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Perspective

Swimming: One of the Treatments for Osteoporosis

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DESCRIPTION

Osteoporosis is a musculoskeletal condition that is characterised by decreasing bone mass and damage to the bone's microstructure. Age, genetics, hormone therapy, and prolonged bed rest are a few reasons for osteoporosis. According to epidemiology, males over the age of 50 and women in their postmenopausal and premenopausal stages are most likely to develop osteoporosis. Older individuals are particularly vulnerable to osteoporotic fracture, and hip fracture is sometimes referred to be the "final fracture" in life. Fractures also affect 20% of males and 30% of women over the age of 50. Human glands undergo a variety of changes as we age, which alter hormone secretion. These alterations disrupt the body's initial balance and impair the function of several organs, including the bones. This disorder mostly affects the skeleton and appears as an increase or decrease in the activities of osteoclasts and osteoblasts, which causes a reduction in Bone Mineral Density (BMD). Furthermore, one should not undervalue the impact of diminished mobility on the skeleton. For instance, the reduced movement might cause skeletal muscle atrophy, which raises the chance of breaking an arm. The uses of monoclonal antibodies to disrupt the signal molecules that osteoblasts or osteoclasts employ to promote respective roles have enhanced pharmacological treatment for osteoporosis currently.

Weight lifting, performing plyometric exercises, or engaging in other high-impact activities are all examples of exercise therapy for osteoporosis that tries to improve the bone's ability to withstand a significant amount of strain and tension. Individualized care should be provided for people of all ages. For example, osteoporosis patients should be given the proper load-bearing and tension to stop further bone loss and consequent injuries such as fractures. Intensive exercise is not recommended for people with osteoporosis who also have cardiovascular and cerebrovascular problems since they do so frequently. Theoretically, bone stimulation can encourage osteogenesis and increase bone mass, even though it is favourably connected with exercise intensity within a specific range. All organs, including the bone, are less receptive to external stimuli in osteoporosis patients than in healthy individuals. Although recent research has revealed that swimming has no apparent impact on enhancing bone mass in these individuals, swimming is expected to become an appropriate physical activity to prevent bone loss in osteoporosis patients. Currently, weight-bearing and non-weight-bearing sports can be roughly divided. The National Osteoporosis Foundation of the United States recommended performing high- and low-intensity weight-bearing exercises simultaneously for skeletal load for at least 30 minutes per day, five to seven days a week. Strong muscles can enhance the bones' supporting function. It can increase bone metabolism, prevent falls, and improve posture. All ages can participate in swimming, which is quickly gaining popularity with the general public. Swimming can enhance cardiovascular function, lower blood cholesterol levels, increase the body's antioxidant capacity, and slow the aging process.

Swimming uses low-intensity vibration to potentially prevent osteoporosis. Low-intensity mechanical signals have been found to increase bone growth in the absence of load which inhibits the production of fat and lower blood triglyceride levels. Additionally, mesenchymal stem cells and adipose cells are the progenitors of both osteoblasts and adipocytes. Adipocyte formation and bone resorption can be reversed in a certain way by low-intensity vibration. Low-intensity vibration can also reduce the negative effects of obesity on the immune system. Swimming is the most effective way to prevent and treat osteoporosis when considering side effects, but additional strategies are still required. Osteoporosis can also be effectively treated by suppressing osteoclasts and encouraging osteoblast formation and maturation. Monoclonal antibodies, parathyroid hormone (PTH), and Dickkopf WNT Signaling Pathway Inhibitor 1 are now successful treatments (DKK1). Tripamine, a 1-34 amino acid fragment of PTH, thus, has the ability to stop bone loss. Osteoblasts are primarily inhibited by DKK1 and sclerostin *via* the WNT signalling pathway. The humanised monoclonal antibody against DKK1 is now in the clinical trial stage. Clinical trials for Sclerostin's monoclonal antibody are now underway, and the results show that it has a very noticeable effect both in the preclinical and clinical trial stages.

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