(87.5%). The highest category of disease duration for the cohort was between 2–5 years with 40% (n=16) patients. 30% (n=12) of the respondents were aware of the 'know your numbers' concept. 27.5% (n=11) knew their cholesterol while 80% did not know their last blood sugar. The majority 90% (n=36) did not know their BMI. Only 25% of the respondents knew their DAS score. 50% knew the significance of the numbers. 40% (n=16) reported that no one had informed them about the numbers. 95% (n=38) of the participants showed interest in knowing their numbers and 27.5% (n=11) suggested that a written record explained and regularly updated would be appropriate whilst 35% (n=14) proposed that a multidisciplinary input would be useful in regularly informing them of the numbers.

Conclusions: Although cardiovascular disease risk assessment and the management has improved in RA. There still remains a gap in patient engagement and activation in taking responsibility in knowing the risks and what they mean. It is well documented that patients with increased level of activation are more likely to engage in positive health behaviours resulting better outcomes. Our study has shown that although patients do not know much about their biomedical data they are interested in knowing about them. Knowing their biomedical may encourage them to take more ownership of their health even leading to more self-managing their RA.

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The EULAR exercise recommendations for physical activity in people with inflammatory arthritis and osteoarthritis

OP0222-HPR PHYSICAL CAPACITY CONTRIBUTES marginally IN EXPLAINING VARIATIONS OF FATIGUE IN PERSONS MODERATELY AFFECTED BY RHEUMATOID ARTHRITIS

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Background: Fatigue is a prominent problem for persons with rheumatoid arthritis (RA). Physical activity, including planned and structured exercise, is an important non-pharmacological intervention that positively affects fatigue. Physical capacity including variables as aerobic capacity, muscle strength and lower limb capacity, are modifiable and their relative contributions to fatigue are thus of interest to better understand and address this detrimental consequence of RA.

Objectives: To explore the contributions of physical capacity in explaining variations in fatigue among people with rheumatoid arthritis (RA).

Methods: This cross-sectional study included 269 participants (82% women, mean age 60 years, mean DAS28 2.8, mean HAQ-DI 0.5) recruited for a physical activity intervention. Data were collected from the Swedish Rheumatology Quality Registers, from questionnaires on fatigue, activity limitation, perceived health, pain and anxiety/depression and from physical capacity tests (lower limb function, grip strength, aerobic capacity). We used logistic regression to estimate the association between severe fatigue (≥ 50 , VAS 0–100) and A) independent variables related to disease and disease impact and B) model A plus physical capacity tests. Pooled odds ratio tests compared model fit.

Results: Severe fatigue was reported by 95 participants (35%). The three variables which were statistically significantly associated with severe fatigue ($p < 0.05$) in both models were perceived health, pain and anxiety/depression. Anxiety/depression demonstrated the largest effect size with odds ratios of 2.43 (95% CI: 1.20 to 4.94) in model A and 2.58 (95% CI: 1.25 to 5.32) in model B. The likelihood ratio test indicated that model B was a better fit to the data than model A with χ^2 (df 3)=2.65, $p=0.048$.

Conclusions: We found that disease impact variables rather than physical capacity variables are predictors of severe fatigue in people with RA. Further studies are needed to assess correlations between physical capacity and fatigue using multidimensional assessments of fatigue to enable separate analyses of physical and mental fatigue respectively.

Disclosure of Interest: None declared

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Immune senescence and ageing

OP0223 TARGETING CHONDROCYTE PLASTICITY VIA CONNEXIN43 MODULATION ATTENUATES CELLULAR SENESCENCE IN OSTEOARTHRITIS

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Background: Chondrocytes in articular cartilage undergo phenotypic changes and senescence, restricting cartilage regeneration and favouring the osteoarthritis (OA) progression, a chronic disease characterised by degeneration of articular cartilage leading to physical disability and pain. Like other wound healing disorders, chondrocytes from OA patients show a chronic increase in the transmembrane channel protein connexin43 (Cx43), which through the exchange of factors or recruitment/release of signalling factors to the membrane regulates signal transduction. Immature or stem-like cells are found in cartilage isolated from OA patients, yet their origin and role in disease progression are unknown.

Objectives: Our objective was to investigate the role of Cx43 in chondrocyte plasticity during osteoarthritis.

Methods: Human mesenchymal stem cells and chondrocytes from osteoarthritic donors were used in this study. Protein levels were evaluated by western blot, immunofluorescence and flow cytometry. RNA expression was evaluated by RT-qPCR. Cell communication was studied by scrape loading assay and flow cytometry.

Results: Here we show that Cx43 acts as a positive regulator of the reversion of human chondrocytes to a less differentiated state. Overactivity of Cx43 in the membrane largely maintain this stem-like phenotype by increasing the basic helix-loop-helix transcription factor TWIST-1 and tissue remodelling and proinflammatory factors such as MMPs and IL-6, which in turn lead to cellular senescence by upregulation of p53 and p16INK4a, contributing to the senescence-associated secretory phenotype (SASP). Downregulation of Cx43 leads to redifferentiation of osteoarthritic chondrocytes (OAC) into more differentiated state decreasing MMPs, proinflammatory cytokines production and cellular senescence. Collectively, these results identify a causal Cx43-sensitive circuit in chondrocytes that regulates dedifferentiation and redifferentiation events involved in wound healing and tissue repair. In addition we show that chronic dedifferentiation drives catabolic processes and cellular senescence.

Conclusions: Targeting Cx43 allows dedifferentiated chondrocytes to revert to a chondrocyte-specific phenotype and its subsequent recovery in a predictable manner. These findings support the use of Cx43 as an appropriate therapeutic target to halt OA progression and to promote cartilage regeneration

Disclosure of Interest: None declared

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Special delivery: intercellular communication

OP0224 ADAMTS-12 PROTECTS AGAINST INFLAMMATORY ARTHRITIS THROUGH INTERACTING WITH PROINFLAMMATORY CTGF

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Background: It has been reported that a disintegrin and metalloproteinase with thrombospondin motifs-12 (ADAMTS-12) is a susceptibility gene for rheumatoid arthritis (RA) development, and its level was significantly increased in RA patients. In addition, ADAMTS-12 was also reported to be required for normal inflammation.

Objectives: This study aims to determine the role of ADAMTS-12 and the underlying mechanisms in the pathogenesis of inflammatory arthritis.

Methods: Collagen-induced arthritis (CIA) was established in ADAMTS-12^{-/-} mice and their control littermates to determine the role of ADAMTS-12 *in vivo*. Connective tissue growth factor (CTGF) deficient Raw264.7 were used to