Background and objectives CD55 (decay-accelerating factor) is a complement-regulatory protein, which is highly expressed by fibroblast-like synoviocytes (FLS). CD55 is also a ligand for CD97, an adhesion-type G protein-coupled receptor abundantly present on leucocytes. We recently showed that lack of either CD55 or CD97 ameliorates disease in murine collagen-induced and K/BxN serum transfer models of arthritis.[1] Little is known regarding the regulation of CD55 expression in FLS. We therefore investigated the effect of toll-like receptors ligation and pro-inflammatory cytokines on CD55 expression.

Materials and methods Synovial fibroblasts, obtained from biopsy samples of arthritis patients, were cultured and stimulated with cytokines (TNF, IFNγ, IL-1β, IL-6, IFNα) or TLR ligands (LTA, poly (I:C), LPS, imiquimod, CpG). Expression of CD55 was measured by flow cytometry using domain-specific monoclonal antibodies and recombinant CD97-loaded fluorescent beads. Chloroquine was used to inhibit TLR3 activity. Upregulation and functionality of dsRNA sensors in response to poly (I:C) or 5′-triphosphate RNA was analysed by PCR. Apoptosis was measured by PI/annexinV staining and was blocked with the pan-caspase inhibitor Q-VD-OPH.

Results Cultured synovial fibroblasts of patients with rheumatoid arthritis (RA), osteoarthritis, psoriatic arthritis, and spondylarthritis express equal amount of CD55. Stimulation of RA-FLS with IL-1β ($p=0.02$) and poly (I:C) ($p=0.001$) induced a significant upregulation of CD55. Engagement of TLR3 by the dsRNA analog poly (I:C) was confirmed using chloroquine, an inhibitor of endosomal acidification that impairs TLR3 signaling. Synovial fibroblasts also expressed the cytoplasmic dsRNA sensors melanoma differentiation-associated gene 5 (MDA5) and retinoic acid-inducible gene I (RIG-I). Stimulation of these receptors with either poly (I:C) or 5′-triphosphate RNA induced CD55 expression, but, in case of MDA5, also induced significant cell death ($p<0.001$) that was caspase-dependent. Upregulation of CD55 in response to dsRNA receptor activation increased the binding capacity of synovial fibroblasts for CD97-loaded beads.

Conclusions We identify dsRNA as a potent inducer of CD55 upregulation on synovial fibroblasts. Our findings suggest that CD55 induction by viral dsRNA or dsRNA may facilitate the accumulation of CD97-expressing inflammatory immune cells in the synovial tissue.

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A61 TRIGGERING OF VIRAL RNA SENSORS INDUCES CD55 EXPRESSION ON SYNOVIAL FIBROBLASTS

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10.1136/ard.2010.149104.5