Keel Bone Integrity in Layers: Observations, Nutritional Remedies, and Myth-Busting

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Introduction

The issue of keel bone deformities and abnormalities is different depending on various housing systems for the birds. Keel fractures have received a lot of attention lately because of the increase in "cage-free" production in the egg industry. European researchers have led a lot of studies on the issue of keel fractures in aviary systems (Vits et al., 2005; Heerkens et al., 2016), but North American scientists have also been studying the issue as well (Blatchford et al., 2015). The research and observations that I will focus on will be related to the conventional cage system. The unique observations that I have to report is probably the most important information for this presentation. Of course, nutritional solutions are also very important since this is partially a nutrition symposium. The final portion of my title relates to providing data and commercial experience to address some common misperceptions in the industry.

The nutritional solutions are answers I have determined to benefit MFI in our feeding program. The information I will be presenting is more validations of previous studies and recommendations by consultants than novel ideas. My contribution is to provide more specifics with clear results under more commercial-type conditions. The bird observations and myth-busting claims will be discussed at the end relative to nutritional studies and programs at MFI.

The nutritional studies/experiences will be divided into three parts: 1) Vitamin D (levels and sources), 2) Calcium (level and sources), and 3) aluminosilicates. The research is focused primarily on Bovans White birds. Although soft bones and keel deformations can be observed with any strain of bird, at MFI we have historically had more soft bone issues with Bovans birds. Hence, I used this strain of bird as my model to minimize strain as a variable in the studies.

Vitamin D_3

The typical industry recommendation for vitamin D_3 for laying hens is 3,000,000 IU/ton. However, Matilla et al. (2004) reported that tibia breaking strength is increased at 68 weeks of age when about double this amount of vitamin D_3 was fed starting at 20 weeks of age. Preliminary studies at our Wakefield facility in commercial houses showed that keel tip softness in Bovans birds could be further decreased by adding additional vitamin D_3 premix to the diet during peak production. The decision was made at MFI to increase our vitamin D_3 content in our vitamin premix to provide 6,000,000 IU/ton of feed rather than 3,000,000 IU/ton.

A vitamin D source trial was conducted at our "project house" which houses approximately 500 birds in Farmer Automatic cages similar to the system used in some of our commercial houses. The basal diet was a typical corn-soybean meal-meat and bone meal-DDGS diet used in our formulation program. The control group was fed 6,000,000 IU/ton vitamin D_3 (all spray-dried). Treatment 2 consisted of adding 4,000,000 IU/ton more vitamin D_3 in the spray-dried form. Treatment 3 consisted of adding 25-OHD_3 (Hy-D from DSM) at the manufacturer's recommendation of 69 *u*g/kg. This level of Hy-D would be equivalent to adding 2,500,000 IU/ton if compared directly to vitamin D_3 activity. Edwards et al. (1994) reported that 800 ICU/kg (20 ug/kg) vitamin D_3 yielded similar protection against rickets incidence and improved tibia bone ash the same as exposing broiler chicks to ultraviolet light. Ledwaba and Roberson (2003) reported a similar result in these parameters when 10 *u*g/kg 25-OHD_3 was fed to broiler chicks in place of exposure to ultraviolet light. Thus, the relative biological activity of 25-OHD_3 to vitamin D_3 is about 2x. Hence, the Hy-D addition to the diet in this trial would be equivalent to adding 5,000,000 IU/ton.

The birds were fed the treatments from housing in the project house at 16 wk of age to 30 weeks of age. There were no differences in production parameters or keel bone softness between vitamin D sources, but keel tip softness was higher in the control group fed 6,000,000 IU/ton compared to birds fed additional vitamin D activity. Kim et al. (2011) reported that cortical bone mineral content of broiler femurs was increased when vitamin D_3 was fed at 9,000,000 IU/ton.

There was no mortality in the trial, demonstrating that keel bone softness is not related to mortality observed in early lay when soft bones are diagnosed in necropsy of mortality in commercial houses. This directly busts the myth spread by some members of the egg industry that birds with soft birds will suddenly die from cage-layer fatigue in peak production. The Hy-D treatment was about 7x the cost of adding spray-dried vitamin D_3 to the diet.

Calcium

Dietary calcium level in peak production has typically been targeted to reach 4.3 g/day in peak production which is slightly above breeder guidelines of 4.1-4.2 g/day. The proportion of large particle limestone contributing to the dietary calcium provided is usually 40-45 % in peak production. Dietary available phosphorus is typically fed at about 500 mg/d in peak production, but may be fed at a 5-10% higher level for Bovans birds.

An additional factor for MFI to consider is the recycling of dried eggshells from our breaker plants to use as a calcium source in our diets. The solubility of eggshells may be lower than the fine particle limestone we use which contributes another factor to consider when feeding to prevent soft keel tips and keel deformities. The use of a minimum of 20 lb/ton fine limestone instead of relying completely on eggshells for small particle calcium has been a vital part of my formulation program to reduce soft bone issues in peak production.

Eggshell solubility has been shown to improve if the shells are ground to a level of about 250 *u*m. Cheng and Coon (1990a) reported that solubility of limestone affects bone structure in layers. Pulverized limestone with 47% solubility fed to layers resulted in high tibia bone ash in peak production (Keshavarz et al., 1993). Solubility of eggshells may be about half this amount.

Research has shown that cortical bone ash weight will be increased at 4.5 g/day calcium consumption compared to 4.0 g/day or lower (Cheng and Coon, 1990b). Studies in the same laboratory showed that bone strength can be improved in laying hens if dietary calcium is fed at 5.0 g/day vs. 3.5 g/day (Zhang, 1994). Recent changes in the MFI feeding program has resulted in lower soft bones issues if dietary calcium is increased to 4.5-4.6 g/day by 25 weeks of age when the flock has reached peak production. This method allows for dietary calcium to be able to supply the amount of calcium needed to produce the shell on the egg. Large particle calcium is continually increased with age as the birds reach 50 weeks of age and older.

Aluminosilicates

I have run several commercial trials on the use of the anti-caking agent T-Bind and observed improved bone structure as well as a positive effect on egg production. The institution of a T-bind program minimized issues with mouth lesions that were prevalent when I started working for MFI in 2005. We have also seen a reduction in pasty vents. T-bind has been used to mitigate issues with mycotoxins in several areas of the country.

The original trial in our project house was conducted using Hy-Line W36 birds. The percent of birds with soft keel tips was decreased when 6 lb/ton T-bind was fed, but more importantly the amount of severely soft tips was cut in half when either 3 or 6 lb/ton T-bind was fed in peak production. Ballard and Edwards (1988) reported that sodium aluminosilicate increased calcium absorption and ^{47}Ca retention in broiler chicks. In a more recent trial with Bovans White birds, the addition of T-bind at two particles sizes (400 or 1100 *u*m) as well as a similar level of Azomite (900 *u*m) still reduced soft keel tip incidence even with changes regarding vitamin D_3 and calcium level increases implemented to the program.

Observations and Myth-Busting

A general observation in my bone health studies has been the sudden increase in keel tip softness as the flock reaches peak production (25 weeks of age) indicating a negative calcium balance (Morgan and Mitchell, 1938). Medullary bone is maintained at the expense of cortical bone during the period of calcium deficiency (Etches, 1987). The development of keel deformation would typically be seen as the birds reached about 35 weeks of age. Keel deformities can begin sooner if there is an environmental situation which results in reduced feed consumption as the birds are coming in peak production. The incidence of bone abnormalities can affect a large portion of the flock with no effect on egg production. This busts the myth that keel abnormalities will result in lower egg production. This belief refers to the claim by Nasr et al. (2013) that keel fractures will decrease egg production by 6%.

The incidence of keel abnormalities will also be reduced as the birds age even during peak production without affecting egg production. This busts another myth that birds must go out of production to heal soft bones. Mortality was almost non-existent during peak production in my project house studies and any dead bird found had very strong bones. There was absolutely no correlation between soft bones and mortality in peak production which busts another myth in the industry that birds found dead with some softness in the bones but no other issues diagnosed must have died from cage-layer fatigue. In a university research trial I conducted about 15 years ago, laying hens had to be fed a diet that was very deficient in phosphorus for several months before mortality was observed due to nutrient deficiency (Roberson et al., 2004).

An important observation in current studies was that soft keel tips that are not severe will completely heal. Severely soft tips will result in bent tips and curvature at the end of the keel tip and possibly a broken keel tip. Keel curvature starts with a deviation in the keel bone which can occur at different areas of the keel, but usually in the mid-section of the bone. Mild deviations will typically heal while severe deviations eventually begin to bend into the S-shaped curve identified with keel curvature. These changes can occur over a one-week period and is very sensitive to changes in feed intake. A change of 5-8% in feed intake has a large influence on changes in keel abnormalities. These changes may not even be noticed if the birds on fed on a phase-feeding program where changes are made monthly or longer. The observation that birds can heal and continue to grow bone in peak production busts the myth that once birds have keel deformations the birds can never fully recover and replace lost trabecular bone tissue (Rennie et al., 1997) and agrees with Zhang (1994) that bone growth can continue to occur in peak production. Osteoclastic activity predominates during active shell formation and osteoblastic activity is more dominant when the shell gland is inactive (Taylor and Belanger, 1969).

Keel abnormalities continue to be a concern for our company as we consider it to be a welfare issue for the bird (although they exhibit no pain during keel palpation). The long-term effects of soft and bent keels on bird health during post-peak are being evaluated more directly in current studies. The primary take-home message is that high levels of keel abnormalities can be observed during peak production with no effect on egg production or mortality and non-severe keel abnormalities can be quickly healed in laying hens with no pause in egg production.

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