Journal of Bone Research
ISSN: 2572-4916
OPEN ACCESS Freely available online
Editorial

Where Evolutionary Advantage Meets Illness is Skeletal Endocrinology

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EDITORIAL NOTE

The skeleton's ability to release endocrine signalling molecules allows it to regulate whole-body homeostasis. Although bone-derived hormones have a number of adaptive benefits, their physiological functions also have trade-offs, which can lead to disease. The skeleton is accountable for ensuring homeostasis.

Homeostasis describes how regulatory mechanisms keep certain variables within a predetermined range. A sensor typically detects the present state of a variable and compares it to a predetermined ideal range. Homeostatic mechanisms are triggered to restore the set point if the status quo and the set point are not consistent. 10 Various tissues and organs may participate in these counter regulatory activities, but the majority of them rely largely on endocrine mediators. The central role of the highly sensitive Fibroblast Growth Factor 23 (FGF23) system in phosphate metabolism has become clear in recent years that bone actively participates in maintaining homeostasis in amniotes (clade of four-limbed vertebrates encompassing birds, reptiles, and mammals) through secreting endocrine signalling molecules. While bone-derived endocrine signalling molecules provide various evolutionary advantages, they also enhance disease vulnerability, particularly in quickly changing environmental settings. In the sections that follow, we'll look at the evolution of two important skeletal hormones, FGF23 and osteocalcin, as well as their various benefits.

Fibroblast growth factor 23 is a protein that is produced by fibroblasts

FGF23 is mostly produced by osteocytes, while other biological sources such as macrophages, cardiomyocytes, enterocytes, and kidney epithelial cells have been documented in the literature. FGF23 levels in the blood are controlled by a variety of processes, including transcriptional and posttranscriptional pathways. Importantly, intact FGF23 (iFGF23) can be cleaved by a previously unidentified (furin-like) protease, resulting in N- and C-terminal fragments (nFGF23 and cFGF23, respectively), which may have biological activities different from the entire molecule.

FGF23 regulates hemodynamics for what reason?

The principal regulator of fluid (i.e., extracellular volume) homeostasis, RAAS, regulates its activity primarily through volume sensing by specialised (juxtaglomerular) epithelial cells located in the kidneys' vasa afferentia. When organ perfusion is insufficient, the two primary effectors of RAAS, angiotensin II and aldosterone, promote salt and water reabsorption, raising blood pressure to restore balance. Surprisingly, FGF23 has been shown to have similar effects. These functions contribute to phosphate ho0meostasis 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 ultimately excretion, these functions contribute to phosphate homeostasis

Osteocalcin

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.

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

Citation: Williams D (2021) Where Evolutionary advantage meets illness is Skeletal Endocrinology. J Bone Res. 9:120.