LAYING CYCLE

WHAT DO BONES HAVE TO DO WITH IT?

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Fig.: research papers see links (doi)

The (egg) laying cycle ends with the last egg laid, then the hen goes broody or starts moult. Broody: she stays put on the egg and goes into The Zone. When she isn't a 'sitter', the cycle ends when it's time to moult. The laying cycle of the first hen is shorter than that of the hen that continues to lay until moult sets in.

Calcium carbonate (CaCo3) for egg shells comes from the hen's bones (medula) and she absorbs it through her gut

She cannot continue laying eggs forever, she has to replenish the calcium. Replenishing the calcium in her bones goes on forever. How efficient this 'calcium metabolism' is determines how many eggs she can lay with a good shell. As does the ability of her gut to extract the calcium from the feed. You under-stand that intestinal disease therefore has an effect on egg shell quality over time, that is, if it cannot be replenished.

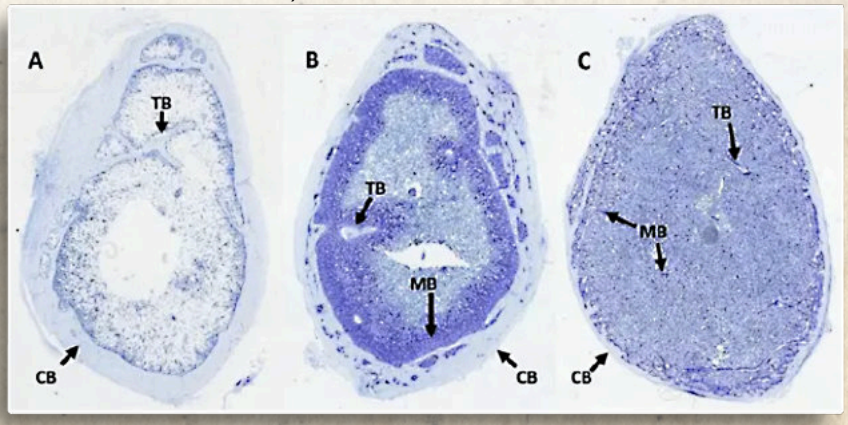
In commercial layers, there is careful management underlying keeping the hen's condition (bones, eggshell) optimal. In hobby hens, we leave this calcium management to the hens themselves. Of course, we give them calcium separately from the feed, so they can decide for themselves if they need it. We don't practice sophisti-cated management, because the hens don't have to be top performance hens like those in the industry.
The hobby chicken decides how many eggs it can lay because its genetics are not tuned to maximum egg production. Hobby chickens are often historical breeds (breed = once local chicken with certain distinctive characteristics).
The hobby hen hen takes care herself of the health of her sekeleton and the quality of the hatching egg and thus the length of the laying cycle.

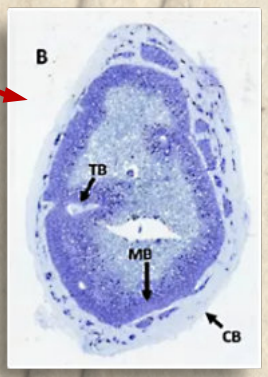
Bones and calcium
A young hen has two distinct bone structures: cortical and trabecular See figure **1A**. The firm cortical bone is the outer hard structure of the bone, the trabecular are the struts for further support. This construction allows the (hollow) bird bones to be light and strong. When the young hen matures, a surge of estrogen is released, causing a third bone type to form, the medullary bone, in response. Medullary bone provides an additional, variable source of calcium to supply the eggshell formation. This source of calcium is replenished on a daily basis depending on egg shell formation.

When egg production starts, the formation of structural bone stops and only medullary bone is formed. In the weeks preceding the laying of the first egg, the diameter of the bone increases by about 20%, increasing the volume in which medullary bone can grow on the inside of the bone. This increase in bone diameter means that more calcium (Ca) and phosphorus (P) are absorbed from the feed.

This changed requirement is therefore why there are different types of chicken feed for chick, growing hens and laying hens.
Besides the absorption of Ca and P from the feed, these are also extracted from the endosteal surfase creating

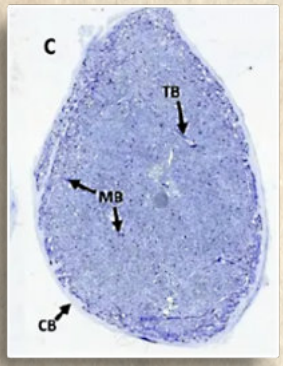
Figure 1 - Cross- sections of tibiae from laying hens of various ages. CB = cortical bone; TB = trabecular bone; MB = medullary bone. **A)** *Layer pullet (16 weeks of age) containing only the cortical shell and trabecular struts. Diffuse staining within the cortical shell is an artifact. Structural bone (CB + TB) tissue shows very little in the way of pore formation at this level of magnification. Sexual immaturity was confirmed at time of sampling by the absence of ovary development.* **B)** *Laying hen after the first egg was laid, showing the cortical shell, trabecular struts, and medullary bone. The medullary bone is present as small spicules of bone tissue, initially deposited on the surfaces of the structural bone tissues. Pores containing medullary bone within the cortical shell are clearly visible.* **C)** *End of lay (67-week old) hen showing depletion of cortical and trabecular bone tissues, and diffuse nature of medullary bone throughout the medullary cavity. MB arrows point to some larger spicules of medullary bone. (Presentation 2021, Korver, Dep. Agriculture Alberta, Canada).*





pores in the cortical layer (which is solid in immature hens) (**1B**). As estrogen levels increase as the hen approaches sexual maturity, two weeks before the first egg is laid, medullary bone in the form of small spicules (like structures) is laid over the surface of the structural bone and also in the pores of the cortical layer (1B).
The amount of estrogen increases as the hen approaches sexual maturity, medullary bone in the form of small spicules (needle-like structures) is laid over the surface of the structural bone and also in the pores of the cortical layer about two weeks before the first egg is laid. Check figure 1B to see this better.

During the night, when the amount of calcium through digestion is limited, medullary bone, as well as structural bone, is put to work to increase calcium levels in the bones.
This calcium is absorbed directly from the feed by the gut.
When the hen is not forming an egg shell and so there is excess of calcium and phosphorus that wouldn't be needed immediately, medullary and not structural bone is replaced. As time passes, the continuous mobilisation and deposition of medullary bone leads to a diffuse structure with spicules everywhere through the medullary cavity (**1C**).

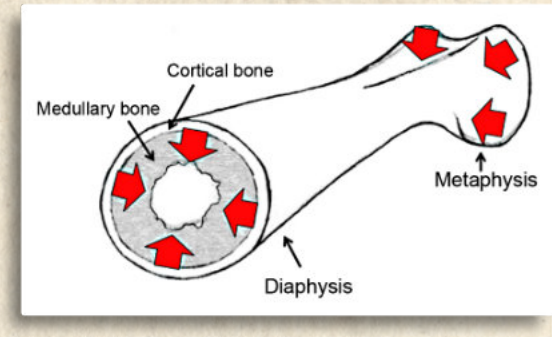


It is even possible that medullary bone can fill the entire cavity of the, originally with hollow bone.
As you can see, in figure 1C, the stuctural bone, which gives strength, has become wafer thin. Structural bone can fall prey to osteoporosis when the ratio of medullary bone to structural bone becomes unbalanced. Because structural bone is not replaced as long as the hen lays eggs. The cumulative effect of supplementing/using medullary bone will cause erosion to the structural bone making it so thin. Osteoporosis is not caused by a lack of calcium but by erosion (the formation of osteoclasts) in the increasingly thin structural bone.

Important to bone: going broody or into moult
When hens go broody or into moulting, the amount of estrogen decreases and medullary bone is taken up, structural bone is replenished.
The gradual loss of structural bone increases the risk of osteoporosis. The gradual increase in egg size as the hen ages, and the ability to deposit only a constant amount of eggshell material after the peak in egg production is reached, can lead to the decrease in eggshell thickness.
It is exactly this mechanism that places restrictions on the length of the laying cycle in industrial layers, among others. In our chickens, their skeletal health is prevented from deteriorating because they have their natural rhythm. Either by going broody which prevents eggs from being formed, or by moulting.

Artificial daylight lengthening and the young hen's development
Forcing young hens into egg laying, in particular by artificially lengthening the day by light, for example, results in the hen being unable to form strong structural bones.
This obviously varies from breed to breed, the time it takes to reach sexual maturity and also the desired body conformation and size.

When a young hen is artificially induced to start laying eggs by extending the day length, she will stop growing herself. Her bones will not get bigger and stronger, only thicker.
This explains why some hens of a certain breed turn out smaller than they should, they started laying eggs too early. Either by artificially extending the daylight or because of the time in the year they were born and thus exposed to longer days too early in their development.
This is sometimes used for bantams that have to 'stay small'. When such

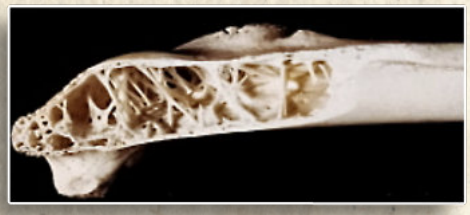


Drawing of a female femoral bone. The location of medullary and cortical bone is highlighted, as well as the metaphysis and diaphysis. The flow of calcium in egg-laying females is indicated with red arrows, illustrating the transfer of calcium from the metaphyses to the diaphysis, and from the hard cortical bone to the soft medullary bone. I skipped this part because it is beyond the scope of the laying cycle. If you want to go into this, here is the research paper: 10.1371/journal.pgen.1002914

hens have chicks at the optimal time of year for this breed, they will grow into larger chickens than their mothers.

Caring for the young hen
Because structural bone stops growing when the hen becomes sexually mature, her skeletal health is important. Medullary bone is not so important for the strength of her skeleton, structural bone is.

Therefore, the size of the young hen should be looked at, not age, whether she is large enough before considering artificial daylight lengthening. This is especially important in hobby chicken breeds or mixes of them that do not go broody. These breeds can continue to lay from moult to moult. This puts a strain on the hens' skeleton. Their rearing is therefore very important to ensure this is done properly because you want to enjoy your hen for as long



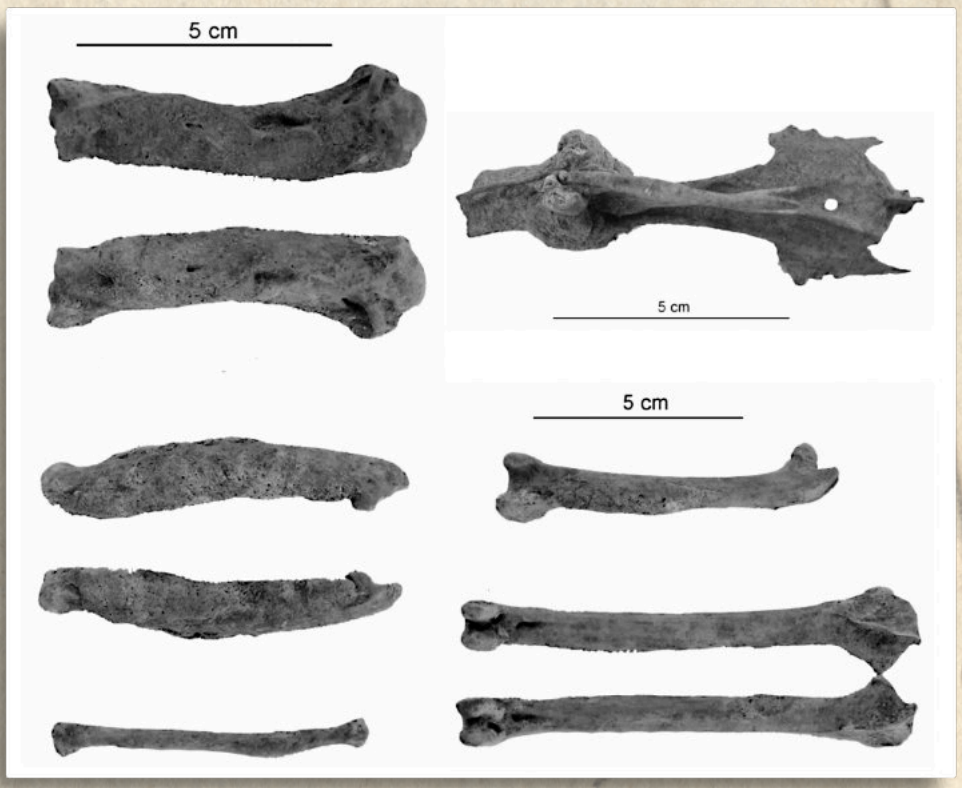
Bird bone. Image from Ornithology course webpage by Gary Ritchison (very interesting: http://people.eku.edu/ritchisong/554notes1.html)

as possible, preferably for years. The more optimal this whole event (everything written above) the better the eggshell will be and also the hen's ability to lay eggs for years to come, albeit in lesser quantity over time.

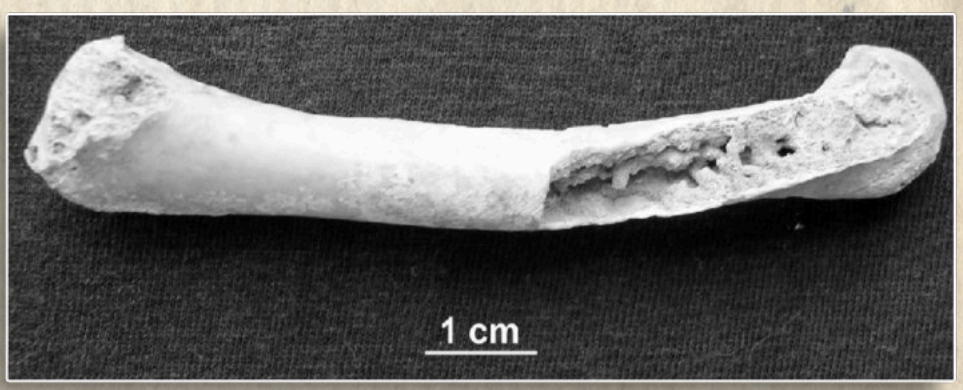
When a hen starts laying eggs too young, i.e. when her bones are not ready for it, she will have less reserves because the bones have less room to store medullar bone. Such hens will regress very quickly in laying eggs after their peak in the first year.
Young hens that also eat at night, for instance (do not stay asleep due to incoming light) will reach sexual maturity earlier than those that do not. Daylight length matters as stated earlier. It is therefore better not to continue breeding when the optimal time window for the breed in question has passed. Especially with Asian breeds, which do not adhere to a winter break like the original land breeds, this is important.

Calcium in feed for young hens
Providing calcium rich layer-feed to young hens makes no sense until they are in the approximately 2 weeks timeframe before their first egg, i.e. earlier from when medullar bone is actually produced. Grower feed contains sufficient calcium.
Too much calcium before they are sexually mature can interfere with calcium metabolism to maintain a uniform egg shell and a healthy skeleton.

If you do want to supplement extra calcium, you can do so based on the experience you have in rearing pullets of your particular breed. There is no general rule for this, it is individual or breed specific. This knowledge is part of 'breeder's art'.
With balanced feed, sufficient calcium is present for the first eggs. If this is not the case, it can be given immediately when you see that the first egg has been laid (and medullar bone formed). Or, the first eggs when it is a group of uniform young hens of the same age. This with observance that they are all the same size and it is a stable breed, so not a project hen, as they vary in size and build and growth rate.



Osteoporosis in a hen skeleton, a sternum, wing bones, leg bones. From the Middle Ages! (Budapest, Teleki Palace). As you see osteoporosis isn't something new, hitting industrial layers, it is from all times. From https://www.researchgate.net/publication/237212653_The_Role_of_Archaeo-Ornithology_in_Environmental_and_Animal_History_Studies



a. Medullary bone in a hen (Gallus domesticus) femur from the period of the Roman Empire. Could be your hen too, the system is the same.

Faulty egg shells
Now, of course, you are wondering where eggs with poor or no shell come from. A weak egg shell may be due to the inability to extract calcium from feed. This happens in the gut. Intestinal disease could be at the root of temporarily weak eggshells. However, there are many other reasons, which I don't know because this is complicated stuff and there is an awful lot of research into it, use google.

Furthermore, an interesting article on keeping laying hens optimal for as long as possible (commercial) and not having to dispose of them after 50-60 weeks. Currently, 100 weeks is possible. Mind you, these are hens bred for laying and are nothing like our hobby hens, maybe you can get something useful out of it. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9942826/ image on the next page is from this research.

Old research and cocks going broody
Old research is interesting to read, they did weird things 70-80 years ago. Here's one on how to break a broody

hen the chemical way. And also getting roosters to go broody... Not that it is of any use to you, it is just fun to read how research on our beloved chickens took shape. This research is from the days when there were no industrial laying breeds. https://www.sciencedirect.com/science/article/pii/S0032579119509932

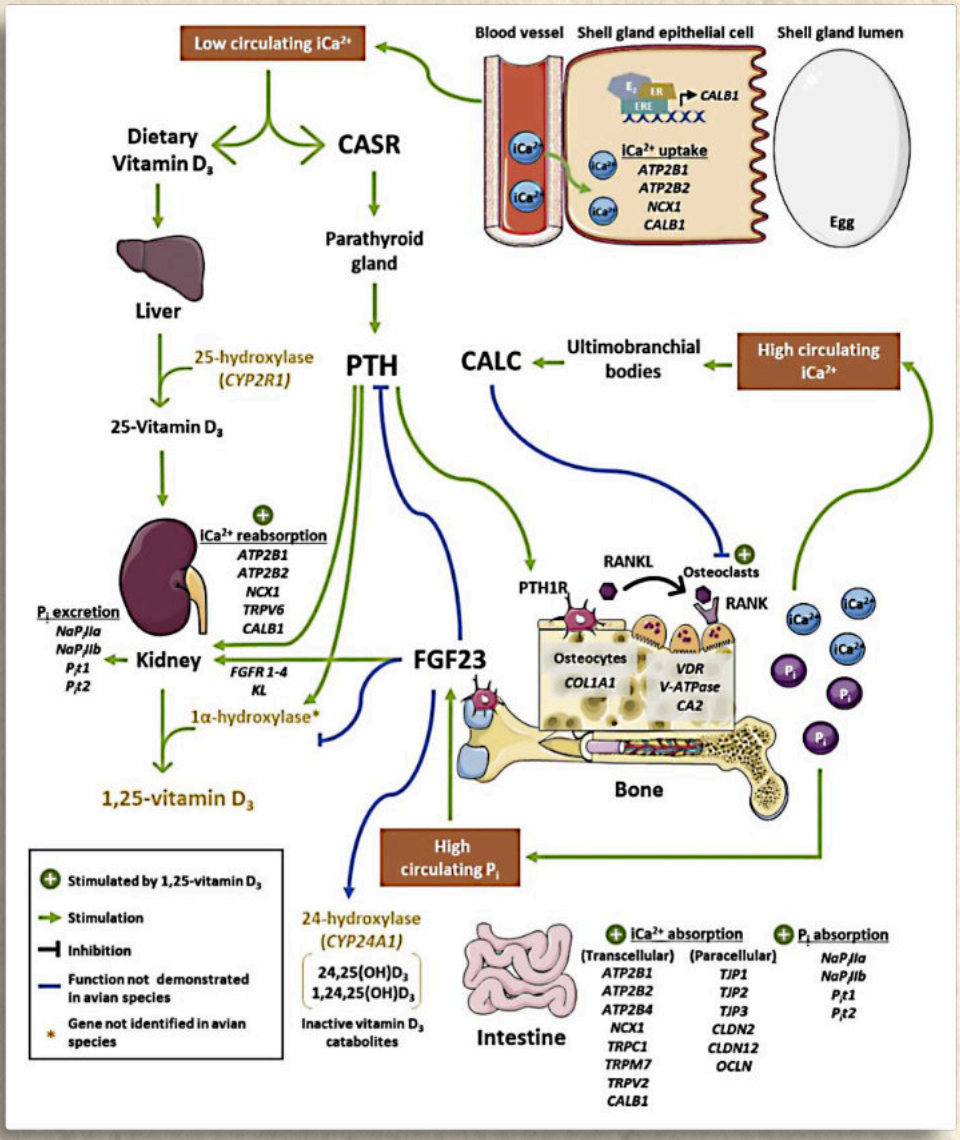
Here more on broodiness: https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/broodiness `

Here more on calcium metabolism: https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/calcium-metabolism

Egg shell quality and age: https://www.sciencedirect.com/science/article/pii/S0032579121003217

Laying hens at smallholders: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9178482/

There is a mega amount of research papers on these topics. In the story above, I told in the most donaldducki'sh way about the laying cycle and why. I hope you will find it useful in your next round of breeding and rearing your young hens.



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NERDIE STUFF

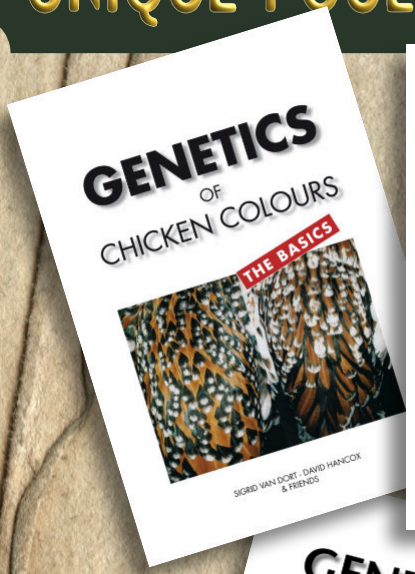
Regulation of calcium and phosphorus homeostasis during eggshell mineralization in laying hens. *During eggshell calcification, high demand for calcium decreases circulating ionized calcium (iCa2+). Low iCa2+ is detected by calcium-sensing receptor (CASR), which stimulates parathyroid hormone (PTH) secretion from the parathyroid gland. Secreted PTH binds to PTH receptor 1 (PTH1R) on osteocytes to promote interaction between receptor activator of nuclear factor-kappa B (RANK) and RANK ligand (RANKL) on the osteoclast surface. This induces vacuolar-type adenosine triphosphatase (V-ATPase) production to facilitate bone resorption alongside carbonic anhydrase 2 (CA2). In contrast, bone accretion is facilitated by deposition of matrix proteins such as collagen type 1 alpha 1 (COL1A1). In the kidney, PTH stimulates inorganic phosphate (Pi) excretion and upregulates production of 1,25(OH)2D3. Bioactive 1,25(OH)2D3, which binds to vitamin D3 receptor (VDR), stimulates osteoclast activity, calcium transport in the kidney, and calcium and phosphorus uptake in the intestine. Impacts of 1,25(OH)2D3 in the shell gland and on paracellular intestinal calcium uptake still need to be elucidated. Transcellular transport of calcium in these tissues is thought to occur through ATPase plasma membrane calcium transporting 1, 2, and 4 (ATP2B1, ATB2B2, ATP2B4; intestine only), sodium-calcium exchanger 1 (NCX1), calbindin-28K (CALB1), transient receptor potential cation channels subfamily C member 1 (TRPC1; intestine only), transient receptor potential cation channels subfamily M member 7 (TRPM7; intestine only), and transient receptor potential cation channel subfamily V member two and six (TRPV2, intestine only; TRPV6, kidney only). Paracellular transport in the intestine is achieved by tight junction proteins 1, 2, and 3 (TJP1, TJP2, TJP3), claudin 2 and 12 (CLDN2, CLDN12) and occludin (OCLN). Transport of phosphorus in these tissues is thought to occur by sodium-dependent phosphorus transporters IIa and IIb (NaPiIIa and NaPiIIb) and sodium-dependent inorganic phosphorus transporters 1 and 2 (Pit1 and Pit2). Shell gland calcium transport by CALB1 may be under the control of estradiol (E2) through estrogen receptor (ER) interaction with estrogen-response elements (EREs) in its promoter region. Bone breakdown releases Pi into circulation, which induces production of fibroblast growth factor 23 (FGF23). In chickens and mammals, this peptide stimulates renal phosphorus excretion, which has been shown to be mediated through its binding to FGF23 receptors (FGFR1, FGFR2, FGFR3, FGFR4) and co-receptor klotho (KL) in mammals. In mice, FGF23 has also been shown to exhibit negative feedback on PTH and 1α-hydroxylase activity, as well as stimulate 24-hydroxylase activity. During periods of elevated iCa2+, calcitonin (CALC) is secreted from cells in ultimobranchial bodies to inhibit osteoclast activity in mammals, but its effects in birds are unclear. Further investigation into several of these processes and how transporters function in a tissue-specific manner is required to determine their role in calcium and phosphorus homeostasis in chickens. Parts of the figure were drawn by using pictures from servier medical art, licensed under a creative commons attribution 3.0 unported license (https://creativecommons.org/licenses/by/3.0/).*

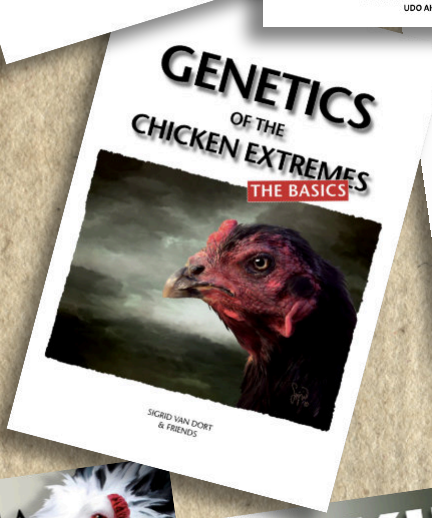
DUH

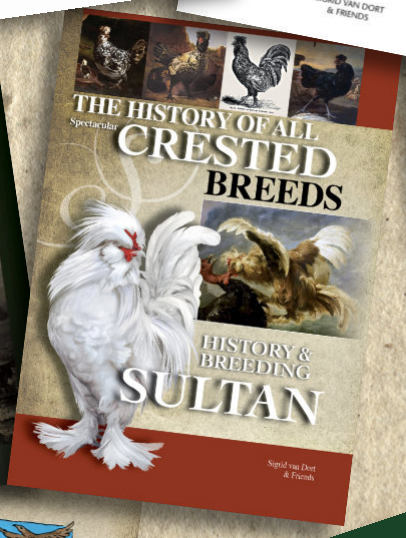
UH

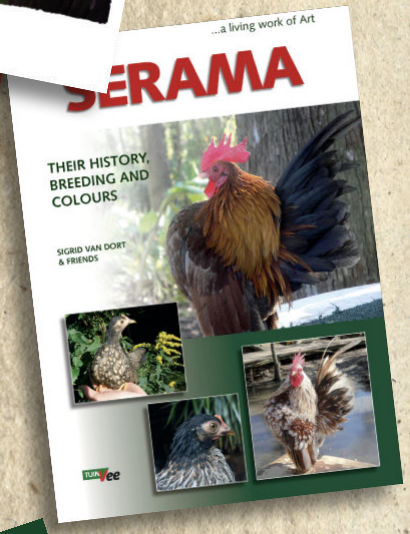
WTF

UNIQUE POULTRY BOOKS FOR CHICKEN ADDICTS









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