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Male Reproduction & Bone Endocrine Regulation of Energy Metabolism

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Perspective

Typically, a specific organ or cell type is researched within the strict confines of vertebrate physiology. With the development of mouse genetics, which has revived the idea of a whole body study of physiology, this methodology has gradually transformed. Skeletal physiology is a striking illustration of how mouse genetics has significantly influenced our comprehension of physiology. According to a genetic approach to bone physiology, the chemical osteocalcin released by osteoblasts makes bone a real endocrine organ that controls male reproduction and energy consumption. A growing number of auxiliary organs are being connected to bone as a result of the on-going corpus of research that displaces bone from its conventional roles [1]. These brand-new, significant hormonal links between energy metabolism, reproduction, and bone function highlight the idea of functional reliance in physiology. A science having two identities is physiology. Its most recent name is also its most well-known one today: molecular physiology [2]. This form of physiology examines how one protein or a collection of proteins, whether they are transcription factors, receptors, or ion channels, work in a specific cell type. However, there has been another physiology that predates molecular physiology and was for the longest time condemned by it to the attic of biology [3]. This is the whole-organism physiology, which was only well characterised by three titans in the field: Claude Bernard, Walter Cannon, who developed the idea of homeostasis, and L.J. Henderson, who posited that organs and functions are interdependent. These two ideas, particularly the final. They could be had. But practically everything Claude Bernard said has turned out to be accurate. Likely, physiology wouldn't exist without the concept of homeostasis. We will revisit the concept of mutual dependence's broad implications below. Ironically, the most complex development in molecular biology—the ability to inactivate a single gene at a time, in a single type of cell at a time, and in a way that can be induced—has revived whole-organism physiology. Because all or most organs interact with one another, the foundation of whole-organism physiology necessitates testing in a living animal where only one molecule is taken from just one type of cell and after embryonic development is complete. These days, this is possible. The role of bone as an endocrine regulator of energy metabolism, reproduction, and now of other processes has become progressively defined and understood molecularly over the past fifteen years in my research [4]. A variety of clinical data relating bone modelling and remodelling to the regulation of energy metabolism and fertility led to the start of this work. A justification for the working hypothesis that bone mass, energy metabolism, or at least certain elements of it, and reproduction may all be governed by the same hormones was that gonadal failure favours the formation of osteoporosis while obesity seems to guard against it and its harmful effects. This theory wasn't just founded on an interpretation, as would be shown later [5]. We first investigated if a hormone likes leptin, which is produced by adipocytes and controls appetite, energy expenditure, and reproduction, affected bone mass. Years of research have demonstrated that leptin is a potent inhibitor of bone mass accumulation in both mice and humans. The identification of osteocalcin hormonal actions led to the emergence of a number of crucial biological and medical issues. Clarifying the signalling processes that this hormone triggers in target cells is one of the most important of these. The discovery of a particular osteocalcin receptor was necessary in order to answer this question [6]. The existence of additional roles for osteocalcin, like many hormones, outside its influence on energy metabolism, was the subject of a second, more general question. The well-known control of bone remodelling by gonads offers an excellent context for answering the previously mentioned query. The idea that bone affects fertility was given significant conceptual weight in light of this hunch. A notable characteristic of mutant mice lacking osteocalcin admittedly considerably aided testing this theory in vivo. Although male mutant mice were not very good breeders, whether their partners were WT or Osteocalcin-deficient, female Osteocalcin-deficient mice were typically fertile. The availability of gain allowed for a more thorough and convincing argument to be made that this phenotype represents a legitimate biological function of osteocalcin, just as it did for energy metabolism. Although the aforementioned result could not be replicated, Gprc6A was formally recognised as an osteocalcin receptor present in Leydig cells based on a number of criteria. Oury and co. Osteocalcin directly binds to WT but not to GPRc6a-deficient Leydig cells, and it also causes WT but not GPRc6a-deficient Leydig cells to produce more cAMP. Third, and most importantly, a Leydig cell-specific deletion of GPRc6a revealed a reproduction phenotype brought on by low testosterone production that was comparable to, if not the same as, the reproduction phenotype seen in cases of osteocalcin inactivation. Fourth, in an even more convincing experiment, compound heterozygous mice lacking one copy of Osteocalcin and one copy of Gprc6a had a reproduction phenotype that was identical in every way to The discovery that CREB is a transcriptional effector of osteocalcin regulation followed the identification of GPRc6A as an osteocalcin receptor. The most important of them is to determine other roles for osteocalcin now that Gprc6a has been identified. Additionally, it enables us to conduct a more in-depth dissection of osteocalcin's molecular method of action in both known and unidentified target cells. The identification of osteocalcin's hormonal actions led to the emergence of a number of crucial biological and medical issues. Clarifying the signalling processes that this hormone triggers in target cells is one of the most important of these. The discovery of a particular osteocalcin receptor was necessary in order to answer this question. Determine whether osteocalcin, a second question with considerably greater implications, similar to many hormones, has purposes beyond from those related to energy metabolism. The well-known role of

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gonads in the regulation of bone remodelling makes this issue the perfect place to start. The idea that bone affects fertility was given significant conceptual weight in light of this hunch. This medical finding indicates that gonads, mostly through sex hormones, influence the functioning of bone cells in one way or another. We won't address bone physiology in this article. The gonads' control over bone mass accumulation suggested that bone, in the course of its endocrine activity, may influence the reproductive processes in one or both genders. As long as the model species are vertebrates, this work shows how powerful the idea of functional dependence in physiology is and how intricately intertwined all the organs and genetic direction are. It also offers a great tool to integrate various physiologies, and therefore many medical fields. Mouse genetics is the most effective method available today and for the foreseeable future for mapping and studying all the interorgan linkages present in vertebrates. Although all of the skeleton's functions are not fully understood, the ones that are suggest that skeleton physiology influences much more than just the skeleton itself, since it already has an impact on fertility, energy expenditure, and glucose homeostasis. Last but not least, and this is a significant result of our work, if the skeleton. In the first step, we investigated how osteocalcin affects two target cells, the b-cell of the pancreas and the Leydig cell of the testis, in the signal transduction pathway. Using this method, it was discovered that osteocalcin only consistently induced the generation of cAMP in these two cell types. This data, according to our interpretation, indicates that the osteocalcin receptor, or another receptor, is most likely a G protein-coupled receptor connected to adenylate cyclase. As a result, the second step of this experimental approach makes use of the dichotomy in osteocalcin function between boys and girls.

References

1. Jeong JH (2008) Expression of Runx2 transcription factor in non-skeletal tissues, sperm and brain. J Cell Physiol 217: 511-517.
2. Gonzaga MFM, De Castro LF, Naves LA, Mendonça JL, Oton De Lima B, et al. (2018) Prolactinomas Resistant to Treatment With Dopamine Agonists: Long-Term Follow-Up of Six Cases. Front Endocrinol 9: 625.
3. Holmberg CI (2001) Phosphorylation of serine 230 promotes inducible transcriptional activity of heat shock factor 1. EMBO J 20: 3800-3810.
4. Baker BM, Handorf AM, Ionescu LC, Li WJ, Mauck RL, et al. (2009) New directions in nanofibrous scaffolds for soft tissue engineering and regeneration. Expert Rev Med Dev 6: 515-532.
5. Legiran S, Brandi ML (2012) Bone mass regulation of leptin and postmenopausal osteoporosis with obesity. Clin Cases Miner Bone Meta 9: 145-149.
6. Ahn N, Kim K (2016) Effects of 12-week exercise training on osteocalcin, high-sensitivity C-reactive protein concentrations, and insulin resistance in elderly females with osteoporosis. J Phys Ther Sci 28: 2227-2231.