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 1: Osteoarthritis Cartilage. 2003 Jun;11(6):455-62.

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Modification of osteoarthritis by pulsed electromagnetic field--a morphological study.

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OBJECTIVE: Hartley guinea pigs spontaneously develop arthritis that bears morphological, biochemical, and immunohistochemical similarities to human osteoarthritis. It is characterized by the appearance of superficial fibrillation by 12 months of age and severe cartilage lesions and eburnation by 18 month of age. This study examines the effect of treatment with a pulsed electromagnetic field (PEMF) upon the morphological progression of osteoarthritis in this animal model. **DESIGN:** Hartley guinea pigs were exposed to a specific PEMF for 1h/day for 6 months, beginning at 12 months of age. Control animals were treated identically, but without PEMF exposure. Tibial articular cartilage was examined with histological/histochemical grading of the severity of arthritis, by immunohistochemistry for cartilage neoepitopes, 3B3(-) and BC-13, reflecting enzymatic cleavage of aggrecan, and by immunoreactivity to collagenase (MMP-13) and stromelysin (MMP-3). Immunoreactivity to TGFbeta, interleukin (IL)-1beta, and IL receptor antagonist protein (IRAP) antibodies was examined to suggest possible mechanisms of PEMF activity. **RESULTS:** PEMF treatment preserves the morphology of articular cartilage and retards the development of osteoarthritic lesions. This observation is supported by a reduction in the cartilage neoepitopes, 3B3(-) and BC-13, and suppression of the matrix-degrading enzymes, collagenase and stromelysin. Cells immunopositive to IL-1 are decreased in number, while IRAP-positive cells are increased in response to treatment. PEMF treatment markedly increases the number of cells immunopositive to TGFbeta. **CONCLUSIONS:** Treatment with PEMF appears to be disease-modifying in this model of osteoarthritis. Since TGFbeta is believed to upregulate gene expression for aggrecan, downregulate matrix metalloprotease and IL-1 activity, and upregulate inhibitors of matrix metalloprotease, the stimulation of TGFbeta may be a mechanism through which PEMF favorably affects cartilage homeostasis.

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