-style-type: none"> <li>• Essential cofactor in <math>\alpha</math>-ketoacid dehydrogenase enzyme complexes in metabolic pathways that include glycolysis (pyruvate dehydrogenase), TCA cycle (<math>\alpha</math>-ketoglutarate dehydrogenase), branched-chain amino acid metabolism (branched-chain <math>\alpha</math>-ketoacid dehydrogenase), and pentose phosphate pathway (transketolase)</li> </ul>	<ul style="list-style-type: none"> <li>• Severe loss of appetite, growth and feed efficiency</li> <li>• General weakness and death</li> <li>• Fatty degeneration and necrosis of cardiac tissue resulting in heart failure</li> <li>• Hepatic fat accumulation and hemorrhage</li> <li>• Neurological paralysis leading to neck retraction and convulsions</li> <li>• Inflammation of intestinal mucosa</li> <li>• Testicular degeneration</li> <li>• Atrophy of the ovary leading to decreased egg production and hatch</li> </ul>

**Table 3 (continued):** Metabolic function and deficiency symptoms of vitamins and trace minerals.

Nutrient	Metabolic Function	Deficiency Symptoms
<i>Riboflavin</i>	<ul style="list-style-type: none"> <li>• Component of coenzymes FAD and FMN which function as intermediaries in the transfer of electrons in oxidative-reduction metabolic pathways</li> <li>• Fatty acid synthesis and degradation</li> <li>• Amino acid catabolism as a cofactor of amino acid oxidase</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of appetite and growth</li> <li>• Severe dermatitis and beak encrustation</li> <li>• Myelin degeneration of nerves leading to curled toes, muscular spasms and paralysis</li> <li>• Inflammation of digestive system mucous membranes resulting in poor feed nutrient absorption and diarrhea</li> <li>• Poor feather-down development</li> <li>• Reduced egg production and hatch; edema in non-hatched embryos</li> </ul>
<i>Pantothenic Acid</i>	<ul style="list-style-type: none"> <li>• Component of Co-enzyme A which is involved in fatty acid synthesis and oxidation, acetylation reactions, and oxidative decarboxylation reactions</li> <li>• Component of acyl-carrier protein involved in fatty acid synthesis and elongation</li> </ul>	<ul style="list-style-type: none"> <li>• Reduced growth, appetite and feed efficiency</li> <li>• Feathers are rough and brittle</li> <li>• Feather depigmentation</li> <li>• Crusts at the corner of the beak</li> <li>• Exudates on eye lids and permanently closed eyes</li> <li>• Dermatitis particularly around the beak, eyes, and feet (more pronounced on the toes compared to biotin deficiency)</li> <li>• Fatty degeneration of liver</li> <li>• Myelin degeneration of neural tissue, impaired adrenal cortex function</li> <li>• Decreased lay performance and hatch</li> </ul>
<i>Niacin</i>	<ul style="list-style-type: none"> <li>• Component of NAD(H) and NADP(H) which are hydrogen carriers integral in carbohydrate, lipid and amino acid metabolism</li> <li>• DNA repair mechanism by ADP-ribosylation</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of appetite, growth, general weakness and diarrhea</li> <li>• Severe bowing of the legs and enlargement of the hock joint (perosis)</li> <li>• Mild to severe dermatitis, especially of the legs and feet</li> <li>• Reduced feather growth</li> <li>• Inflammation of the gastro-intestinal tract</li> <li>• Impaired neural function</li> </ul>
<i>Pyridoxine</i>	<ul style="list-style-type: none"> <li>• Involved widely in amino acid metabolism including transamination, decarboxylation, R-group interconversion and racemization</li> <li>• Neurotransmitter synthesis</li> <li>• Tryptophan-niacin conversion</li> <li>• Synthesis of porphyrin precursors to heme</li> <li>• Glycogen conversion to glucose</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of appetite, growth and feed efficiency</li> <li>• Dermatitis and inflamed edema of eye lids</li> <li>• Rough feathering and reduced feather growth</li> <li>• Anemia</li> <li>• Demyelination of peripheral nerves leading to poor coordination of movements</li> <li>• Muscular convulsions followed by paralysis</li> <li>• Reduced egg production and fertility</li> </ul>
<i>Biotin</i>	<ul style="list-style-type: none"> <li>• Carboxyl carrier for four carboxylase enzymes</li> <li>• Required for gluconeogenesis, fatty acid synthesis, and replenishment of mitochondrial oxaloacetate</li> <li>• Catabolism of branched-chain amino acids, methionine, threonine and odd-chain fatty acids</li> </ul>	<ul style="list-style-type: none"> <li>• Loss of appetite, growth and feed efficiency</li> <li>• Dermatitis, especially around the beak, feet and eyes; some similarities to pantothenic acid deficiency</li> <li>• More pronounced dermatitis of footpads than toes; compared to pantothenic acid deficiency</li> <li>• Rough and brittle feathers</li> <li>• Deformation of the beak</li> <li>• Fatty infiltration of liver and kidney</li> <li>• Shortened and thickened bones (perosis)</li> <li>• Poor hatchability and malformation of embryonic skeleton</li> </ul>

**Table 3 (continued):** Metabolic function and deficiency symptoms of vitamins and trace minerals.

Nutrient	Metabolic Function	Deficiency Symptoms
<i>Folic Acid</i>	<ul style="list-style-type: none"> <li>Metabolic transfer of single carbon units such as formyl, methenyl, methylene, methyl, and formimino</li> <li>Synthesis and catabolism of nucleic acids</li> <li>Synthesis and catabolism of amino acids</li> </ul>	<ul style="list-style-type: none"> <li>Loss of appetite, growth and feed efficiency</li> <li>Stiff and extended neck (cervical paralysis)</li> <li>Macrocytic anemia</li> <li>Rough feathering with weak shafts and depigmentation</li> <li>Nervous disorders, skin inflammation and feather loss</li> <li>Shortened and thickened bones (perosis)</li> <li>Poor hatchability, embryos mostly dying late in incubation</li> <li>Deformed beaks and crooked tibiotarsals in non-hatched embryos</li> <li>Increased susceptibility to infections as synthesis of immunoglobulins is impaired due to leucopenia</li> <li>White, watery diarrhea</li> </ul>
<i>Vitamin B<sub>12</sub></i>	<ul style="list-style-type: none"> <li>Carbon rearrangement reaction required for propionate catabolism</li> <li>Leucine catabolism</li> <li>Methionine synthesis from homocysteine which is required for regeneration of tetrahydro-folic acid</li> </ul>	<ul style="list-style-type: none"> <li>Loss of appetite, growth and feed efficiency</li> <li>Reduced egg size and hatchability</li> <li>Neurological disorders due to impaired myelin maturation</li> <li>Fatty infiltration of liver</li> <li>Many symptoms are similar to that of folic acid deficiency due to the trapping of tetrahydro-folic acid as methyl tetrahydro-folic acid</li> </ul>
<i>Choline</i>	<ul style="list-style-type: none"> <li>Structural component of cell membranes as phosphatidyl choline</li> <li>Transport of lipids</li> <li>Neurotransmitter</li> <li>Intracellular signal transduction</li> <li>Supplies labile methyl groups for transmethylation reactions</li> </ul>	<ul style="list-style-type: none"> <li>Reduced growth</li> <li>Fatty infiltration of liver and kidney</li> <li>Shortened and thickened bones (perosis)</li> </ul>
<i>Manganese</i>	<ul style="list-style-type: none"> <li>Constituent of certain metalloenzymes and activator of certain enzymes</li> <li>Bone development from the synthesis of mucopolysaccharide, an important component of bone organic-matrix</li> <li>Choline synthesis</li> <li>Carbohydrate metabolism</li> </ul>	<ul style="list-style-type: none"> <li>Shortened and thickened bones (perosis)</li> <li>Decreased egg numbers, hatch and poor shell quality</li> <li>Embryonic chondrodystrophy due to impaired cartilage development</li> </ul>
<i>Iron</i>	<ul style="list-style-type: none"> <li>Present in enzymes responsible for electron transport (cytochromes), and activation of oxygen by oxidases and oxygenases</li> <li>Component of hemoglobin and myoglobin which are involved in oxygen transport</li> <li>Detoxification of peroxides</li> </ul>	<ul style="list-style-type: none"> <li>Hypochromic macrocytic anemia</li> <li>Poor feathering and feather depigmentation</li> <li>Increased embryonic mortality and decreased hatching poult livability</li> </ul>
<i>Copper</i>	<ul style="list-style-type: none"> <li>Iron absorption and mobilization</li> <li>Reduction of oxygen to water in the final step of electron transport in the cytochromes</li> <li>Cross-linking of connective tissues such as collagen</li> </ul>	<ul style="list-style-type: none"> <li>Anemia</li> <li>Aortic ruptures from the failure of converting lysine to desmosine which is the cross-linking residue of elastin</li> <li>Bent and fragile long bones due to the failure of collagen cross-linking in the organic matrix</li> <li>Shortened and thickened bones (perosis)</li> <li>Decreased egg numbers and hatch</li> </ul>

**Table 3 (continued):** Metabolic function and deficiency symptoms of vitamins and trace minerals.

Nutrient	Metabolic Function	Deficiency Symptoms
<i>Iodine</i>	<ul style="list-style-type: none"> <li>● Synthesis of thyroid hormones which are involved in thermoregulation, intermediary metabolism, reproduction, tissue development &amp; differentiation, and muscle function</li> </ul>	<ul style="list-style-type: none"> <li>● Goiter</li> <li>● Decreased egg production and egg size</li> <li>● Abnormal feathering</li> </ul>
<i>Zinc</i>	<ul style="list-style-type: none"> <li>● Part of the enzyme systems involved in nucleic acid metabolism, protein synthesis and carbohydrate metabolism</li> <li>● Cellular division, growth and repair</li> <li>● Production, storage and secretion of hormones such as testosterone, insulin and adrenal corticosteroids</li> <li>● Spermatogenesis</li> <li>● Reproductive development</li> <li>● Skin and wound healing</li> <li>● Vitamin A mobilization from the liver</li> </ul>	<ul style="list-style-type: none"> <li>● Loss of appetite, growth and feed efficiency</li> <li>● Decreased egg numbers, failure of normal embryonic development</li> <li>● Shortened and thickened bones (perosis)</li> <li>● Rough and brittle feathering</li> <li>● Dermatitis; especially on the feet, legs and around the beak</li> </ul>
<i>Selenium</i>	<ul style="list-style-type: none"> <li>● Closely linked to vitamin E function in preventing cell membranes from oxidative damage</li> <li>● Sparing of vitamin E as a nutrient</li> <li>● Spermatozoa function</li> <li>● Immune function</li> </ul>	<ul style="list-style-type: none"> <li>● Exudative diathesis characterized by hemorrhaging on the thigh and breast muscle</li> <li>● Muscular dystrophy</li> <li>● Gizzard myopathy showing severe hyaline degeneration of the muscular tissue</li> <li>● Cardiac and skeletal myopathy</li> <li>● Pancreatic atrophy resulting in poor growth and feathering</li> <li>● Impaired immunocompetence</li> <li>● Reduced egg numbers and hatch; and increased embryonic mortality</li> <li>● Increased abnormal spermatozoa</li> </ul>