A214 THE INFRAPATELLAR FAT PAD OF OSTEOARTHRITIC PATIENTS HAS AN INFLAMMATORY PHENOTYPE

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Background and objectives Obesity is a risk factor for the development of osteoarthritis (OA) in hands and knees. It has become apparent during the last years that adipose tissue can secrete different adipokines with powerful immunomodulatory effects. Because the infrapatellar fat pad (IFP) is an intra-articular organ in the vicinity of synovium and cartilage, the authors hypothesised that IFP-derived soluble factors could contribute to pathological processes in the knee joint. Therefore the authors have extensively compared the release of inflammatory mediators and characterised the adipocytes and immune cell infiltrate in IFP and Sc adipose tissue (ScAT).

Materials and methods Paired IFP and ScAT samples were obtained from 27 primary OA patients. The stromal vascular cell fraction (SVF) was isolated and characterised by FACS. Cytokine and adipokine release in fat- and adipocyte-conditioned media was measured by luminex.

Results IFP secreted higher levels of inflammatory mediators, like IL-6, adipsin, adiponectin and visfatin than ScAT. This could be due to differences in the phenotype of adipocytes or/and in composition and phenotype of the SVF cells. Indeed, a similar trend was seen for IL-6 and adipsin when adipocyte-conditioned media from IFP and ScAT were compared. Moreover, the SVF fraction of IFP contained more cells per gram tissue, a lower percentage of T cells and a higher percentage of mast cells than ScAT. In addition, IFP-derived T cells displayed a predominantly proinflammatory phenotype, while macrophages in IFP presented a mixed pro- and anti-inflammatory phenotype. Finally, tumour necrosis factor- α release by IFP was correlated with BMI, indicating BMI-related inflammatory changes in IFP.

Conclusions The authors show profound differences in secreted inflammatory factors and immune cell composition between IFP and ScAT. These data indicate that IFP is qualitatively different from ScAT and IFP-derived soluble mediators could contribute to pathophysiological processes in the OA knee joint.