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Editorial

Cadmium and Bone Disorders-Current State of Knowledge

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Editorial

Disorders in the skeleton can be connected with different lifestyle, dietary and environmental factors [1]. Cadmium (Cd), one of the most important environmental and occupational pollutants, is known to produce many toxic effects, one of them being the effect on bones. Thus, Cd-induced multiple bone fractures as a consequence of ostemalacia and osteoporosis were observed in Itai-Itai disease in Japan when general population was exposed to high dietary Cd intake through rice resulting in severe kidney and bones impairments, particularly in postmenopausal women. Compelling evidence also confirms correlation between increased Cd industrial exposure and bone mineral loss and increased skeletal fragility in humans [1-4].

The question of the estimation of the risk of bones fractures caused by low lifetime exposure to this metal was raised in the last decades. Data obtained from experimental studies indicate that even relatively low chronic exposure to Cd may pose a risk for bones. In a study performed by Brzóska [5] life time exposure to Cd in doses corresponding to human exposure in non-Cd polluted areas decreases mineralization of long bones and weakens their strength in female rats. The influence of Cd exposure during skeletal development on the risk of bone fractures at the stage of skeletal maturity was also proved in female rat model. In this study Brzóska et al. [6] demonstrated weakening of mechanical properties of tibia in mature rats as a consequence of exposure to low Cd doses (corresponding to low human environmental exposure) during their development. On the other hand, the study performed on male rats showed that male rats are less susceptible to bone disorders than female rats since only higher doses of Cd corresponding to relatively high environmental or occupational exposure to Cd produced markedly increased bone susceptibility to fractures in male rats [7].

However, the mechanisms of Cd toxicity in bones are not fully elucidated. Experimental studies proposed at least two different mechanisms of Cd-induced bone disorders: first one being its direct impact on bone cells by stimulating osteoclasts differentiation and activity [8,9] and the second one being its effect on the gastrointestinal tract and kidneys thus resulting in disturbances in the metabolism of vitamin D and bone-associated minerals. Brzóska and Moniuszko-Jakoniuk [10] have proved that lifetime exposure of female rats to low doses of Cd affected the metabolism and function of calciotropic hormones which are known to have a crucial role in the regulation of Ca and phosphate homeostasis and significantly influence bone metabolism [11]. Furthermore, the role of oxidative stress as a mechanism of Cd toxicity in many organs, as recently reviewed by Matović et al. [12,13], was also investigated in Cd-induced bone damage and Cd-induced disorders in oxidative status in bones was confirmed [14].

Having in mind that general population is exposed to Cd during lifetime through food chain and tobacco smoke, as well as the fact that bone loss prevention is critical to maintain an active lifestyle, understanding the exact mechanism by which Cd produces toxic effects on bones in experimental animals will provide a better insight into Cd effects on bones in humans. This knowledge is essential for developing appropriate strategies for prevention of Cd-induced bone disorders in humans.

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