TNF-α, IL-8, MCP-1, MIF were detected by ELISA method. Western blot method was used to detect the expression of MyD88, NF-κB, ASC, and Caspase-1 in macrophages.

**Results:** The expression level of MyD88, NF-κB, ASC, and Caspase-1 protein in macrophages of model group was significantly up-regulated, and the levels of IL-1β, TNF-α, IL-8, MCP-1, MIF and NO in the supernatant is significantly increased. TGP, SIN, TGP + SIN and Colchicine group compared with the model group, the expression of NF-κB, MyD88, ASC, Caspase-1 protein in macrophage and the level of IL-1β, TNF-α, IL-8, MCP-1, MIF and NO in supernatant were significantly decreased, the difference was statistically significant (p<0.05). TGP + SIN group compared with Colchicine group, the expression of inflammatory pathways proteins and the levels of inflammatory cytokines were no difference (p>0.05) (table 1).

**Conclusions:** The anti-inflammatory effect of Chinese herbal extracts (TGP and SIN) on the multiple targets of the inflammatory model may be one of its mechanisms of action for the prevention and control of acute gout flares.

**REFERENCE:**

**Disclosure of Interest:** None declared

**DOI:** 10.1136/annrheumdis-2018-eular.4201

---

**Figure 1**

Conclusions: Compression significantly reduced the degradation of type II collagen, which in combination with unaltered formation as measured by ProC2, may suggest suppression of the involved catabolic processes and increased deposition of type II collagen in the cartilage matrix. Furthermore, compression elevated the sGAG release without reduction in the explant GAG content, indicating an increased turnover of GAG. In conclusion, the cartilage ECM turnover is significantly modulated by dynamic compression. This method adds essential translational value to the continuous research addressing OA.

**Disclosure of Interest:** None declared

**DOI:** 10.1136/annrheumdis-2018-eular.6731

---

**Abstract AB0085 – Table 1.** Expression of major inflammatory cytokines and signalling pathway proteins

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>IL-1β (pg/ml)</th>
<th>TNF-α (pg/ml)</th>
<th>MCP-1 (pg/ml)</th>
<th>N</th>
<th>MyD88 (±S)</th>
<th>NF-κB (±S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>9</td>
<td>60.25±5.15</td>
<td>130.96±15.04</td>
<td>399.77±17.76</td>
<td>3</td>
<td>0.37±0.01</td>
<td>0.44±0.01</td>
</tr>
<tr>
<td>Model</td>
<td>9</td>
<td>115.36±8.02</td>
<td>638.11±35.63</td>
<td>568.33±13.06</td>
<td>3</td>
<td>0.83±0.03</td>
<td>0.75±0.03</td>
</tr>
<tr>
<td>Colch</td>
<td>9</td>
<td>78.56±4.46</td>
<td>391.53±11.29</td>
<td>339.77±5.68</td>
<td>3</td>
<td>0.53±0.02</td>
<td>0.56±0.02</td>
</tr>
<tr>
<td>TGP</td>
<td>9</td>
<td>86.84±3.75</td>
<td>461.17±33.01</td>
<td>390.13±8.46</td>
<td>3</td>
<td>0.67±0.02</td>
<td>0.74±0.02</td>
</tr>
<tr>
<td>SIN</td>
<td>9</td>
<td>93.20±4.71</td>
<td>466.50±28.21</td>
<td>431.98±28.39</td>
<td>3</td>
<td>0.69±0.03</td>
<td>0.77±0.03</td>
</tr>
<tr>
<td>SIN</td>
<td>9</td>
<td>79.90±4.49</td>
<td>405.83±20.82</td>
<td>343.72±26.39</td>
<td>3</td>
<td>0.63±0.02</td>
<td>0.57±0.02</td>
</tr>
<tr>
<td>TGP+ SIN</td>
<td>9</td>
<td>63.84±5.16</td>
<td>391.53±27.32</td>
<td>339.77±28.39</td>
<td>3</td>
<td>0.53±0.02</td>
<td>0.56±0.02</td>
</tr>
</tbody>
</table>

**Note:** *p<0.05 vs Control; *p<0.05 vs Model; *p<0.05 vs Colchicine; *p<0.05 vs SIN

**Abstract AB0086 – Figure 1**

Conclusions: Compression significantly reduced the degradation of type II collagen, which in combination with unaltered formation as measured by ProC2, may suggest suppression of the involved catabolic processes and increased deposition of type II collagen in the cartilage matrix. Furthermore, compression elevated the sGAG release without reduction in the explant GAG content, indicating an increased turnover of GAG. In conclusion, the cartilage ECM turnover is significantly modulated by dynamic compression. This method adds essential translational value to the continuous research addressing OA.

**Disclosure of Interest:** None declared

**DOI:** 10.1136/annrheumdis-2018-eular.6731

---

**Cartilage, synovium and bone**

**AB0086**

**TYPE II COLLAGEN AND GLYCOSAMINOGLYCAN TURNOVER IN BOVINE ARTICULAR CARTILAGE IS MODULATED BY LONG-TERM DYNAMIC COMPRESSION**

A. Engstren1,2, A.-C. Bay-Jensen1, M.A. Karstad1, C.S. Thudium2, 1Department of Biomedical Sciences, University of Copenhagen, Copenhagen; 2Nordic BioScience, Herlev, Denmark

**Background:** Osteoarthritis (OA) patients suffer from progressive degradation of articular cartilage, to which there is no available treatment to block or reverse the catabolic mechanisms. Articular cartilage confers a biomechanical function in the joints, and in concordance chondrocytes have shown to be mechanosensitive. The effect of dynamic compression on cartilage extracellular matrix (ECM) could prove to be crucial for translational research in development and screening of new OA drug candidates.

**Objectives:** This study investigates the effect of long-term dynamic compression of a bovine articular cartilage ex vivo model through quantification of cartilage associated biomarkers.

**Methods:** Full depth bovine cartilage explants were cultured for 2 weeks. The explants were treated 3 times a week with either OSM [10 ng/mL] and TNF-α [2 ng/mL] (O-T), or TGF-1 [50 ng/mL]. Untreated samples were included as negative controls (w/o). For each condition two groups were established: an unloaded group and a group compressed 3 times a week. Compression was applied using Electroforce 5500 (TA Instruments), in a sine wave with a maximum load per cycle of 1 MPa, at 1 Hz frequency for 1200 cycles. Metabolic activity was measured once a week using AlamarBlue. Biomarkers released to the supernatant were assessed using the following well-described ELISAs: formation of type II collagen was measured by ProC2, and MMP-degradation of type II collagen was measured by C2M. Sulfated glycosaminoglycans (sGAG) released to media, and sGAG extracted from the explants on the last day using a 4 mM GuHCl solution were quantified using a 1.9-dimethylamino-5-methylbenzoquinone (DMSQ) assay.

**Results:** Compression of bovine explants significantly decreased release of C2M compared to unloaded samples in O-T and w/o groups with 42% and 48% respectively. ProC2 release was not affected by compression. Compression of w/o or TGF-b treated explants increased sGAG release with 195% and 152% relative to their unloaded controls. However, the total amount of sGAG extracted from the explants on the last day remained unchanged. Compression of the explants did not affect the metabolic activity.

**Note:** None declared

**AB0087**

**SYNOVIAL TISSUE MACROPHAGES POLARISATION (M1, M2) IN PATIENTS WITH UNDIFFERENTIATED ARTHRITIS MEETING DIAGNOSTIC CRITERIA FOR RHEUMATOID ARTHRITIS OR PSORIATIC ARTHRITIS ALONG THE FOLLOW UP**

A. Cuervo Aguilera1, S. Fuentelsaz-Romero2, L. Estrada-Capetillo2, R. Celis1, R. Samaniego3, J. Ramírez2, A. Puig-Kröger3, J.D. Cañete1, R. Rheumatology, Arthritis Unit, Hospital Clinic and IDIBAPS, Barcelona, 2Laboratorio de Imuno-Metabolismo, 3Unidad de Microscopía Confocal, Hospital General Universitario Gregorio Marañón, Instituto de Investigación Sanitaria Gregorio Marañón, Madrid, Spain

**Background:** Undifferentiated arthritis (UA) is defined as an inflammatory oligo- or polyarthritis that does not fulfill criteria for a definitive diagnosis. Earlier diagnosis would permits a better functional prognosis. Synovial tissue (ST) macrophages have been associated with disease activity, radiographic erosion and response to therapy in RA. Furthermore, it has been reported that M1 polarised macrophages predominate in RA synovitis, whereas M2 predominate in SpA synovitis.

**Objectives:** To analyse polarised macrophages (M1 proinflammatory and M2 anti-inflammatory) in ST of patients with UA which evolved to RA or PsA, after a long follow-up, to explore their diagnostic value.

**Methods:** To determine the polarisation state of macrophages in ST obtained by arthroscopy from patients with UA that evolved to RA (UA-RA) or PsA (UA-PsA), the expression of proteins associated to GM-CSF-driven polarisation M1 (INHBA, TNFα and MMP12) and M-CSF-driven polarisation M2 (CD209) was assessed in ST. CD163+ macrophages. Patients with established RA(n=12) or PsA(n=10) were included as control. 4 μm cryosections of ST in OCT were blocked for 10 min with 1% human immunoglobulins and incubated with primary antibodies. Imaging was performed with an inverted confocal microscope (SPE, Leica Microsystems) and glyceral immersion objective (AC5-AP0 20x/NA 0.60). Single cell mean fluorescence intensity (MFI)
assessed using Imagej software (National Institutes of Health, Bethesda, MD, USA). At least three random fields were evaluated for each type of ST, quantifying the expression of INHBA, TNF-α, MMP12 and CD209 in all segmented CD163+macrophages. Macrophage density was normalised based on selected tissue area (mm²). After background subtraction, data were plotted using GraphPad software (GraphPad Software, La Jolla, CA, USA).

**Results:** CD163+ sublining (SL) macrophages from UA-RA expressed abundantly the INHBA-encoded activin A, whereas TNFα and MMP12 were variably detected. Regarding the M-CSF-associated marker CD209, 2 populations of CD163+ macrophages were found in the SL of UA-RA, CD163+CD209+ and the other CD163+CD209-, with higher than 100 arbitrary units (au) for CD163+CD209+ and lower than 100 au for CD163+CD209-. Similarly, INHBA, MMP12 and TNFα expression and 2 populations of CD163+ macrophages were detected in CD163+macrophages from UA-PsA. Macrophage density was also found comparable between UA-RA, with 650/mm² in UA-RA and 649/mm² in UA-PsA. Quantification of the above indicated markers in CD163+ST macrophages from established RA and PsA revealed similar levels of INHBA, TNFα, MMP12 and CD209 than those from UA-RA and UA-PsA.

**Conclusions:** This study shows for the first time that the polarisation state of ST CD163+macrophages in UA progressing to RA and PsA is similar to that of established RA and PsA in terms of INHBA, MMP12, TNFα and CD209 expression. Therefore, the inflammatory polarisation state of macrophages is similar in RA and PsA and it is already detected at the earlier steps.

**REFERENCE:**


**Acknowledgements:** Financed “Fondo de Investigación Sanitaria” (PI14/00785. JD Cañete) Instituto de Salud Carlos III. Co-financed BECA FER-2015.

**Disclosure of Interest:** None declared

**DOI:** 10.1136/annrheumdis-2018-eular.4846

---

**AB0099**

**VERBASCOSIDE AND HYDROXYTYSOL DOWNREGULATE STRESS-RELATED PATHWAYS IN HUMAN OSTEOARTHRITIC ARTICULAR CHONDROCYTES**

Z. Elmazoglu1, M. Colakoglu1, S. Baraner2, B. Bak1, C.N. Aitekin1, B. Goker1, C. Karasu1, Gazi University, Middle East Technical University, Rheumatology; Ankara Research and Education Hospital, Ankara, Turkey

**Background:** Osteoarthritis (OA), is a leading cause of joint dysfunction and the disease is characterised by progressive destruction of the articular cartilage. At the molecular level, degeneration of the cartilage is attributed to multifactorial events including oxidative stress, mitochondrial dysfunction, apoptosis and associated changes in inflammatory and catabolic gene expression. Recent studies have revealed the essential role of plant-derived antioxidants in preventing pathological events observed in joint diseases by modulating redox signaling. Objectives: Here, we studied whether the polyphenolic compounds verbascoside and hydroxytyrosol exert chondroprotective effects by suppressing oxidative stress pathways and IL-1, IL-1β-converting enzyme (ICE) expression in human OA chondrocytes.

**Methods:** Chondrocytes were isolated from the joint cartilages of OA patients and OA synovial tissues based on chronic OA synovial tissue in rheumatoid arthritis (RA) patients have been reported. There are, however, few studies comparing histopathological changes in the synovial tissue in RA and PsA revealed similar levels of INHBA, TNFα, MMP12 and CD209 than those from RA and PsA.

**Conclusions:** This study shows for the first time that the polarisation state of ST CD163+macrophages from established RA and PsA revealed similar levels of INHBA, TNFα, MMP12 and CD209 than those from UA-RA and UA-PsA.

**REFERENCES:**


