A59 CHONDROGENIC AND OSTEOGENIC POTENTIAL OF ADIPOSE DERIVED STEM CELLS FROM RA AND OA PATIENTS

U Skalska,[1] T Burakowski,[1] I Janicka,[1] A Kornatka,[1] P Maldyk,[2] W Maslinski,[1] E Kontny[1] *[1]Department of Pathophysiology and Immunology, Institute of Rheumatology, Warsaw, Poland; [2]Rheumoorthopaedic Clinic, Institute of Rheumatology, Warsaw, Poland*

10.1136/ard.2010.149104.3

Background and objectives Adipose derived stem cells (ADSC) have a potential to differentiate into cells of mesodermal origin (chondrocytes, osteoblasts, adipocytes) and recently emerged as an attractive source of mesenchymal stem cells for cell therapy. Bone and cartilage engineering is the obvious therapeutic application of ADSC in rheumatoid arthritis (RA) – in RA chronic joint inflammation leads to cartilage and bone destruction causing patients disabilities.

The aim of our study was to analyse the percentage of ADSC from RA patients and to evaluate their chondrogenic and osteoblastogenic potential in comparison to ADSC derived from osteoarthritic (OA) patients. We examined also the effect of tumour necrosis factor α (TNF-α) – critical cytokine for RA pathogenesis, on the ADSC differentiation.

Materials and methods Intra-articular adipose tissue was obtained from RA and OA patients during total knee joint replacement surgery. ADSC cells were isolated and cultivated in DMEM/F12/10% FCS medium. After forth passage cells were analysed using flow cytometry for three stem cells markers: CD73, CD90 and CD105. Differentiation of ADSC was performed in chondrogenic or osteoblastogenic medium in the absence or presence of TNF-α (10 ng/ml). The expression of chondrogenesis (Sox9, aggrecan, collagen 2a) and osteogenesis (Runx2, BMP-2, osteopontin - OPN) markers was evaluated by RT-PCR reaction.

Results There was no significant difference between ADSC percentage in RA and OA intra-articular adipose tissue. 77.3% (RA) and 77.6% (OA) of cells had co-expression of three stem cells markers: CD73, CD90 and CD105. Expression of Runx2 and BMP-2 was similar in RA and OA ADSC, while OPN expression was significantly higher in RA than in OA ADSC. TNF-α enhanced BMP-2 and Runx2 expression in RA and OA cells, but inhibited osteopontin expression. The expression of Sox 9, aggrecan and collagen 2a were similar in RA and OA. TNF-α significantly inhibited expression of all three chondrogenesis markers.

Conclusions ADSC frequency in intra-articular adipose tissue is similar in RA and OA patients. ADSC from OA and RA patients have similar chondrogenic and osteoblastogenic potential. Interestingly OPN mRNA expression was higher in RA ADSC. Further studies are required to investigate whether this protein, which is thought to play proinflammatory role as an extracellular form, is released from RA ADSC. TNF-α inhibits chondrogenesis of ADSC from both OA and RA patients. It is possible that TNF-α may enhance early stages of osteogenesis (upregulates Runx 2 and BMP-2 mRNA expression) and inhibit late stages (diminishes OPN mRNA expression) both in RA and OA patients.