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Opinion Article

Effect of Fluoride on Bone Health and its Treatment

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DESCRIPTION

Although Fluoride (F^-) is frequently disregarded and a persistent toxin, it poses concerns to an estimated 200 million highly exposed people worldwide, largely through drinking water. The skeletal system, which takes on almost 99% of the weight of F^-, is particularly vulnerable to its harmful consequences. The amount of F eaten, the timing of exposure, and the age of the affected person, the tissue metabolism, and hereditary factors all play a role in how much of it is assimilated by bone tissue. Numerous negative health effects of F^- exposure have been documented by the U.S. National Research Council and others, including changes in biochemical (e.g., inhibits enzyme action, including phosphatases) and physiological (e.g., bone quality and fracture risk) as well as cardiovascular, reproductive, endocrine, gastrointestinal, and neurological development. Fluoride easily combines with calcium and hydroxyapatite to generate fluorapatite by replacing the hydroxyls in the crystals. The effects of excessive F^- exposure on the physical and chemical characteristics of enamel cells, bone minerals, bone cells, and bone remodeling processes, including encouragement of osteoblast activity and delayed mineralization of new bone, have been studied extensively in both humans and animals. Chronic Skeletal Fluorosis (SF), a disease characterized by a variety of bone lesions, including osteosclerosis (i.e., increased bone density), osteoporosis, degenerative joint alterations, and ligament calcifications, is the end result of these numerous disorders. Additionally, investigations on animals have demonstrated that F^- may change the collagen and non-collagenous proteins that make up the bone matrix, affecting the flexibility and biomechanical integrity of the bone and decreasing bone strength (or quality).

Bone is made up of a network of cells and mineralized fibrils, as well as non-collagenous proteins and water, all of which work together to define the mechanical properties of the bone. As a result, it is challenging to describe bone quality exactly because it depends on a number of factors, such as bone density, features of its microstructure, and collagen composition, which affect the bone's resistance to fracture. Despite the fact that the effects of F^- on bone-forming and resorbing cells are well known, its impact on the quality of human bone is not well known or understood. Without taking into account changes to bone microstructure, collagen content, or elasticity caused by F^-, the diagnosis of SF continues to be based solely on conventional X-ray imaging for assessing bone density.

Fluoride (F) is easily absorbed from the stomach and small intestine, where it deposits in calcified tissues before being excreted through the urine. Although F's anabolic effect on bone mass has been extensively researched, there is still debate regarding the type of bone that is really created. Since F treatment can have a biphasic, dose-dependent action on bone cells, boosting bone growth at a low dose and being toxic at a high dose, evaluating its efficacy is challenging. F treatment for osteoporosis may be more successful if it is administered sooner using low-dose regimens that prevent hazardous levels and don't interfere with mineralization.

Although the F dose is very important, it is not the sole element affecting how F affects bone. Patients undergoing F treatment for osteoporosis make up about one-third of non-responders. Additionally, 5% of children in high F localities did not have dental fluorosis, and roughly 40% of individuals in places with naturally high F levels in water were unaffected by skeletal fluorosis. Genetic factors affect how sensitive trabecular bone is to anabolic and catabolic stimuli, which may help to explain why not all patients who receive F for low bone mass respond to the drug.

The fluoride ion's major impact is to promote the growth of new, mostly woven and inadequately mineralized bones. Accelerated bone resorption could happen concurrently. On radiographs, the pathologic alterations to the bones appear as varied degrees of osteoporosis, osteomalacia, and osteosclerosis. On radiographs of people with skeletal fluorosis, osteosclerosis, osteomalacia, or rickets, osteoporosis, ligament and tendon ossification, growth lines, and periosteal response may be visible.

Recent research has demonstrated that calcium, vitamin C, and vitamin D intake can effectively guard against fluoride poisoning. However, studies have indicated that vitamin C supplementation alone has no impact on skeletal fluorosis, and long-term use of analgesic and anti-inflammatory medications can have negative effects. The combination of methionine and

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vitamin E may be useful in the treatment of skeletal fluorosis. Methionine and vitamin E can lessen fluoride's negative effects on soft tissues and stop it from building up too much in bones. The management of skeletal fluorosis has also been demonstrated to be impacted by several nutrients. In mice given fluoride treatment, choline reduces damage to the chondrocyte matrix and to breakdown enzymes. Taurine also improves thyroid gland function and renal antioxidant status in rats, reversing the renal toxicity brought on by fluoride. Rat liver tissues and erythrocytes are shielded from fluoride-induced oxidative damage by pomegranate (*Punica granatum*) juice. According to the studies, fluoride harms not only bones but also other organs. According to additional research into the causes of the condition, there is now a theoretical foundation for treating skeletal fluorosis.