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Role of Connexin43 expression in osteoblastic cells on cell viability and bone material properties

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Connexin43 (Cx43) is the major gap junction protein expressed in bone. Reduced Cx43 function in osteoblastic cells *in vitro* or its deletion in mice results in osteoblast dysfunction and delayed ossification. Furthermore, Cx43 is required for some of the effects of bisphosphonates, PTH, and mechanical stimulation on osteocytes and osteoblasts. We have shown that Cx43 expression in osteoblasts and osteocytes is also required for normal development of cortical bone. Indeed, mice lacking Cx43 specifically in osteoblasts and osteocytes exhibit increased osteocyte apoptosis in vertebral cortical bone and femoral midshaft, which consists exclusively of cortical bone. In contrast, osteoblast viability was not affected by deletion of Cx43, demonstrating a different role of the protein in these cell populations. Cortical osteocyte apoptosis was associated with increased endosteal bone resorption and periosteal bone apposition, resulting in widening of the femoral midshaft and increased marrow cavity area. This led to increased moment of inertia, a measure of resistance to bending. Surprisingly, there was no change in the femoral stiffness, suggesting that the quality of the bone material was compromised. Indeed, cortical Young's modulus, a measure of the stiffness of the bone material obtained after correction by size, was decreased. On the contrary, compression tests on vertebrae, which are made mainly of cancellous bone, revealed no difference in material stiffness. These findings indicate that Cx43 is differentially required for maintaining osteocyte viability in cortical but not cancellous bone. Furthermore, our findings are consistent with a role for osteocytes in maintaining material properties of cortical bone.

Biography

Plotkin L. I is an Assistant Professor, Department of Anatomy and Cell Biology, Indiana University School of Medicine. She obtained her degree in Immunology at Universidad Nacional de Buenos Aires, Argentina, and performed postdoctoral training at the University of Arkansas for Medical Sciences. Her research focuses on connexin43 role in intracellular signaling activated by pharmacotherapeutic, hormonal and mechanical stimuli in bone. She published 46 manuscripts, and is member of the editorial board of Bone, Actualizaciones en Osteología, Cell Biology: Research & Therapy, and Endocrinology and Metabolism. Her research is supported by local grants, the National Osteoporosis Foundation and the NIH.

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