important role in the etiology of PFP. It has been suggested that a change in cartilage composition, due to deterioration of structural components like collagen and glycosaminoglycan’s (GAGs), precedes morphological characteristics of cartilage damage in OA patients. With innovative MRI sequences, including T1p and T2 mapping, it is possible to measure these early changes in cartilage composition quantitatively by measuring cartilage content. Therefore, the purpose of this study was to investigate differences in cartilage composition between patients with PFP and control subjects using quantitative T1p and T2 mapping MRI.

**Methods:** Patients diagnosed with PFP and healthy control subjects aged 14-40 years were included in a cross-sectional case-control study. Measures included a questionnaire, physical examination and MRI at 3T. MRI comprised morphologic, T1p and T2 mapping sequences. T1p and T2 mapping sequences were conducted to measure cartilage glycosaminoglycan and collagen content, respectively. In-house developed software was used for image post-processing in order to calculate T1 and T2 relaxation times (see Figure 1). Higher relaxation times equal less content and less content equals a lower cartilage quality. Differences within the patellar and trochlear cartilage. However, follow-up research will demonstrate potential regional differences within the patellar and trochlear cartilage.

**Results:** 59 patients and 67 control subjects were included. BMI was significantly lower and sports participation significantly higher in control subjects. Mean T1p relaxation times of the patellar (46.8 vs 46.1 milliseconds (ms), p = 0.94) and trochlear cartilage (50.9 vs 50.1 ms, p = 0.52) did not significantly differ between patients and control subjects. In addition, no significant difference was seen between patients and control subjects in mean T2 relaxation times of patellar (33.4 vs 32.8 ms, p = 0.16) and trochlear cartilage (36.8 vs 36.6 ms, p = 0.70) (see Table 1).

**Conclusions:** Our findings suggest that cartilage composition, measured by T1p and T2 mapping, does not play a role in the etiology of PFP. However, follow-up research will demonstrate potential regional differences within the patellar and trochlear cartilage.

**Table 1**

<table>
<thead>
<tr>
<th></th>
<th>Patients (N=59)</th>
<th>Controls (N=67)</th>
<th>Mean difference (± 95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>T2 trochea</td>
<td>36.81 (2.55)</td>
<td>36.63 (2.39)</td>
<td>0.18 [-0.69, 1.05]</td>
<td>0.70a</td>
</tr>
<tr>
<td>T2 patella</td>
<td>33.41 (2.86)</td>
<td>32.82 (2.56)</td>
<td>0.59 [-0.36, 1.55]</td>
<td>0.16b</td>
</tr>
<tr>
<td>T1p trochea</td>
<td>50.85 (3.57)</td>
<td>50.13 (4.03)</td>
<td>0.72 [-0.72, 2.17]</td>
<td>0.52b</td>
</tr>
<tr>
<td>T1p patella</td>
<td>46.79 (4.21)</td>
<td>46.09 (4.43)</td>
<td>0.70 [-0.94, 2.34]</td>
<td>0.94b</td>
</tr>
</tbody>
</table>

sd = standard deviation
CI = confidence interval
a: adjusted for BMI, sports participation and gender
b: adjusted for BMI and sports participation


**AUTOPHAGY AND OSTEOARTHRITIS DEVELOPMENT**

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**Purpose:** We studied here macro-autophagy (thereafter referred to as autophagy), a lysosomal recycling process. Lysosomal protein recycling processes promote cell survival during nutrition depletion by recycling cellular building blocks. Autophagic activity modulation during osteoarthritis progression had been pointed out. However, there is no study directly studying the role of autophagy in osteoarthritis progression.

**Methods:** To functionally address the role of autophagy in vivo, we generated mice with cartilage specific ablation of Atg5 gene, which is indispensable for autophagy. We analysed the knee joints of these mice at 2, 6 and 12 months of age.

**Results:** At 2 and 6 months, no differences in bone length or bone and cartilage shape were observed between transgenic animals and wild type in both strains. However, at the age of 1 year, Atg5cko joints showed features of OA whereas wild type animals had healthy joints (OARSI score: WT, 0.3±3.19; Atg5cko: 12.6±3.99; N=5; p=0.007). The analysis of 2 months old Atg5cko knees have higher cell death level (TUNEL, WT, 5.2±3.13; Atg5cko: 17.0±4.06; N=6; p=0.024). This was correlated with an increase in caspase 3 (WT: 1.88±1.82; Atg5cko: 8.45±2.3; N=5; p=0.031) and caspase 9 (WT: 0.72±0.39; Atg5cko: 3.58±1.09; N=5; p=0.043) positive cells.

**Conclusions:** Our results suggest that autophagy protects chondrocytes from caspase mediated apoptosis and inhibition of this process leads to chondrocyte apoptosis and tissue breakdown.

**487 CONTRIBUTION OF ELF3 TO CARTILAGE DAMAGE IN A NON-INVASIVE MECHANICAL LOADING MOUSE MODEL WITH OSTEOARTHRITIS-LIKE PATHOLOGY**

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**Purpose:** The E74-like Factor 3 (Elf3) has been shown to contribute to cartilage degradation and mediate inflammatory responses in chondrocytes via up-regulation of downstream targets genes, including Mmp13. In this study, we aimed to assess whether cartilage-specific ELF3 deficiency protects against OA, and attenuates the progression of cartilage damage in vivo, using a non-invasive mechanical loading mouse model that triggers OA-like pathology.

**Methods:** We generated Col2a1Cre-driven cartilage-specific Elf3-knockout (Col2Cre:Elf3f/f) mice, that showed no obvious differences in size, weight and growth plate morphology when compared to wild-type counterparts. We subjected the left knees of 12-weeks-old male Col2Cre:Elf3f/f (KO) and Elf3f/f Cre-negative controls (WT) to 1 and 4 weeks of non-invasive mechanical loading, at 9N for 1200 cycles/day. The right knees were used as non-loaded controls. At 1-week post-loading, we isolated knee articular cartilages from three KO or three WT mice, and the loaded and control cartilages from the KO or WT littersmates were pooled together for the isolation of total RNAs and subsequent RTqPCR analyses. At 4-weeks post-loading, mice were harvested followed by fixation and decalcification in paraformaldehyde and EDTA, respectively. Histological assessment of OA was conducted on Safranin-O/fast green stained serial coronal sections, following the OARSI recommendations for assessment of OA in the mouse.

**Results:** Our initial RTqPCR analyses showed increased Mmp13 and Ptg2 expression in the loaded legs of WT animals compared to unloaded controls. Interestingly, the expression of these genes was down-regulated in KO animals, highlighting the role of Elf3 in controlling these downstream effectors. Preliminary histological analyses of WT (n=3) and KO (n=3) knee joints at 4-weeks post-loading indicated a trend towards reduced cartilage degradation scores in the KO animals, which is in agreement with the results obtained by RTqPCR and with the notion that Elf3 contributes to cartilage degradative processes.

**Conclusions:** We here provide preliminary evidence that Elf3 contributes to cartilage degradation in vivo, using a non-invasive mechanical loading mouse model. Cartilage-specific Elf3 KO animals show a trend towards reduced cartilage erosion following loading, which correlated