INTRODUCTION:
Antimicrobial peptides (AP) are abundant and widely distributed effectors of the innate immune response and are able to kill microbes by destructing their cell membranes (1). Defensins are antibiotic peptides involved in host defense at epithelial and mesenchymal surfaces. Previous studies have shown the induction of human \( \beta \)-defensin-3 (HBD-3) in osteoarthritic joints suggesting additional functions to their ability to kill microbes (2). Aim of this study was to investigate the production of human \( \beta \)-defensin-2 (HBD-2) in osteoarthritis and to determine its regulation after being challenged by cartilage degrading cytokines.

For that purpose healthy and osteoarthritic cartilage were assessed for HBD-2 expression by RT-PCR, immunohistochemistry and ELISA. Furtheron stimulation experiments with cultured chondrocytes were performed after addition of IL-1/-6 and TNF-alpha.

METHODS:
Cell culture: Immortalized human chondrocytes (cell line C28/I2) were seeded and cultured under standard conditions (2). Reaching 80% confluence cells were exposed to IL-1/-6 or TNF-alpha (10ng/ml).

RNA preparation and cDNA synthesis: Frozen tissue-samples (100 mg) of healthy and OA-human cartilage were crushed in an achate mortar under liquid nitrogen. Cell lysates were prepared from cultured chondrocytes.

Reverse Transcription Polymerase Chain Reaction (RT-PCR), Real-time RT-PCR, HBD-2 ELISA and Immunohistochemistry were performed as previously described (2, 3).

RESULTS

Figure 1a: RT-PCR analysis revealed transcripts of HBD-2 (255 bp) in osteoarthritic cartilage (lane B and C), but not in healthy articular cartilage (lane D). Lane A: positive control (human epidermis), lane E: negative control, bp: base pair ladder.

Figure 1b: ELISA experiments revealed a strong expression of HBD-2 in osteoarthritic cartilage but neglectable amounts in healthy samples.

DISCUSSION:
After exposure to the stimulators IL-1, IL-6 and TNF-\( \alpha \), real-time RT-PCR and ELISA revealed a clear induction of HBD-2 mRNA and protein levels in cultured chondrocytes. Compared to bacterial stimulation, transcription and protein levels were significantly lower in case of cytokine stimulation. The induction of \( \beta \)-defensin-2 in tissue samples of aseptic osteoarthritic cartilage could not barely explained by recent investigations, because up to now, no in vivo data exist concerning the induction of HBD-2 in sealed-off compartments without contamination due to microbial colonization.

Here we illustrate the induction of HBD-2 in osteoarthritic cartilage without bacterial stimulus. Cytokines in the pathogenesis of osteoarthritis, namely tumor necrosis factor-\( \alpha \), interleukin-1 and -6, were transcriptional inducers of HBD-2 in cultured chondrocytes.

REFERENCES: