

Skeletal Endocrinology a Field of Medicine where Evolutionary advantage meets Illness

David Williams*

Editorial office, Journal of Bone Research, Spain

EDITORIAL NOTE

Skeleton's ability to release endocrine signalling chemicals, the skeleton can control whole-body homeostasis. Although bone-derived hormones provide a variety of adaptive benefits, their physiological roles come with trade-offs that can lead to disease. The skeleton is in charge of maintaining homeostasis. The term "homeostasis" refers to the way regulatory mechanisms keep specific variables within a set range. A sensor measures a variable's current condition and compares it to a specified ideal range. If the status quo and the set point are not consistent, homeostatic mechanisms are initiated to restore the set point. These counter-regulatory processes might include a variety of tissues and organs, although the majority of them rely heavily on endocrine mediators.

Bone actively participates in maintaining homeostasis in amniotes (clade of four-limbed vertebrates that includes birds, reptiles, and mammals) via secreting endocrine signalling molecules, as has been obvious in recent years. While bone-derived endocrine signalling molecules have a number of evolutionary benefits, they also increase disease vulnerability, especially in rapidly changing environmental conditions. The evolution of two crucial skeletal hormones, FGF23 and osteocalcin, as well as their many benefits, will be discussed in the sections that follow.

Fibroblast growth factor 23 (FGF23) is a protein made by fibroblasts- FGF23 is mostly produced by osteocytes, however other biological sources have been described in the literature, including macrophages, cardiomyocytes, enterocytes, and kidney epithelial cells. Several systems, including transcriptional and posttranscriptional pathways, regulate FGF23 levels in the blood. Importantly, a hitherto unidentified (furin-like) protease may cleave intact FGF23 (iFGF23), resulting in N- and C-terminal fragments (nFGF23 and cFGF23, respectively), which may have biological activity distinct from the complete molecule.

FGF23 regulates hemodynamics for what reason?- The main regulator of fluid homeostasis, RAAS, regulates its activity primarily by volume sensing by specialised (juxtaglomerular) epithelial cells in the kidneys' vasa afferentia. When organ perfusion is insufficient, angiotensin II and aldosterone, the two principal effectors of the RAAS, encourage salt and water reabsorption, raising blood pressure to restore balance. FGF23 has been proven to have similar effects, which is surprising. Phosphate homeostasis is aided by these functions because:

- The vast majority of substances found in terrestrial vertebrates require prior filtration through the glomerulus
- This filtration capacity is primarily regulated by modulation of intra glomerular blood pressure
- An increase in glomerular pressure will result in enhanced filtration and eventually excretion, contributing to phosphate homeostasis

Osteocalcin (OCN) is the most prevalent non-collagenous protein found in the bone matrix, and it is mostly produced by osteoblasts. OCN is made up of 46 to 50 amino acids and undergoes posttranslational changes such as vitamin K-dependent gamma- carboxylation, which are important for its skeletal activities. Mild changes in OCN knock-out animals were characterised by increased bone production rather than decreased bone mass, implying that the protein plays a negative regulatory role in skeletal homeostasis.

Correspondence to: David Williams, Editorial office, Journal of Bone Research, Spain, E-mail: williams_d@gmail.com

Received: December 19, 2021; **Accepted:** December 25, 2021; **Published:** December 31, 2021

Citation: Williams D (2021) Skeletal Endocrinology is the Field of Medicine where Evolutionary advantage meets Illness. J Bone Res. 9:151.

Copyright:©2021 Williams D. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.