OA-FLS and MSC on proteome level revealed 598 differentially expressed proteins. We observed no differences in the expression of classical fibroblast markers such as vimentin, tenascin C and decorin as confirmed on RNA level. Remarkably, fibronectin, which is mainly produced by fibroblasts, is significantly lower expressed at both protein and RNA levels in OA-FLS together with collagen type 1 and CD106. Conversely, CD9, CD54 and fibroblast-specific protein-1 were expressed significantly higher in FLS at both levels, while hyaluronan synthase 1-3 remained unchanged. Of note, in terms of mitochondrial function, human OA-FLS show a significantly lower basal respiration and ATP production than MSC, but a comparable spare respiratory capacity and cellular mitochondrial dehydrogenase activity (NADH amount) per cell. Additionally, we identified the pyruvate dehydrogenase kinase (PDK) 3 to be highly expressed in OA-FLS, while the expression of mitochondrial ATP synthase subunits, electron transport chain complexes and glycolytic enzymes was comparable with MSC. Finally, inhibition of PDK by using DCA resulted in a significant increase in oxygen consumption rate and ATP production in OA-FLS. Thus, our data newly suggest, that PDKs may play a crucial role in the pathogenesis of OA and possibly RA.

Conclusion: Our data provide evidence that, although the classical fibroblast markers do not discriminate between MSC and FLS, the latter demonstrate a significantly higher expression of PDKs, known to inhibit the pyruvate entry into the TCA cycle which finally limits the mitochondrial ATP production. Therefore, shifting the metabolism of FLS from glycolysis to mitochondrial respiration via inhibition of PDKs might be a novel approach in OA for the development of disease-modifying osteoarthritis drugs in order to unleash the metabolic flow.

Disclosure of Interests: None declared

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AB0041

TO ESTIMATE THE PREVALENCE OF ACETABULAR OSSIFICATIONS IN THE ADULT POPULATION WITH ASYMPTOMATIC, MORPHOLOGICALLY NORMAL HIPS AT CT AND TO DETERMINE WHETHER THE PRESENCE OF LABRAL OSSIFICATIONS IS ASSOCIATED WITH PATIENT-RELATED OR HIP-RELATED PARAMETERS

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Background: In clinical practice, ossifications adjoining the acetabular rim are frequently seen in healthy-appearing hip joints, at all ages. The origin of these acetabular rim ossifications (ARO) is unclear. While ARO have been described by some authors as variants that may mimic marginal acetabular osteophytes, others have hypothesized that ARO may be the cause or the consequence of femoroacetabular impingement. Overall, the literature on ARO is limited, in particular in the healthy population, and their characteristics and origin not fully understood.

Objectives: To estimate the prevalence of acetabular rim ossifications in the adult population with asymptomatic, morphologically normal hips at CT and to determine whether the presence of these ossifications is associated with patient-, or hip-related parameters.

Methods: We prospectively included all patients undergoing thoracoabdominal CT over a 3-month period. After exclusion of patients with a clinical history of hip pathology and/or with signs of osteoarthritis on CT, we included a total of 150 hips from 75 patients. We analyzed the presence and the size of ossifications around the acetabular rim. The relationships between the size of acetabular rim ossifications and patient-related (sex, age, BMI), or hip-related parameters (joint space width, and cam- and pincer-type femoroacetabular impingement morphology) were tested using multiple regression analysis.

Results: The prevalence of acetabular rim ossifications in this population of asymptomatic, non-osteoarthritic hips was 96% (95%CI=[80.1;100.0]). The presence of ossifications and their size were correlated between right and left hips (Spearman coefficient=0.64 (95%CI=[0.46;0.79]), p<0.05). The size of acetabular rim ossifications was significantly associated with age (p<0.0001) but not with BMI (p=0.35), gender (p=0.05), joint space width (p≥0.53 for all locations) or any of the qualitative or quantitative parameters associated with femoroacetabular morphology (p≥0.34).

Conclusion: Acetabular rim ossifications are highly prevalent in asymptomatic, non-osteoarthritic adult hips at all ages. Their size is not correlated with any patient-, or hip-related parameters except for the age. These findings suggest that ossifications at the acetabular rim, when present in isolation, should not be considered as signs of osteoarthritis or femoroacetabular impingement morphology.

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Table 1. Association between the average severity size of ossifications and patient-related, and hip-related parameters

		Regression coefficient	p- value
Patient-related parameters	BMI	0.02	0.35
	Age	0.04	<0.001
	Gender	-0.43	0.05
Joint space width	Apical	0.17	0.53
	Supero-medial	0.17	0.65
	Supero-lateral	0.06	0.82
Cam morphotype parameters	Angle alpha 45	0.00	0.81
	Offset	0.09	0.34
Pincer morphotype parameters	Acetabular version angle	-0.01	0.83
	Lateral center-edge angle	0.02	0.56
	Acetabular index	0.03	0.53
	Crossover sign	0.26	0.41
	Posterior wall sign	-0.25	0.47

Figure 1. Coronal reformats of CT examinations showing different sizes of labral ossifications in the posterosuperior quadrant (arrows). (a) No labral ossification, (b-c-d-e) Increasingly large labral ossification from left to right. Two lines are visible at the location of the ossification, formed by the contour of the acetabular rim and of the ossification (double line-sign).

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Rheumatoid arthritis - aetiology, pathogenesis and animal models

AB0042

THREE HEMATOLOGIC/IMMUNE SYSTEM-SPECIFIC EXPRESSED GENES ARE CONSIDERED AS THE POTENTIAL BIOMARKERS FOR THE DIAGNOSIS OF EARLY RHEUMATOID ARTHRITIS THROUGH BIOINFORMATICS ANALYSIS

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Background: Rheumatoid arthritis (RA) is a common chronic autoimmune connective tissue disease that mainly involves the joints. The incidence of RA is 5 to 10 per 1000 people[1]. Early diagnosis and treatment of RA can effectively prevent disease progression, joint damage, and other complications in 90% of patients[2]. At present, serum biomarkers used in the diagnosis of established RA are rheumatoid factor and anti-cyclic citrullinated peptide antibody[3]. However, early RA especially serum RF and anti-CCP antibody-negative is difficult to diagnose due to the lack of effective biomarkers. Therefore, it is vital to identify new and effective biomarkers for the early diagnosis and treatment of RA.

Objectives: This study aimed to identify new biomarkers and mechanisms for RA disease progression at the transcriptome level through a combination of microarray and bioinformatics analyses.

Methods: Microarray datasets for synovial tissue in RA or osteoarthritis (OA) were downloaded from the Gene Expression Omnibus (GEO) database, and differentially expressed genes (DEGs) were identified by R software. Tissue/organ-specific genes were recognized by BioGPS. Enrichment analyses were performed and protein-protein interaction (PPI) networks were constructed to understand the functions and enriched pathways of DEGs and to identify hub genes. Cytoscape was used to construct the co-expressed network and competitive endogenous RNA (ceRNA) networks. Biomarkers with high diagnostic value for the early diagnosis of RA were validated by GEO datasets. The ggpubr package was used to perform statistical analyses with Student's t-test.

Results: A total of 275 DEGs were identified between 16 RA samples and 10 OA samples from the datasets GSE77298 and GSE82107. Among these DEGs, 71 tissue/organ-specific expressed genes were recognized. Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analysis indicated that DEGs are mostly enriched in immune response, immune-related biological process, immune system, and cytokine signal pathways. Fifteen hub genes and gene cluster modules were identified by Cytoscape. Eight hematologic/immune system-specific expressed hub genes were verified by GEO datasets. GZMA, PRC1, and TTK may be biomarkers for diagnosis of early RA through combined the analysis of the verification results and the receiver operating characteristic (ROC) curve. NEAT1-miR-212-3p/miR-132-3p/miR-129-5p-TTK, XIST-miR-25-3p/miR-129-5p-GZMA,