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The results indicate prominent antagonistic effects of IL-1α on TGF-β regulated interferon signaling, as well as on a wide variety of other genes and pathways in fibroblasts.
Tissue Engineered Airways: A Prospects Article
Stephanie L. Bogan, Gui Zhen Teoh, and Martin A. Birchall

This article aims to highlight advances in airway tissue engineering and provide an overview of areas to explore and utilize in accomplishing the aim of developing an ideal tracheal prosthesis.

Macrophage-Associated Osteoactivin/GPNMB Mediates Mesenchymal Stem Cell Survival, Proliferation, and Migration Via a CD44-Dependent Mechanism
Bing Yu, Gregory R. Sondag, Christopher Malcuit, Min-Ho Kim, and Fayez F. Safadi

The objective of this study was to investigate the potential role of OA/GPNMB in macrophage-induced MSC function. We found that reparative M2 macrophages express significantly greater levels of OA/GPNMB than pro-inflammatory M1 macrophages. Furthermore, using loss of function and rescue studies, we demonstrated that M2 macrophages-secreted OA/GPNMB positively regulates the viability, proliferation, and migration of MSCs. More importantly, we demonstrated that OA/GPNMB acts through ERK and AKT signaling pathways in MSCs via CD44, to induce these effects. Taken together, our results provide pivotal insight into the mechanism by which OA/GPNMB contributes to the tissue reparative phenotype of M2 macrophages and positively regulates functional activities of MSCs.
Calcium Sensing Receptor Function Supports Osteoblast Survival and Acts as a Co-Factor in PTH Anabolic Actions in Bone

Saja A. Al-Dujaili, Amy J. Koh, Ming Dang, Xue Mi, Wenhan Chang, Peter X. Ma, and Laurie K. McCauley

Anabolic actions of PTH in bone involve increased deposition of mineralizing matrix. Regulatory feedback of the process may be important to maintain calcium homeostasis and, in turn, calcium may inform the process. This investigation clarified the role of calcium availability and the calcium sensing receptor (CaSR) in the anabolic actions of PTH. CaSR function promoted osteoblastic cell numbers, with lower cell numbers in post-confluent cultures of primary calvarial cells from Col1-CaSR knock-out (KO) mice, and for calvarial cells from wild-type (WT) mice treated with a calcilytic. Increased apoptosis of calvarial cells with calcilytic treatment suggested CaSR is critical for protection against stage-dependent cell death. Whole and cortical, but not trabecular, bone parameters were significantly lower in Col1-CaSR KO mice versus WT littermates. Intact Col1-CaSR KO mice had lower serum P1NP levels relative to WT. PTH treatment displayed anabolic actions in WT and, to a lesser degree, KO mice, and rescued the lower P1NP levels in KO mice. Furthermore, PTH effects on whole tibiae were inhibited by osteoblast-specific CaSR ablation. Vertebral body implants (vossicles) from untreated Col1-CaSR KO and WT mice had similar bone volumes after 4 weeks of implantation in athymic mice. These findings suggest that trabecular bone formation can occur independently of the CaSR, and that the CaSR plays a collaborative role in the PTH anabolic effects on bone.

Mitochondrial Inhibitory Factor Protein 1 Functions as an Endogenous Inhibitor for Coupling Factor 6

Misato Kawai, Tomohiro Osanai, Makoto Tanaka, Koji Magota, Hirofumi Tomita, and Ken Okumura

We tested the hypothesis that IF1 may antagonize the biological action of CF6 in human embryonic kidney 293 cells. We generated mature and immature IF1 expression vectors and those labeled with GFP at the C-terminus. In the immature IF1-GFP overexpressing cells, the mitochondrial network of IF1-GFP was newly found at the plasma membrane after peripheral translocation, whereas in mature IF1-GFP transfected cells, a less punctuate rather homogenous pattern was found in the cytoplasm. IF1 protein was detected in the exosome fraction of culture media, and it was enhanced by mature or immature IF1 transfection. Extracellular ATP hydrolysis was enhanced by CF6, whereas immature or mature IF1 transfection suppressed ATP hydrolysis in response to CF6. Intracellular pH was decreased by CF6 but was unchanged after immature IF1 transfection. CF6-induced increase in apoptotic cells was blocked by immature or mature IF1, being accompanied by protein kinase B (PKB) phosphorylation.