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Osteoimmunology and Nutritional Science

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The concept of osteoimmunology was established more than a decade ago and is based on the reciprocal relationship between immune and bone cells. This way of looking to these two organs skeletal and immune system, has resulted in the development of clinical therapeutics for seemingly disparate but linked by the common themes of inflammation and bone remodeling.[1] Osteoimmunology includes osteoporosis, rheumatoid arthritis and periodontal disease which have prominent effects on quality of life, increase the incidence in the population, and have crucial socioeconomic issues.[2] Immune system and bone are both intricate organs which have different tasks.[3-5] To preserve balance in bone, while responding to different inputs (such as nutrition, mechanical stress, ageing, and inflammation), the bone marrow cells are controlled by immune systems in concert with endocrine and neural systems.[5] As a fact, if we look at the bone marrow space as "loosely compartmentalized lymphoid organ" there would be no doubt in intensive interact and influential effects between these two systems.[6] In 1997, the receptor activator of the nuclear factor-kappa-B ligand (RANKL)/RANK/Osteoprotegerin (OPG) pathway was recognized as an important signal transduction pathway that regulate the coupling mechanism between osteoblasts and osteoclasts or bone remodeling process.[3]

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As we mentioned above, one of the inputs which can disturb the bone homeostasis, is nutritional diet.

The close relationship between diet and bone has long been established. Calcium, phosphate, and vitamin D are essential for normal bone structure and function. Protein, calories, and other micronutrients such as boron, copper, fluoride, iron, manganese, zinc and some vitamins such as, K and A also help developing and maintaining bone.[7] Several clinical studies also mentioned that products of lipid and lipoprotein oxidation may interact in pathophysiology of osteoporosis as a component of osteoimmunology.[8-18] High fat diet and its outcome hyperlipidemia and the subsequent effects of increased oxidized lipid level, can induce osteoclasts differentiation and inhibit the differentiation of osteblasts.[12] T lymphocytes exposed to oxidized lipoprotein secret RANKL, a key mediator of osteoclast differentiation, and have higher RANKL:OPG ratio which could disrupt bone remodeling process. On the other hand, oxysterol levels have crucial roles in the cross-talk between lipid metabolism and immune regulation.[19] Oxysterols are formed by a) auto oxidation, as a secondary byproduct of lipid per oxidation, b) enzymatic pathways by specific monooxygenases, most are members of the cytochrome P450 family of enzymes. They also may be derived from the diet. Oxysterols interact in physiologic and pathologic processes such as cellular differentiation, inflammation, apoptosis, steroid production and atherogenesis. Specific oxysterols induce the osteoblastic differentiation of marrow stromal cells and have osteogenic effects.[20]

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By this introduction, understanding infrastructure of bone loss induced by hyperlipidemia is a controversial subject that needs more studies to elucidate different aspects of relationship between osteoimmunology and nutritional science.

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