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MICROGRAVITY-INDUCED OSTEOPOROSIS: A CHALLENGE FOR THE FUTURE OF SPACE PROGRAMS

Abstract

Objectives

Assess the impact of microgravity-induced osteoporosis and its implications on the future of space programs.

Background

Osteoporosis consists in unbalanced bone resorption and formation. Microgravity-induced osteoporosis has been observed in astronauts since first human missions.

Methods

We performed a narrative review of the available studies about this topic.

Results

During a 4.5 to 6 month stay in space most of the astronauts develop a reduction in bone mineral density (BMD) in spine, femoral neck, trochanter, and pelvis between 1% and 14% measured by Dual Energy X-ray Absorption (DEXA). The same BMD reduction up to -2.6% has been documented in recent RCT on 36-week bed rest, as well as increasing calciuria by the 4th week (200 mg/day lost per month) despite calcium/vitamin-D supplementations, and loss in calcaneal BMD of 5% per each month. High calcium intake (¿1000 mg/d) and vitamin D supplementation (650 IU/d) has been shown to not efficiently counteract the development of space osteoporosis during several European missions. Attempts to prevent disuse osteoporosis with mechanical/biochemical means, including exercise, skeletal compression, increased hydrostatic pressure to the lower body, supplemental calcium and/or phosphorus/calcitonin/etidronate were not successful. More powerful treatments have been tested in animal models (i.e. residronate) with the same negative results, suggesting that bisphosphonates can impair the ability of mature osteoclasts to resorb bone, but cannot overcome the strong stimulus for osteoclast recruitment caused by long-term disuse. Microgravity has been shown to induce changes in genes encoding prolactins, apoptosis/survival molecules, bone metabolism and extra-cellular matrix composition proteins, chemokines, insulin-like growth factor and other molecules, thus suggesting an epigenetic framework of interactions between human genes and external environment. Diminished bone formation was shown in rats on Soviet Cosmos biosatellites with post-flight normalization of cortical bone, but persistent decline in trabecular bone mass with indications

of involvement of mechanical unloading and/or hypersecretion of corticostcroids. In other studies, Gemini, Apollo, and Skylab astronauts exhibited a negative calcium balance due primarily to hypercalciuria. Calcaneal BMD in Skylab crew-members declined by 4% after 84 days of orbital flight. In the International Space Station (ISS) astronauts a 4% decrease in tibial cortical thickness and a 15% increase in cortical porosity at landing was observed. Remodeling marker returned to preflight values within 6 months but tibial cortical porosity or trabecular bone failed to recover, resulting in compromised strength.

Conclusions

Microgravity-induced osteoporosis still represents a major challenge for the future of space programs with human crew.