

# info

## An Inside and Outside Look at Rickets

Rickets is a disease of young growing poult characterized by soft poorly mineralized bones and irregular, thickened growth plates. In 1982, Riddell reported losses in turkey flocks, ranging from 1 to 14 percent due to rickets. Improved quality control within feedmills, faster recognition of the problem and more effective treatment has dramatically reduced the mortality associated with rickets. However, in spite of preventive measures, the incidence of rickets within commercial flocks remains too high.

---

**With the improvements in genetic potential of turkeys, especially growth rates and daily gains, the impact of rickets on leg quality and final product performance is economically significant and often not recognized.**

---

A retrospective survey of skeletal lesions in turkey flocks reported that flocks which had rickets during brooding had a much higher incidence of leg problems during grow out. The poult leg weakness observed was most commonly subclinical with relatively few lame birds directly due to rickets but significant losses occurred later due to other skeletal disorders.

This fact sheet will discuss the appearance of rickets, causes, treatment and prevention.

### Appearance of Rickets

Rickets typically occurs between 10 and 25

days of age. Affected poult are reluctant to move, remain sitting on their hocks and will use their wings to help support their bodies if required to walk, thus the term “wing walkers” is frequently used. These birds may have difficulty breathing due to painful ribs. In chickens, right-sided heart failure has been reported as a sequel to rickets.

On gross post mortem the following lesions are typical of rickets :

- ↪ Metatarsal (shank) bones do not “snap” crisply when broken but are rubbery.
- ↪ Beaks are soft and flex easily.
- ↪ Ribs are thickened and tend to bend so that the chest is flattened.
- ↪ Beading of the ribs is noticeable at their junction with the spine.
- ↪ Ends of long bones are enlarged.
- ↪ Growth plates are thickened. (Visible when slicing through the top of the tibia.)
- ↪ Enlarged parathyroid glands

Microscopically, the appearance of the various zones within the bone varies depending upon the underlying causes of the rickets. These are often very difficult to distinguish and will not be described here. What is important to note is that microfractures are present within the bones. Reluctance of birds to walk is likely due to the pain associated with these microfractures. Affected birds have a decreased bone ash (calcium phosphate).

**Hybrid Turkeys**  
Head Office  
650 Riverbend Drive  
Suite C  
Kitchener, ON N2K 3S2  
Canada  
Tel. 519-578-2740  
Fax 519-578-1870  
E-Mail:  
info@hybridturkeys.com

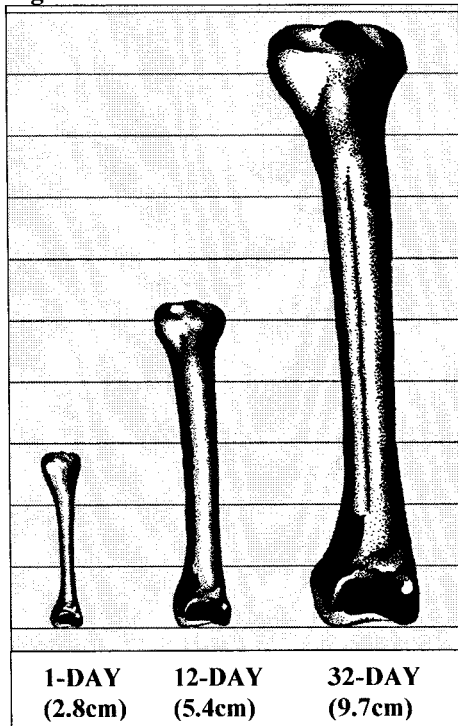
**Hybrid Turkeys**  
European Office  
P.O. Box 30  
5830 AA Boxmeer  
The Netherlands  
Telephone +31 485 589922  
Fax +31 485 575205  
E-Mail: euribrid@e-mail.com



Treatment causes these fractures to heal very quickly. However the forces put upon the bone during this time of repair and remodeling will often determine whether bone growth is normal. It must be remembered that rickets occurs while the poult is experiencing maximal skeletal growth. Figure 1 illustrates the 2mm daily growth during this time. This represents a 25% increase in tibial length between 2 and 3 weeks of age.

It has been reported that poults that have unilateral footpad infections often develop leg deformities in the unaffected leg. This may be the result of the bird shifting its weight onto its good leg which markedly increases the force put on that leg. When force is not applied uniformly to the growth plate, one side grows faster than the other resulting in an angular leg deformity.

**Figure 1: Tibial Growth**



**What Causes Rickets ?**

At 1 to 4 weeks of age, poults have their greatest demand for Vitamin D, calcium and phosphorus for incorporation of mineral into the skeleton. Table 1 summarizes the essential nutrients necessary and their role in normal bone formation.

**Table 1**

Nutrient	Role in Bone Formation
Calcium and Phosphorus	Must be present in the correct level and ratio to form hydroxy apatite crystals required for calcification of cartilage.
Vitamin D <sub>3</sub> (a fat soluble vitamin)	Vital role in the absorption and retention of calcium. Vit D present in the feed is absorbed through the intestine into the bloodstream and transported to the liver where it is converted into 25 (OH) D <sub>3</sub> . This is sent to the kidneys which converts it into the active forms of D <sub>3</sub> : 1, 25 (OH) <sub>2</sub> D <sub>3</sub> and 24, 25 (OH) <sub>2</sub> D <sub>3</sub> .
Gastro intestinal tract (GIT)	Fully functional GIT required to absorb nutrients and vitamins in a timely manner to meet the demands of rapid growth.

The following have been shown to be causes of rickets.

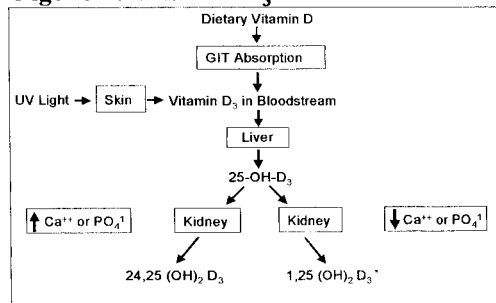
**Lack of or imbalance in the ratio of calcium to phosphorus**

Newly hatched poults have minimal stores of calcium. Consequently deficiency develops quickly if they do not receive or consume an adequate supply.

**Vitamin D deficiency**

Vitamin D is essential for skeletal formation and maintenance of calcium and phosphorus homeostasis. This is achieved by controlling the intestinal absorption of calcium and mobilization of bone stores through the hormonal action of 1, 25 – (OH)<sub>2</sub> D<sub>3</sub> (Figure 2). Thus factors that interfere with Vitamin D availability and absorption will have a negative impact on bone strength and performance of turkeys.

**Figure 2: Vitamin D<sub>3</sub> Metabolism**



\* Cholecalciferol (Vit-D<sub>3</sub>) metabolism in fowl

In many cases of rickets, the diets have been found to be adequate in vitamin D<sub>3</sub>, calcium and phosphorus. However further investigation often reveals other factors present which render the vitamin D unavailable or which compromise absorption. These include :

- ↪ Diets high in saturated fats such as tallow which bind with calcium in the gastrointestinal tract (GIT) to form soaps. Such undigested fat reduces the availability of vitamin D.
- ↪ Rancid fat used in feed may cause enteritis and may also destroy available vitamin D<sub>3</sub>.
- ↪ Turkey poults have low digestibility of fat due to low lipase activity during the first 2 weeks. Poor utilization of dietary fats contributes to poor absorption of fat soluble vitamins such as vitamin D<sub>3</sub>.
- ↪ Excess vitamin A. Vitamin A and D<sub>3</sub> compete for the same site of absorption in the GIT. This can occur when poults are being supplemented with a product which contains a high level of vitamin A or an inappropriate ratio of vitamin A to D.
- ↪ Freshness of the prestarter ration. The stability of nutrients and vitamin D<sub>3</sub> may be compromised by longer storage times (>10 days ) especially during summer heat.
- ↪ In light restricted confinement housing, poults receive insufficient quantities of vitamin D<sub>3</sub> via photolysis and must rely upon dietary supplement. Under these conditions, turkeys appear to be more vulnerable to low biopotent vitamin D in their ration or interference with absorption of vitamin D from their diet than chickens or other food-producing animals.

### ***Enteric Disease***

Enteritis, in young poults, is becoming increasingly more common and unfortunately accepted as being normal. The effects on the skeletal system and association with rickets are due to several factors.

Firstly, poults with enteritis back off feed. Their gall bladders become distended with bile due to the anorexia. Bile traps vitamin D

metabolites secreted by the liver, which normally would be reabsorbed by the GIT. Lack of bile in the GIT prevents Vitamin D from being absorbed. This is happening at the same time that maternal reserves of Vitamin D are just about gone. Because the GIT is not absorbing adequately, fat in the gut lumen binds with calcium to form calcium soap. This makes the calcium unavailable for absorption. Finally with decreased intestinal absorption, the amount of nutrients being absorbed and the effectiveness of absorption are both reduced.

### ***Mycotoxins***

Mycotoxins in feed, especially those produced by Fusarium molds, are associated with rickets due to the toxic effects on the liver and kidneys. Both of these organs are required to convert Vitamin D<sub>3</sub> into the active forms required for calcium absorption.

In addition, poults show decreased feed intake which results in an inadequate intake of vitamin D and calcium.

### **Treatment of Rickets**

Most cases of rickets will respond to water soluble vitamin D<sub>3</sub> if given early in addition to top dressing the feed daily with dicalcium phosphate until the problem is resolved.

Some cases which do not respond are likely due to GIT, liver or kidney damage. These birds would benefit more from the more active form of vitamin D<sub>3</sub>: 1, 25 (OH)<sub>2</sub> D<sub>3</sub>.

### **Prevention of Rickets**

Under typical commercial conditions, the following are recommended to prevent the occurrence of rickets.

- ↪ Feed a balanced ration with adequate calcium, phosphorus (1.5%:0.75%) and vitamin D<sub>3</sub> (4,500 IU/kg feed). Chemical antioxidants can be added to help preserve vitamin D<sub>3</sub>. Note that poultry require vitamin D<sub>3</sub>, a form differing from vitamin D<sub>2</sub>, which is often fed to other types of livestock.



- Use mold inhibitors to prevent mycotoxins in the feed and /or carefully screen incoming raw ingredients. Feed bins should be checked regularly for the presence of molds. Bin maintenance should include regularly checking for pin point holes and leaks in the tanks in addition to cleaning and spraying with a mold inhibitor when possible between flocks. Feedlines can be cleaned by running corn soaked with propionic acid through the lines.
- Provide feed as fresh as possible to poults. This is to prevent vitamin degradation and to stimulate appetite. Poults should have good quality fresh crumbs available at all times. Remember that inadequate intake of nutrients in a rapidly growing bird can contribute to both enteritis and rickets.
- Direct supplementation of diets with 1, 25 (OH)<sub>2</sub> D<sub>3</sub> has shown to have a positive effect on bone ash and decreased the incidence of rickets at 3 and 20 weeks of age.
- Recent research has shown that the addition of 25-hydroxycholecalciferol (25-OH-D<sub>3</sub>) reduced the incidence and severity of tibial dischondroplasia in broilers. Research on the effect of 25-OH-D<sub>3</sub> for turkeys is limited. However, it is possible that 25-OH-D<sub>3</sub> addition to turkey diet could improve leg strength particularly on farms with a history of leg problems.
- Avoid high levels of vitamin A in the feed (> 12,000 IU/kg) and in multi-vitamin or poult stress packs.
- On farms with a history of rickets (often due to enteritis challenge), give water soluble vitamin D<sub>3</sub> on days 8, 10 and 12 when lipase activity and fat digestibility are at low levels. Do not give excess vitamin D<sub>3</sub> otherwise rickets may occur due to toxicity.

- Prestarter and starter diets should contain ingredients that are easily digested. Avoid rye, barley and high levels of soyabean meal.

## Conclusion

The impact of rickets on subsequent leg strength and quality cannot be overemphasized. Prevention, early recognition and prompt treatment are critical to optimizing flock performance.

### *Rickets References*

- Edwards H.M. (1992). Nutritional factors and leg disorders. Chp. 10 Bone Biology and Skeletal Disorders in Poultry. Poultry Science Symposium No. 23.
- Hedstrom O.R., Cheville N.F. and R.L. Horst. (1986) Pathology of Vitamin D deficiency in growing turkeys. Vet. Path. 23 : 485-498.
- Perry R.W., Rowland G.N., Glisson J.R., Steffens W.L. and Quinn, J.A. (1991) Skeletal lesions associated with a naturally occurring poult enteritis. Avian Diseases, 35 : 58-164.
- Perry R.W., Rowland G.N. and Glisson J.R.. ( 1991) Poult malabsorption syndrome. II. Pathogenesis of skeletal lesions. Avian Diseases, 35 : 694-706.
- Riddell C. (1980) A survey of skeletal disorders in five turkey flocks in Saskatchewan. Canadian Journal Comparative Medicine, 44 : 275-279.
- Walser M.M., Hanlon G.F., Newman J.A., Dziuk H.E., Olsen W.G. and Waibel P.E. (1980) "Field rickets" in turkey poults: field observations and pathological, radiological and serological findings. Avian Diseases 24 : 309-316

### © Hybrid Turkeys

Unless otherwise specified, the information provided here is the property of Hybrid Turkeys. Before reproducing or publishing this material in any manner, please obtain approval by calling Hybrid Turkeys' head office in Kitchener, Ontario, Canada at +1-519-578-2740.