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TOWARDS THE IDENTIFICATION OF THE MOLECULAR PATHWAYS UNDERLYING THE ALTERATION OF CALCIUM METABOLISM IN HENS AT THE END OF LAY

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The current objective of the egg industry is to increase the hen's productive carrier up to 100 weeks. However, numerous studies have reported that egg quality and hen's bone strength deteriorate with age, because of altered calcium (Ca) homeostasis. This study was designed to make an integrative description of Ca homeostasis pathways in laying hens at peak and end of lay (23 and 90 weeks old). Brown laying hens aged 20 or 87 weeks were reared in individual cages to record oviposition and fed a commercial layer's diet containing 3.5 g/kg CaCO₃ (1/3 fine, 2/3 coarse particles). At 23 or 90 weeks of age, hens were euthanized at 0-1h, 9-10h or 18-19h post ovulation (PO) prior to the collection of plasma and tissue samples (6 hens per stage PO). Plasma samples served for the assay of iP and vitamin D metabolites. RNA were prepared from parathyroid gland (PG), liver, jejunum, medullary bone (MB) and kidney for a quantitative study of candidate gene expression. All data were analyzed using a linear model by robust regression. The effects of the main factors PO stage, age and the interaction were tested. In case of significant effects in the model ($P < 0.05$), all pairwise comparisons were performed using the least-square means method and a Tukey adjustment. Parathyroid hormone (PTH) gene expression in the PG did not vary with age, while the expression of several Ca transporters and vitamin D receptor (VDR) in the jejunum, and that of the transient receptor potential channel subfamily V member 5 (TRPV5), mediating Ca reabsorption in the kidney, decreased with age ($p < 0.05$). This could explain the previously reported reduced Ca retention in older hens. The higher expression of the Carbonic Anhydrase 2 (CA2) gene ($p < 0.05$) and the lower expression of collagen type I alpha 1 chain (COL1A1) gene ($p < 0.05$) in the MB also suggested exacerbated bone resorption and impaired bone accretion in the older hens. An increased expression of Fibroblast growth Factor 23 (FGF23) was also observed in the MB. This likely triggered the decreased plasma levels of 1.25(OH)₂D₃ and expression of target genes under its regulation. Our data highlights the molecular mechanisms underlying the osteoporotic syndrome previously documented in aged laying hens, thus providing new perspectives for future dietary interventions.