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Effects of simulated spaceflight on mitochondrial oxidative stress in bone remodelling

A-S. Schreurs[1], S. Torres[1], T. Truong[1], E. L. Moyer[1], A. Kumar[1], Candice. G. Tahimic[1], J.S. Alwood[1] and R.K. Globus[1]

[1] Space Biosciences, NASA-Ames Research Center

Microgravity and ionizing radiation may contribute to cellular stress; resulting in increased generation of reactive oxygen species (ROS), DNA damage, cell cycle arrest, and cell death. We hypothesized that suppression of excess ROS in osteoblasts and osteoclasts will improve bone microarchitecture. To test our hypothesis, we used irradiated transgenic mCAT mice overexpressing human anti-oxidant catalase gene targeted to the mitochondria (main site for ROS production). mCAT mice expressed the transgene and displayed elevated catalase activity in bone and *ex vivo* osteoblast and osteoclast cultures. Treated bone from wildtype mice showed elevated levels of oxidative damage whereas mCAT mice did not. Also, increased catalase activity correlated with decreased MDA levels and that increased oxidative damage correlated with decreased % bone volume. E*x-vivo* osteoblast colony growth positively correlated with osteoblast catalase activity. mCAT mice displayed reduced % bone volume. Treatment caused significant bone loss in wildtype mice. Treatment also caused slight deficits in microarchitecture of mCAT mice. In conclusion, ROS signaling in both osteoblast and osteoclast lineage cells contribute to skeletal development and remodeling and quenching oxidative damage could play a role in bone loss prevention.