Opinion

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Exposure and Effects of Cadmium and Significant Risk Factors on Bone Tissue

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Introduction

Heavy metals are dense metals with a high atomic number and atomic weight, and most are hazardous even at modest dosages. Basic metals, transition metals, metalloids, actinides, and lanthanides are all heavy metals. Cadmium (Cd), lead (Pb), mercury (Hg), and chromium (Cr) are examples of common heavy metals (Cr). However, depending on the context, iron (Fe), copper (Cu), aluminium (Al), zinc (Zn), beryllium (Be), cobalt (Co), manganese (Mn), and arsenic (As) may also be considered heavy metals. Notably, whereas helpful heavy metals have negative effects only at high concentrations, harmful heavy metals can be hazardous even at low concentrations. Because of their toxicity and nonbiodegradability, as well as their ability to accumulate in the environment and in living beings, metal contamination is a significant concern all over the world [1].

Exposure to Cadmium

Industrial Practices: Commercial cadmium manufacturing did not commence until the early twentieth century. It was first used in electroplating, but it is now mostly used in nickel-cadmium batteries, pigments, coatings, and plating, as well as as a stabiliser in plastics and other materials (including nonferrous alloys, semiconductors and photovoltaic devices) [2]. Cadmium production and refining, nickel-cadmium battery manufacture, cadmium pigment manufacture and formulation, cadmium alloy production, mechanical plating, zinc smelting, brazing with silver-cadmium silver alloy solder, and polyvinylchloride compounding have the highest potential occupational exposures. Smelting of non-ferrous metal ores, fossil fuel burning, ferrous metal manufacturing, municipal waste incineration, and cement manufacture are the primary anthropogenic sources of cadmium in the atmosphere.

Electronic and electrical waste (e-waste) disposal and recycling has also been highlighted as a possible source of cadmium exposure, particularly for youngsters. Smelting and mining operations, as well as the effluent generated by air pollution control, pollute the aquatic environment (gas scrubbers, in the absence of strict control measures) [3].

Food and drinking-water: Cadmium may be taken up by some crops and aquatic creatures and accumulate in the food chain if it is present in soil and water. For nonsmokers, food is the primary source of cadmium in the environment. Cadmium levels are highest in the kidney and liver of mammals given cadmium-rich diets, as well as in some oyster, scallop, mussel, and crab species. Vegetables, grains, and starchy roots have lower cadmium amounts. Cereals/grains, vegetables, beef and poultry organ meats, and seafood are the food groups that contribute the most to cadmium exposure in various nations when consumption is taken into consideration (especially mollusks).

Smoking: The leaves of the tobacco plant naturally acquire rather high levels of cadmium. As a result, smoking tobacco is a significant source of exposure, and heavy smokers' daily consumption may exceed that obtained through diet. Cigarette smoking can produce large increases in cadmium concentrations in the kidney, which is the major organ affected by cadmium poisoning [4].

Products: Cheap jewellery, toys, and plastics can be important sources of cadmium exposure, especially for children; however, several nations have taken steps to limit or prohibit the use of cadmium in such goods.

Cadmium Toxicity in Bone Tissue

Cadmium poisoning can cause problems with calcium metabolism, the production of kidney stones, and bone deterioration. Osteomalacia (softening of the bones) and osteoporosis can occur in those who are exposed to cadmium through living or working in contaminated areas; for example, itai-itai disease (characterised by osteomalacia, osteoporosis, painful bone

fractures, and kidney dysfunction) was once common in a region of Japan where soil was contaminated with cadmium from zinc/lead mines [5].

Chronic exposure to Cd reduces the mineralization of vertebral bodies, changing their biomechanical characteristics and making them more vulnerable to deformation and fracture, according to in vivo investigations in experimental animals. Cd also affects bone formation and mineralization by lowering the expression of markers of osteoblastic development (Runx2, osteocalcin), extracellular bone matrix proteins (type I collagen), and enzymes involved in the mineralization process (alkaline phosphatase-ALP). Other studies show that prolonged Cd exposure reduces bone volume and increases the percentage of tartrate resistant acid phosphatase (TRAP) positive cells in the subchondral tibial bone; the increase in TRAP activity would indicate that the increased resorption causes osteopenia. The percentage of fatty bone marrow also increased, suggesting that Cd inhibits mesenchymal cell differentiation to osteoblasts, promoting adipogenesis. Cd has various impacts on different kinds of bone, according to the same research (long bones vs. craniofacial bone).

In vitro investigations have demonstrated that Cd promotes the development of osteoclasts in cocultures of osteoblasts and osteoclast precursor cells by increasing RANKL expression, TRAP activity, and generation of TRAP-positive cells in the presence of RANKL. According to the findings, Cd may influence RANKL expression in osteoblasts and hence indirectly stimulate osteoclastogenesis through RANKL. Cd has also been found to cause osteoblast apoptosis by altering the cytoskeleton, as well as DNA fragmentation, an increase in the frequency of micronuclei and nuclear bridges and an increase in reactive oxygen species by activating the p38 MAPK pathway and inhibiting the Erk1/2 pathway.

Bone tissue is constantly remodelled throughout one's life. The resorption, synthesis, and mineralization of the bone matrix are all coordinated in this process. Overall, metals offer two issues: direct toxicity on bone cells on the one hand, and buildup in the bone matrix on the other. Their direct toxicity mostly affects osteoblasts, reducing differentiation, synthesis activity, and extracellular matrix mineralization. The metal has a different impact on osteoclasts, boosting or reducing TRAP enzyme activity and suppressing precursor development [6].

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