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Perspective

The Effectiveness of Physical Activity on Osteoporosis

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DESCRIPTION

Osteoporosis is the loss of calcium and further minerals from a person's bones, which makes the bones prone to fracturing (breaking). Women are probably to possess osteoporosis as the hormonal changes of menopause build bone loss. A nutritious diet, as well as calcium-rich foods and regular exercise throughout a person's life (including throughout childhood and adolescence); can reduce the risk of osteoporosis in later years. People with existing osteoporosis may get benefit from exercise. Physical exercise regularly will slow down the speed of bone loss which reduces the danger of fractures from osteoporosis.

Osteoporosis is a skeletal disease characterized by low bone mass or Bone Mineral Density (BMD), deterioration of bone micro-architecture, and accumulated risk of fracture. The fast growth of the aging population has been a concern in several aspects of human health, and among this osteoporosis has been one of the most public health issues in aging people, especially for people aged above 55 years [1]. The exercise or physical training may improve bone mass and strength and consequently promote bone formation, which might effectively treat and prevent osteoporosis with no side effects. Thus, exercise has been suggested by WHO as a therapy for non-drug osteoporosis prevention and treatment. Exercise or physical activities built multiple mechanical loadings, like tension, compressive, and fluid shear stress, that have useful effects on reducing bone loss, increasing bone strength and preventing osteoporosis in aging people. Studies have shown that the increased forces impacting the body throughout exercise correlate with elevated bone mass density and bone strength in athletes.

Bone is extremely vascularized tissue with a large network of blood vessels and capillaries that provide oxygen and nutrients for bone formation and development, which is mediated *via* the regulation of various signaling pathways between epithelial tissue cells and bone cells [2]. Blood vessels play major roles within the method of osteoporosis and are formed *via* 2 distinct biological processes. Within the early stages of embryogenesis, hemangioblasts are derived from mesodermal cells, which migrate to a particular site and combine to create the primary vessels within the process of vasculogenesis. Most of the new blood vessels sprout by the process of angiogenesis, which is followed by an enlargement of the existing vascular networks through multiple steps like epithelial tissue cell proliferation, migration, development of vessel pruning, and colligation. Thus, it seems that the vasculature in bone is created chiefly by ontogenesis.

Bone formation takes place in 2 different ways: One is endochondral ossification and the alternative is intramembranous ossification. Endochondral bone formation needs the availability of bone-forming osteoblasts, and progressive neovascularization attended with growing bone. Overall, bone formation happens during a spatial and temporal relationship with vascularization of the ossifying tissue, which is named angiogenesis-osteogenesis coupling [3,4]. Within the process of angiogenesis, epithelial cells proliferate, migrate, form tubes, and eventually turn out conduits wherever blood flows and provides the mandatory nutrients, oxygen, growth factors, and hormones for the bone cells. Hematopoietic precursors of osteoclasts are transmitted by blood vessels to the sites of cartilage and bone resorption so as to eliminate the end-products of the degraded extracellular matrix. Moreover, the subendothelial walls of vessels contain pericytes, which seem to be a vital cell-attached the coupling between osteogenesis and angiogenesis.

Angiogenesis within the bone microenvironment is needed for bone growth and development, postfracture repair, and maintenance of normal bone health. As an example, Holen and Coleman found that the blood supply within the people with osteoporosis or osteopenia is comparatively below that within the people with normal bone mass, indicating that bone blood supply and bone mineral density are extremely correlated [5]. Notch signaling in epithelial tissue cells seems to be attached to the age-dependent regulation of hematopoietic stem cell niches in bone, which accompanied by increase in the variety of CD31-positive capillaries, Platelet-Derived Growth Factor Receptor-β (PDGFRβ)-positive perivascular cells, and arteriole formation. Exercise or physical training can prevent osteoporosis in the older as a non-drug preventive strategy. The interaction of mechanical loading, hormones or cytokines, and signaling pathways induced by exercise accumulated bone formation and reduced bone resorption, resulting in a healthy skeleton.

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Dysregulation of bone angiogenesis is related to several bone diseases like osteoporosis, and exercise improves angiogenesis in bone *via* the regulation of key angiogenic mediators.

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