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Commentary

Association of Chemerin Level with the Risk for Osteoporotic Fracture

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DESCRIPTION

Osteoporosis is a disease adversely affecting bone health and consequently resulting in an increased risk of bone fractures during which hip fracture is recognized as the most serious consequence of osteoporosis. With the aging global population, the incidence of osteoporotic fractures markedly increased in the elderly, with high resultant morbidity and mortality within the elderly population. Nowadays, it's turning into a serious public health concern worldwide, particularly in Asia. Osteoporosis is characterized by decreased bone strength and low bone mass, so its risk prediction is largely supported by the baseline of Bone Mineral Density (BMD).

Although regional and social variations can be observed in the frequency of osteoporotic fracture and BMD values, there was a relationship between BMD value and osteoporotic fracture. Compared with BMD alone, its combination with other powerful risk factors might improve the identification accuracy for individuals at high fracture risk [1]. It's so necessary to explore extra biomarkers which will improve fracture risk prediction independent of, or combined with, BMD. Adipocytes and osteoblasts secrete various fatty tissue-derived adipokines that regulate bone formation and resorption. As a recently discovered adipocyte, chemerin had a cross-sectional association with bone health and it was also a protein-ligand for the G protein-coupled receptor chemokine-like receptor 1 (CMKLR1) that modulated adipogenesis *via* binding to C-C motif chemokine receptor-like 2 (CCRL2) and G-protein-coupled receptor 1, however, the pharmacology and signaling properties of those chemerin receptors have been much less characterized.

Fracture risk can be accumulated in people with extraordinarily low calcium intake, but a meta-analysis of experimental studies relating calcium intake to fracture risk did not show any association between calcium and hip fracture. The active hormonal form of vitamin D was a principal regulator of calcium and phosphorus homeostasis through actions on the gut and resorption of calcium from bones, consequently determining the amount of renal excretion of those substances. Chemerin has an important role in regulating bone metabolism, as demonstrated by a previous study that high chemerin levels decreased the bone mass peak, thereby promoting age-related bone loss. Epidemiological studies verified that chemerin may need a relationship with osteoporosis and BMD which confirmed that body fluid chemerin level was inversely correlated to BMD in the lumbar spine and femoral neck [2,3].

A recent study advised that there was a positive association between chemerin level and a medium or high risk for osteoporotic fractures in obese men and women of the northeastern German population, which may be due to a chemerin-induced negative impact on bone metabolism *via* abrogation of osteoblastogenesis or stimulation of adipogenesis. So, it can be speculated that high serum chemerin levels are severally associated with stronger restrictive bone formation and stronger supporting bone resorption.

Chemerin was chiefly secreted into the extracellular medium from fat and different tissues. The secreted chemerin activated CMKLR1, a G supermolecule coupled receptor expressed in various cell types, particularly in mesenchymal stem cells, consequently resulting in accumulated adipogenesis and reduced osteoblastogenesis. Conversely, stimulation of CCRL2, a non-signaling receptor for chemerin, appeared to not promote any signaling within the cells or induce receptor internalization. Compared with control groups, the transcription levels of CMKLR1 and CCRL2 receptors related to chemerin didn't take issue considerably within the hFOB1 [4]. Thereby, these chemerin receptors underlying the regulation of bone metabolism still need to be explored in future analysis.

Bone is a dynamic tissue, which was remodeled continuously through the removal of mineralized bone by osteoclasts, the bone-resorbing cells, and the formation of new bone *via* the osteoblasts, the bone-forming cells. The crucial role of BTM, the markers for osteocalcin formation (BAP and P1NP) and for bone resorption (β-CTX and NTX) is confirmed to be independently predictive values of fracture risks. This was according to our results that the serum chemerin level was completely related to bone metabolisms connected markers, like P1NP, and β-CTx. Additionally, osteoblast differentiation was commonly monitored by a series of markers and therefore the most

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frequently used markers were COLA1, ALP, osteopontin, bone sialoprotein, osteocalcin, and endocrine gland hormone-related protein receptor, and RUNX2 [5]. RUNX2 was a master gene for the differentiation of mesenchymal stem cells into osteoblasts, and it reached its highest level in immature osteoblasts, yet decreased during osteoblast maturation. In older age, osteoporosis resulted from the increased bone resorption and decreased bone formation, the rates of which might be evaluated by assaying serum and urinary bone matrix components.

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