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Electromagnetic fields counteract IL-1β during chondrogenesis in synovial bovine mesenchymal progenitor cells

Alessia Ongaro[1], Agnese Pellati[1], Federica Francesca Masieri[1], Angelo Caruso[1], Stefania Setti[2], Michela Battista[2], Ruggero Cadossi[2], Monica De Mattei[1]

[1] Department of Morphology and Embryology, University of Ferrara, Ferrara, Italy,
[2] Laboratory of Clinical Biophysics, IGEA, Carpi, Italy.

Objective. Mesenchymal stem cells (MSCs) isolated from synovium and from synovial fluid, have shown a chondrogenesis potential suggesting that synovium is an excellent source of MSCs for cartilage regeneration. Electromagnetic fields (EMFs) display several effects on cartilage: increase the synthesis of proteoglycans (PGs), prevents the catabolic effect of the pro-inflammatory cytokine interleukin-1β (IL-1β), appear useful for the treatment of osteoarthritis. Our goal was to evaluate if the chondrogenic differentiation of synovial bovine mesenchymal progenitor cells, may be influenced by EMFs. Further, as chondrogenic differentiation of MSCs could be altered in an inflammatory environment and EMFs can counteract IL-1β activity, we also evaluated the role of EMFs during chondrogenic differentiation in the presence of IL-1β.

Design. Synovial fluid was aspirated from the metacarpophalangeal joints of bovine. Synovial cells at the 3rd passage were centrifuged to obtain pellet cultures. Pellets were cultured in chondrogenic medium alone (control) or supplemented with 10 ng/ml TGF-β3 and/or 50 ng/ml IL-1β. The pellets were unexposed or exposed to EMF (75 Hz, 1.5 mT) (Igea, Carpi, Italy), during the whole period in culture (34 days). Alcian blue for sulphated glycosaminoglycans and immunostaining for type II collagen, were performed. PG synthesis was measured by radioactive 35S-sulphate incorporation.

Results. Pellets cultured in the presence of TGF-β3 exhibited positive staining for type II collagen and Alcian blue, compared to control, indicating chondrogenic differentiation of synovial bovine mesenchymal progenitor cells. In the presence of IL-1β, type II collagen and Alcian blue staining dramatically decreased compared to TGF-β3 treatment alone. When pellets treated with both TGF-β3 and IL-1β were exposed to EMF, the histochemical staining for type II collagen and Alcian blue increased compared to EMF-unexposed pellets, suggesting that EMF might counteract the IL-1β effect. Biochemical analysis on PG synthesis confirmed histochemical data.

Conclusions. The presence of inflammatory cytokines, such as IL-1β in human joints, may explain why existing methods of cartilage engineering repair strategies, that rely on the in situ differentiation of MSCs, fail to provide a reliably successful. Results of this study support the hypothesis that EMF treatment may favour chondrogenic differentiation in inflammatory conditions, suggesting a possible strategy for improving the clinical outcome of cartilage repair procedures.