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LETTER

Response to 'Adiponectin associates with markers of cartilage degradation in osteoarthritis and induces production of proinflammatory and catabolic factors through mitogen-activated protein kinase pathways'

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See related research by Koskinen *et al.*, http://arthritis-research.com/content/13/6/R184

Different mechanisms and mediators in different stages of osteoarthritis (OA) may be involved in progression of the disease. Cross-sectional studies performed regarding cartilage degradation in OA may therefore not be generalised to whole stages. Koskinen and colleagues' study should be evaluated in light of this knowledge [1].

I read with great interest Koskinen and colleagues' article about the role of adiponectin on cartilage degradation in OA [1]. The authors showed that plasma adiponectin levels and adiponectin released from OA cartilage were higher in patients with the radiographically more severe OA. They suggested that adiponectin is associated with cartilage destruction in OA [1]. Making an extrapolation based on a cross-sectional study in order to account for the mechanism of cartilage degradation in OA, however, may not be very accurate.

Chondrocytes are often believed to exhibit aberrant behaviour and shift to other phenotypes such as anabolic, catabolic or hypertrophic phenotypes over the course of the disease, upon physiological and mechanical stress [2]. The level of biochemical markers and mediators involved in the OA process differ for each one of these phases [3]. Some of these mediators are directly involved in the progression of the OA process or they may be secondary changes in the course of OA. Koskinen and colleagues showed that plasma adiponectin levels were higher in patients with grade IV and V disease than those in patients with grade I, II and III disease (Ahlback classification) [1]. It is my belief that these results do not lead us to conclude that adiponectin could be accountable for degradation of the cartilage. Rather, increased levels of adiponectin may be a secondary phenomenon to the late stage of OA, which could be deemed an indication of severity. Another explanation could be that increased levels of adiponectin may serve as a protective response to the catabolic process in OA. The current understanding of cytokines and growth factors has been shown incapable of determining a single factor that could be responsible for all chondrocyte responses [3].

Another point to be raised in this study would be the effect of adiponectin on OA cartilage and primary chondrocytes *in vitro*. Koskinen and colleagues reported that adiponectin enhanced nitric oxide, IL-6, matrix metalloproteinase-1 and matrix metalloproteinase-3 production in primary OA chondrocytes in a dose-dependent manner [1]. However, the authors only investigated factors involved in the degradation of cartilage. Adiponectin may have a dual effect on cartilage homeostasis, because it may also stimulate anabolic mediators such as tissue inhibitor of metalloproteinase-1 and tissue inhibitor of metalloproteinase-2 in addition to catabolic mediators [4]. Adiponectin may be involved in the joint metabolism by changing the balance of matrix metalloproteinases and tissue inhibitors of metalloproteinases [4].

In a cross-sectional study, as a result, studying a single molecule does not solve the mystery of cartilage destruction. A more versatile approach is required to determine the mechanism of cartilage destruction in OA.

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Abbreviations
IL, interleukin; OA, osteoarthritis.

Competing interests
The author declares that he has no competing interests.

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