Natural vitamin D_3 metabolite supports bone health of laying hens

Proper skeleton calcium (Ca) stores is key to reduce bone disorders. Early sexual maturation, alongside production of the first egg at a younger age, fast increase to peak production and improved laying persistency, has increased the demand for proper skeletal Ca stores at the start of egg production. Only then can bone and shell quality be maintained throughout life.

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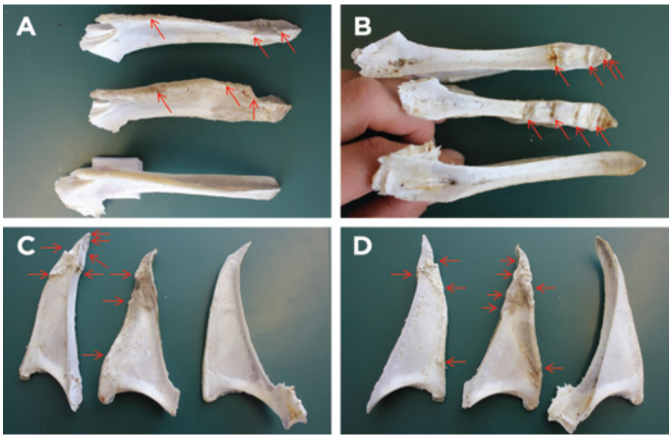


Fig. 1. Keel bones from Danish layers aged 78 weeks with and without fractures and deviations. Three keel bones shown from different angles. The bone at the bottom in **A,B** and to the right in **C,D** is without fractures, but it has a deviation. Fractures visible on the photos are marked with red arrows on the two damaged keel bones. The different angles: **(A)** the ventral side – the tip is to the right, **(B)** the dorsal side – the tip is to the right, **(C)** the right side of the keel bones – the tip is at the top, and **(D)** the left side of the keel bones – the tip is at the top (Anja B. Riber, 2018).

Laying hens have two types of bone: cortical/trabecular bone and medullary bone. The former maintains the physical integrity of the skeleton and acts as structural bone, whereas the latter serves as a mobile Ca pool for eggshell formation. Medullary bone is mobilised on a daily basis to supply Ca during eggshell formation, when Ca absorption from the intestine is inadequate. When eggshell formation ends, Ca absorption enables repletion of these Ca stores. If the quality of structural bone decreases, the skeleton becomes weaker leading to osteoporosis and fractures. Osteoporosis is characterised by a progressive loss of structural bone throughout lay, making bones fragile and susceptible to fracture. When hens start egg production too young, the Ca pool might still be too small, resulting in increased mobilisation of structural bone during egg production and thus increased osteoporosis.

Keel bone damage (KBD) is another bone disorder that gains importance in modern laying hens, as it compromises bird welfare and has a negative impact on production.

The keel bone is the pronounced bone that extends from the sternum and runs axially over the midline of the carcase. It anchors the breast muscles pectoralis major and pectoralis minor, which are used for wing motion. Keel bone fractures are characterised by sharp bends, shearing, and/or fragmented sections of the keel bone, whereas keel bone deviations refer to bent, S-shaped, twisted, or curved keels.

It is a bone with an abnormally shaped structure that has not resulted from a fracture but contains vertical or horizontal sections that deviate from the straight line (Fig. 1). Several studies indicate that the prevalence of keel bone fractures increases during the laying period.

The development of deviations likely takes place over a period of time as a result of bone pressure related to vigorous wing-flapping during roosting and/or perching. One possible cause of KBD is the stop of skeletal growth at sexual maturity at about 16-18 weeks of age, while ossification of the keel bone normally continues until approximately 40 weeks of age.

However, due to the increased Ca demand when egg production starts, parts of the keel bone remains cartilaginous. In modern laying hens, egg deposition has a higher priority over bone calcification.

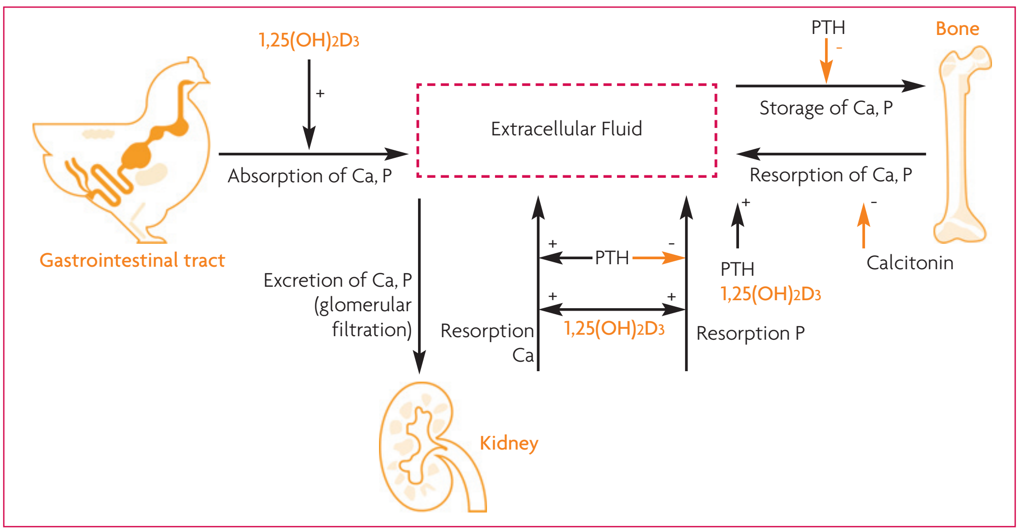
The development of KBD is affected by a multitude of factors like hen age, strain, housing system and other management factors, making it difficult to estimate the average KBD prevalence.

Nutritional approach

To minimise the incidence of osteoporosis, keel bone damage and related disorders, the supply of Ca, P and vitamin D_3 should be in concert with the bird's demand at maturation. The daily Ca supply for egg shell formation is coming from both feed (60-75%) and bone (40-25%). Calcium from bone is mobilised preferentially from medullary bone but might also come from trabecular bone. The latter

Continued on page 30

Fig. 2. The role of $1,25(OH)_2D_3$ in Ca and P metabolism (Herbonis Animal Health).



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reduces bone strength and cannot be restored after Ca has been mobilised. $1,25(OH)_2D_3$ (the bioactive vitamin D_3 metabolite) is a key player in Ca-homeostasis together with PTH (Fig. 2). $1,25(OH)_2D_3$ is needed to increase Ca absorption from the intestinal tract and Ca mobilisation from bone during eggshell formation and to restore it after eggshell formation is finished.

$1,25(OH)_2D_3$ has a direct effect on skeletal formation

$1,25(OH)_2D_3$ is crucial for calcium homeostasis and bone metabolism. It also has direct effects on skeletal formation, as all cells related to skeleton formation – chondrocytes, osteoblasts, and osteoclasts – contain vitamin D_3 receptors (VDR). $1,25(OH)_2D_3$ stimulates bone matrix formation and bone maturation and enhances osteoclast activity.

Osteoclasts, which originate from bone marrow stem cells, mobilise bone to initiate normal bone remodelling and mediate bone loss in pathologic conditions by increasing their resorptive activity.

In addition, $1,25(OH)_2D_3$ increases the activity of alkaline phosphatase and the expression of bone calcification genes in osteoblasts (cells required for bone synthesis and mineralisation, both during the initial formation of bone and during bone remodelling), to maintain the concentration of Ca and P and promote ossification. Decreased levels of $1,25(OH)_2D_3$ may cause decreased vascular invasion associated with reduced activity and thereby contribute to growth plate abnormalities such as tibial dyschondroplasia and rickets.

1α-hydroxylase activity is reduced in aged laying hens

The balance between bone formation and bone resorption tends to become disturbed with age.

In aged laying hens the circulating $25(OH)D_3$ and $1,25(OH)_2D_3$ are reduced by more than 15% and 50%, respectively in aged (93 week-old) versus young (34 week-old) laying hens. The reason for such low conversion of $25(OH)D_3$ into $1,25(OH)_2D_3$ in aged hens is related to the reduced activity in the kidney of the enzyme 1α-hydroxylase.

Increasing Ca levels is not the best option

In addition to the abovementioned points on Ca homeostasis, increased dietary limestone particle size is a known possibility to extend the duration of Ca supply from the intestines during the dark period, when egg formation continues and feed intake stops.

Increased availability of Ca from the intestines reduces the need for Ca mobilisation from the bones.

Feeding an optimum blend between fine and coarse limestone is a way to ensure that sufficient Ca remains available during peak eggshell formation (evening hours).

Layer management guides recommend 50:50 fine versus coarse particle size in the pre-laying phase and a gradual decrease of fine particles throughout life to a ratio of 25:75 at about 75 weeks of age.

Efficiency of Ca absorption in laying hens reduces with age. Its consequence cannot really be solved by increasing dietary Ca contents, as this further reduces the efficiency of Ca absorption.

Moreover, higher levels of unabsorbed Ca remaining in the intestinal lumen will adversely impact the digestibility of other nutrients.

As the basis is in the vitamin D_3 metabolism, it is a better option to increase $1,25(OH)_2D_3$ levels rather than dietary Ca to improve Ca homeostasis in ageing laying hens.

$1,25(OH)_2D_3$-glycosides, the unique source of the bioactive form of vitamin D_3

$1,25(OH)_2D_3$-glycosides (Panbonis) is derived from dried and ground Solanum glaucophyllum leaves.

Being a source of the active form of vitamin D_3, $1,25(OH)_2D_3$-gly needs neither activation in the liver nor in the kidney. Instead, only the glycosidic bonds of $1,25(OH)_2D_3$-gly need to be cleaved in the intestine.

Thereby, the $1,25(OH)_2D_3$ is gradually absorbed and immediately functional. Afterwards, $1,25(OH)_2D_3$ is quickly inactivated and excreted. ■

References are available from the authors on request