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MEDIATION OF ACUTE STRESS RESPONSES BY OSTEOCALCIN

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Introduction. The complex dynamic balance or homeostasis of all organisms constantly challenged by internal or external adverse forces called stressors should be maintained. Stress comes when homeostasis is threatened or seen as such; homeostasis is restored with various adaptive responses in physiology and behavioral adaptive responses. If any stressor exceeds some severity or time threshold, a compensatory response that functionally matches the stressor is activated by the adaptive homeostatic systems of the organism. When coordinating this cycle, the stress system has mechanism that ensures it homeostatic balance.

Aim of study is to however answer the question around the endocrine functions of the bone as to why an organ-bone, can be seen as an endocrine organ having an endocrine function. It should be noted that the acute stress response (ASR) is an evolution maintained physiological process aimed at maintaining or restoring homeostasis in animals at immediate risk.

Materials and Methods: Osteocalcin plays an independent role of the signaling of corticosterone and catecholamine, but however, osteoblasts (in bone tissue) need glutamate to increase bioactive osteocalcin before it could be released. Osteocalcin is needed by signaling through its receiver for the production of several main ASR manifestations. G protein-coupled receptor family C group 6 member A (GPRC6A) (which functions as a receptor of L α-amino acids, cations (calcium), osteocalcin, and steroids. It is also a membrane androgen receptor). Also testing whether any protein made in bone is affected during ASR showed that circulating levels of osteocalcin increased significantly after electrical foot shocks in 2- to 6-month-old mice, in both sexes, in two different genetic backgrounds and at two different times of the day. Circulating bioactive osteocalcin levels also rose in rats after restraint and in humans submitted to a public speaking and cross-examination stress that increased heart rate and blood pressure.

Results: Bioactive Osteocalcin is released during an ASR from the osteoblast cells: sensitivity to stressors enhance the circulating bioactive osteocalcin levels, and

they occur irrespective of sympathetic signage via b-adrenergic receptor. Apart from the conventional pathways of osteocalcin synthesis, scientist looked for other pathways that would account for the release of bioactive osteocalcin during an ASR into general circulation. Osteocalcin is activated via decarboxylation, a bone resorption lacunae response. Glutamate mediates bioactive osteocalcin stressor-induced release from osteoblasts: In order for hormonal responses to be released into the body, there must be a neuro-regulatory agent from the brain, more like a neurotransmitter, only glutamate among all the neurotransmitters tested significantly increased the amount of bioactive osteocalcin contained in the osteoblast supernatant of the mouse. A specific glutamate transporter, Eaat1 or Glast is expressed in osteoblasts at three rates of magnitude lower than any other glutamate transporter and any other cell type studied, except osteoclasts. In several ways, the role of glutamate transport in osteoblasts for osteocalcin release was described . Osteocalcin inhibition of the parasympathetic tone triggers an ASR. An investigation on the interaction between osteocalcin and the autonomous nervous system's sympathetic arm because the sympathetic tone is an ASR mediator and osteocalcin prefer catecholamine synthesis-norepinephrine-in the brain. Research shows, however, that circulating norepinephrine, its urinary secretion, its lung output, Ucp1 expression in brown fat, protein content in the brain, phospho-CREB aggregation (an indicator for adrenergic signaling) in the heart, and expression of multiple adrenergic receptors in trachea and core were all identical in WT littermates. Also, intravenous administration of osteocalcin caused a rapid and significant decrease in the hepatic parasympathetic nerve activity measured in WT mice. Thus, bioactive osteocalcin appears to regulate the function of post-ganglionic parasympathetic neurons.

Regulation of osteocalcin secretion by stressors: The first feature of a stress hormone that within minutes of exposure to stressors its circulating levels spikes. After testing for all stressors, the increase in circulating osteocalcin was observed and it extends from rodents to humans. Osteocalcin's specificity between bone-synthesized hormones and molecules that occurs after stressors in the amygdala and probably other areas of the brain is that it is independent of glucocorticoid, sympathetic stimuli, and bone resorption. The rise in circulating osteocalcin related to stressors is more likely to rely on glutamate reaching the osteoblast of mice by means of the Gl transporter.

Conclusion: The ability of osteocalcin to facilitate ASR, favor memory, and increase muscle function during exercise indicates that osteocalcin provides an advantage over the survival of bony vertebrate in a hostile environment, such as the wild environment [5]. In the younger rodents and primates with high circulating levels of osteocalcin, this is particularly important. Osteocalcin is a fitness hormone for these younger animals. An unusual characteristic of osteocalcin is the indirect performance of all its functions. Osteocalcin is needed not only to uprule the aforesaid physiological functions; it is also interesting to know that research have proven that it could reverse aging events in the brain and muscle. Osteocalcin is needed not only to uprule the aforesaid physiological functions; it is also interesting to know that research have proven that it could reverse aging events in the brain and muscle. This capacity of exogenous osteocalcin to counteract objective symptoms of aging such as memory loss and reduced muscle function in the mouse, together with the fact that its circulating rates decline with age as memory, muscle function and fertility all drop, indicate that osteocalcin is also an antigeronic hormone.